

Breaking Bad: How Health Shocks Prompt Crime

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Abstract

Exploiting plausibly exogenous variations in the timing of cancer diagnoses, we establish that health shocks elicit a large and persistent increase in the probability of committing a crime. This effect materializes in a substantial rise in both first crimes and re-offenses. We uncover evidence for two mechanisms. First, an economic motive leads individuals to compensate the loss of legal revenues with illegal earnings. Second, cancer patients face lower expected cost of punishment through a lower survival probability. Welfare programs that alleviate the economic repercussions of health shocks are effective at mitigating the ensuing negative externality on society.

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1. INTRODUCTION

Do health shocks elicit criminal behavior? An 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 potential punishment, and the personal attitude towards risk. Health shocks affect to an extent all these dimensions. First, health shocks can diminish a person’s human capital and her ability to earn legal income, thereby making illegal activities, *ceteris paribus*, more attractive.¹ Second, 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](#)). In this paper, we leverage rich administrative data from Denmark to link health and criminal records at the individual level and empirically explore whether (and why) individuals who experience health shocks “break bad.”

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 variation 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 include person-fixed effects to account for the impact of unobservable invariants at the individual level. Our identification strategy exploits that, conditional on age, invariant traits,

¹For instance, because individuals who have been diagnosed with an illness are less productive, work fewer hours, or are less likely to be promoted ([Dobkin et al. 2018a](#), [Fadlon and Nielsen 2021](#)).

and on developing cancer at some point, the timing of the cancer diagnosis is as good as random.

We find that the probability of committing a crime increases on average by 14% 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. Furthermore, we show that cancer leads individuals without a criminal record to violate the law for the first time and drives offenders to increase the propensity to commit crime again.

In the second part of our analysis, we seek empirical evidence for the mechanisms that link health shocks to crime. Specifically, we consider several mechanisms: Cancer may contribute to increased criminal activity through i) changes in economic circumstances, ii) a decrease in survival probabilities which reduces perceived costs of punishment, and iii) alternative channels, including psychological distress and changes in risk attitudes. First, we document that cancer leads to a change in economic circumstances; cancer has a negative impact on income, likelihood of employment, and hours worked. In line with the presence of an economic motive, we find that the incentive to break the law is stronger for individuals who lack insurance through home-equity (Gupta et al. 2018) or marriage (Fadlon and Nielsen 2021). Furthermore, we find that, in absolute terms, most of the crimes that follow a cancer diagnosis are economically motivated. Yet, the *relative* increase in economic crimes is smaller than that of non-economic crimes (14% versus 38%), pointing to the presence of additional channels. In particular, we provide evidence for a survival probabilities mechanism: individuals experiencing a greater decline in their survival probability from cancer increase criminal activity to a larger extent. Finally, we find that the cancer-crime relation is stronger for people who seek psychological help, suggesting the presence of a psychological distress mechanism as well. By contrast, we do not find evidence that cancer alters risk preferences. Taken together, our results indicate that several mechanisms are at play.

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.² We document that a decrease in the generosity of social security fosters an increase in the sensitivity of crime to health shocks.

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. [Otsu and Yuen \(2022\)](#) and [Schroeder et al. \(2011\)](#) find a contemporaneous negative correlation between self-reported measures of health status and criminal behavior. Furthermore, [Corman et al. \(2011\)](#) 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 identifies 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 \(2024\)](#) 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 et al. 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 crimes at older ages. In fact, after excluding people who are old enough to retire, the average age of individuals who are diagnosed with cancer is 52.

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 an income channel by investigating the effects of job loss, loss of supplemental security income, access to the labor market, and returns to crime ([Bennett and Ouazad 2020](#), [Britto, Pinotti and Sampaio 2022](#), [Deshpande and Mueller-Smith 2022](#), [Draca, Koutmeridis and Machin 2019](#), [Grönqvist 2011](#), [Miguel 2005](#), [Öster and Agell 2007](#), [Pinotti 2017](#), [Massenkoff and Rose 2024](#), [Yang 2017](#)). These papers document an increase in criminal activity from the loss of income that ranges from 20% to 32%;

²A necessary limitation of this analysis is that the welfare reform had effects on a variety of policies affecting undiagnosed individuals as well.

almost double the 14% increase we estimate in response to cancer diagnoses. Yet, the effect of job loss is typically short-lived (see, e.g., [Bennett and Ouazad 2020](#) and [Massenkoff and Rose 2024](#)) compared to the more persistent long-run effects of cancer. Furthermore, [Jácome \(2022\)](#) studies the effect of worsened mental healthcare on criminal propensity, finding that a loss of Medicaid eligibility increases crime propensity by 14%, which is comparable to our main effect. Regarding punishment, previous literature focuses mainly on the implications of a greater likelihood of apprehension (e.g., [Ayres and Levitt 1998](#), [Di Tella and Schargrotsky 2004](#), [Draca, Machin and Witt 2011](#), [Fella and Gallipoli 2014](#), [Fu and Wolpin 2018](#)). We provide novel evidence by documenting that survival probabilities affect the expected cost of punishment, leading to increased incidence of crime.

Finally, our paper adds to the literature on the consequences of health shocks. First, we confirm existing literature and show that health impacts labor market outcomes, in particular, earnings, employment, and hours worked (e.g., [Dobkin et al. 2018a](#), [García-Gómez et al. 2013](#), [Heinesen and Kolodziejczyk 2013](#), [Jeon and Pohl 2019](#), [Moran, Short and Holtenbeak 2011](#)). The conventional approach in this literature is to consider the implications of health shocks for the affected individual and her close family (e.g., [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 through increased crime.

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. INSTITUTIONAL 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, which consists broadly of three parts. First, short-term sick pay and, depending on the occupation, employer-based policies (lump sum payment for critical illness). Short-term coverage is then followed by state-funded sickness benefits. When state-funded sickness benefits run out, individuals are eligible to either nothing or some social insurance, early retirement programs, or permanent Social Disability Insurance.

Regarding the first component, workers are eligible to full pay during an initial period of absence due to sickness. Coverage termination depends on the employee's contract and on whether the employer lets the employee go after the contractual obligation to retain her expires. Additionally, employer-based insurance policies and private pension plans have become standard, and these provide a lump sum source of income to those who experience critical health shocks.

Second, when employment is terminated, or the employment contract does not include full wage insurance during sickness, the employee can apply for state-funded sickness benefits at the municipality of residency. Sickness benefit duration varies somewhat over the period of interest, and as of 2019, lasts for a maximum of 22 weeks, though extended coverage is negotiable with the municipality if certain conditions are met. The sickness benefits amount to a maximum of 4,355 Danish kroner (DKK) per week in 2019 (\$702).

In the final stage, when an individual is permanently unable to work, she can apply for a disability pension with her municipality of residence. Different municipalities administer both sick leave benefits and disability benefits to some degree differently. Approved ap-

plicants receive benefits that, in 2019, amounted to DKK 192,528 (\$31,053) per year for married or cohabitating individuals and DKK 226,500 (\$36,532) for singles.³

2.2. Administrative registry data

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

Individual identifiers and information on individual and household characteristics are obtained from the *Population Registry* (Statistics Denmark 2024) and the *Household and Family Registry* (Statistics Denmark 2023c). Information on income and wealth are linked through the *Income and Tax Registry* (Statistics Denmark 2023f), while labor market variables come from the *Work Classification Module* (Statistics Denmark 2023d) and the *Employment Registry* (Statistics Denmark 2023e). Additionally, education data are obtained from the *Education Registry* (Statistics Denmark 2023m).

We obtain data on criminal offenses from the *Danish Central Crime Registry* maintained by the Danish National Police (Statistics Denmark 2023h and Statistics Denmark 2023g for convictions, Statistics Denmark 2023j for charges and Statistics Denmark 2023i for imprisonments). 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* (Statistics Denmark 2023k) and from the *Cause of Death Registry* (Statistics Denmark 2023a and Statistics Denmark 2023b). 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.

³At older ages, individuals can choose to go into early retirement, depending on contributions, either at age 60 through the Voluntary Early Retirement Pension (VERP), or depending on the time period, through an old-age pension at ages 65–67.

All monetary values are expressed in nominal Danish kroner inflated to 2018 prices following the Danish national inflation index ([Statistics Denmark 2023I](#)), 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 reports that the average individual in our sample is 48 years of age, has 13 years of education, and has a total income of DKK 313,255 (\$50,525) 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 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 over our sample period.

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. Table I shows that the average probability of being convicted of a crime in a given year for the people in our sample is 0.69%. We further classify crimes into *Economic crime* or *Non-economic crime* based on whether they are likely to be economically motivated or not, and into *Property crime*, *Sexual crime* or *Violent crime*. Online Appendix Table I.I illustrates how the different types of crime map into these categories and reports the crime conviction 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. Online Appendix Figure I.1 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 et al. 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 use 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. 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. Our identifying assumption is that the exact timing of the cancer diagnosis

is unpredictable, conditional on time 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 the timing of the cancer diagnosis (see Online Appendix Table I.II). Third, we compare key observables for individuals who will be diagnosed in 1 year versus in 10 years. We find that these individuals are similar in terms of the distribution of key covariates when accounting for time trends, age, and gender (see Online Appendix A, panel a of Online Appendix Table I.III, and Online Appendix Figure I.2).

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 6.1, we confirm that our results are similar when we impose that treatment and control observations are diagnosed exactly Δ years apart where Δ varies from 3 to 8. (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. Finally, Online Appendix A documents that same-age individuals who are diagnosed 9 years apart are highly comparable before cancer, thereby lending support to our choice of utilizing variations in the time of diagnosis for identification.

Online Appendix Section B examines the raw crime data. In particular, Online Appendix Figure I.3 shows the average crime rates in event years for treated, yet-to-be-treated, and a randomly drawn sample of never-treated individuals. Event year 0 corresponds to the cancer diagnosis for the treated group and to the average age when people are diagnosed with cancer for the other two groups. There are two insights we draw from this graph. First,

⁴Consider 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.

⁵Comparison 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).

this figure presents the first evidence of the positive effect of cancer on crime. Comparing crime rates for treated and yet-to-be-treated, we observe the latter declining both before and after event time 0. By contrast, crime rates for treated individuals decline before and during the diagnosis year but flatten out after that. Second, this figure shows similar crime trends before diagnosis for treated and yet-to-be treated individuals. However, people who never develop cancer are on a slightly different criminal trajectory, which is potentially consistent with the conjecture that they are *ex ante* different in terms of unobservables. This finding supports our choice of using yet-to-be treated individuals as controls, rather than never-treated individuals.

3.2. Baseline specification

We estimate a 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 research 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 2024](#), [De Chaisemartin and D’Haultfœuille 2020](#), [Goodman-Bacon 2021](#), [Sun and Abraham 2021](#)). To overcome this issue, we estimate a specification with a full set of post-treatment variables. Under the assumption of 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:

$$C_{i,t} = \alpha_i + \beta_{t,a} + \sum_{\substack{\tau=-6 \\ \tau \neq -1}}^{10} \gamma_{\tau} \mathbb{1}\{T_{i,t} = \tau\} + \lambda X_{i,t} + \epsilon_{i,t}, \quad (1)$$

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 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 deter-

minants (e.g., personality, IQ, genetic heritage, childhood experiences). Year-by-age fixed 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).⁶ 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.

We empirically test the parallel trend assumption by including a set of lead indicators in Equation (1). Notably, we need to exclude at least two lead variables to avoid multicollinearity (see [Borusyak, Jaravel and Spiess 2024](#)). We omit the event year before treatment ($\tau = -1$) and a number of leads distant from the treatment ($\tau < -6$).

We 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 period. For each specification, we report the ATE and the relative treatment effect (RTE)—the ATE scaled by the average crime rate across all event years within the specific sub-population for which the treatment effects are being estimated—in the bottom right corner of each figure.

4. MAIN RESULTS

4.1. *The effects of cancer on crime*

Figure 1 reports the estimates for the effect of cancer on crime. The coefficients estimated using Equation (1) are plotted over the event years. 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.

⁶Notably, 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).

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 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 estimate a value of 0.10 percentage points (significant at the 1% level): cancer patients are thus 14% 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. Importantly, we find that there are no pre-trends, as evidenced by the statistically insignificant lead coefficients.⁷ In Section 6, we consider that criminal ability might decrease after cancer, evaluate the impact of attrition, and employ never-treated individuals as controls.

4.2. *First crimes and re-offenses*

Do individuals with a clean record start violating the law because of cancer? To test this hypothesis, we run a specification that replaces our baseline crime variable with a first-time crime indicator (*First Crime*), which equals one if a crime was committed for the first time, and zero otherwise. Panel a of Figure 2 shows that after a cancer diagnosis individuals are 0.04 percentage points (14%) more likely to commit their first infraction.

We further test how the cancer diagnosis impacts the propensity to re-offend. To that end, we run a specification that replaces our baseline crime variable with a re-offense in-

⁷We 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). Furthermore, we run a host of robustness checks in Online Appendix C where we: i) explicitly estimate the coefficient $\tau = -1$; ii) allow for the possibility of heterogeneous treatment effects by estimating separate coefficients for different cohorts (following Sun and Abraham 2021); iii) estimate lead coefficients using untreated observations only (following Borusyak, Jaravel and Spiess 2024); iv) use the procedure of De Chaisemartin and d’Haultfoeuille (2024) to estimate placebo treatments. In all cases, we find no evidence of differential trends prior to the cancer diagnosis. These findings validate our empirical design and mitigate concerns that individuals in our sample anticipate cancer diagnoses.

indicator (*Reoffense*), which equals one if a crime was committed that was not a person's first crime, and zero otherwise. The results, shown in panel b of Figure 2, indicate that the probability of a re-offense increases by 0.06 percentage points (14%) following diagnosis. Overall, we find that health shocks significantly increase both first crimes and re-offenses, with both effects exhibiting comparable magnitudes.

5. WHY DOES CANCER PROMPT CRIME?

Guided by the theoretical framework that we develop in Online Appendix D, 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 of human capital by seeking illegal revenues (economic mechanism). Furthermore, decreased survival probabilities might increase time discounting and therefore reduce the expected cost of future punishment (survival probabilities mechanism). Finally, cancer may induce changes in risk preferences or induce psychological distress (alternative mechanisms), potentially contributing to an increased propensity to engage in criminal behavior. We explore these mechanisms in detail below. 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*

Unforeseen health shocks can adversely impact both income and employment.⁸ These negative shocks may raise criminal propensities as individuals compensate the loss of legal income with illegal income. We investigate this economic mechanism by i) exploring the effects of cancer on labor market outcomes, ii) separating the effect of cancer on different types of crimes, and iii) estimating heterogeneity in the treatment effects by socio-economic background.

⁸An extensive literature on health shocks and their impact on labor market outcomes relies both on survey-based investigations (e.g., Charles 2003, Dobkin et al. 2018b, Gallipoli and Turner 2011, Gertler and Gruber 2002, Meyer and Mok 2019) and larger-scale studies linking administrative health and labor market data (e.g. Fadlon and Nielsen 2021, García-Gómez et al. 2013, Gupta, Kleinjans and Larsen 2015, Halla and Zweimüller 2013, Lundborg, Nilsson and Rooth 2014). A number of studies specifically focus on the labor market effects of cancer, see, e.g., Heinesen and Kolodziejczyk (2013), Jeon and Pohl (2019), and Moran, Short and Hollenbeak (2011).

We begin our analysis by exploring the effect of cancer on labor market outcomes by replacing the main dependent variable in Equation (1) with i) salary income, ii) total income (salary, income from self-employment, and government transfers), iii) employment, and iv) the number of hours worked. We report the results in Figure 3. Salary income and total income decline by DKK 12,460 (−4.8%) and DKK 4,990 (−1.5%), respectively (panels a and b).⁹ The probability of employment, defined as having salary income above DKK 50,000 in a given year, falls by 1.5 percentage points the year of diagnosis, with an average effect of −0.9 percentage points (−1.0%) (panel c), indicating that some cancer patients permanently leave the labour force after diagnosis. Conditional on remaining employed, the number of hours worked annually decreases by 40 hours in the diagnosis year (panel d), and then increases progressively over the following 10 years to revert back to pre-diagnosis levels. Overall, we find a detrimental impact of cancer on labor market outcomes, which creates an economic incentive to violate the law, especially the first years after diagnosis.

Building on the findings above, we further investigate whether the surge in crime can be explained by economic motives. We separate the broadest definition of crime into two narrower categories: *Economic crime* and *Non-economic crime*. The former includes crimes that are typically motivated by economic factors (e.g., theft, burglary, or drug dealing). The latter category comprises offenses that are less likely to be influenced by monetary incentives, such as sexual violence or vandalism. We also employ a second classification framework by 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. Our empirical design is motivated by the following consideration: if the documented effects were exclusively attributable to economic motivations, the impact should manifest primarily in an increase in economic or property crimes, with non-economic crime rates remaining unaffected after the cancer diagnosis.

The results in panel (a) of Figure 4 document an increase in both economic and non-economic crimes. We find a 0.06 percentage point increase (14%) in economic crimes

⁹Based on the estimates presented in Figures 1 and 3, the implied elasticity of crime to income appears implausibly high. This arises from the fact that the income decline is not the sole channel at play in our setting. We show in Section 5.2 that the survival probability channel also plays a significant role.

versus a 0.02 percentage point increase (38%) in non-economic crimes.¹⁰ In Panel (b) of Figure 4, we find that property and violent offenses increase significantly by 0.06 and 0.02 percentage points (15% and 21%) respectively, while the incidence of sexual offenses does not change significantly after the cancer diagnosis. In Online Appendix Figure I.5, we further separate crimes based on the associated sentences.¹¹ We document that cancer patients do not commit crimes that carry the most severe sentences (e.g., homicide, human trafficking, aggravated robbery, terrorism) but rather crimes that result in less than one year of prison time or fines (such as theft, minor assault, vandalism, or fraud).¹²

Next, we explore the heterogeneity in responses to cancer on the basis of the socioeconomic and demographic background of the cancer patient. Figure 5 shows that the treatment effect is positive and significant both for individuals with above and below median total income in the year before diagnosis. While people with low income face a greater increase in criminal propensity in absolute terms, the relative increase is larger for high-income households. Although this result may seem surprising, this is consistent with social benefits that are capped in Denmark (see Section 2.1 for details). As government transfers only cover income losses up to a certain threshold, high-income households face larger declines in their total income following diagnosis. Consequently, the economic channel might be more relevant for this group of individuals.

We further find that the increase in criminal activity is driven by individuals who do not own a home, are single, low-educated, and men.¹³ By contrast, we find no significant effect for women.¹⁴ This finding is consistent with previous papers that argue that men are more likely to commit crime in response to life-changing events, such as criminal victimization, divorce, and job loss (Broidy 2001, Kaufman 2009). Such different response could stem

¹⁰Online Appendix Figure I.4 shows results using an alternative non-economic crime measure where all previously unclassified crimes are labelled as non-economic. The results are significant, but the RTE is smaller.

¹¹Felonies are crimes for which the potential jail or prison sentence exceeds one year. Misdemeanours are crimes that result in a jail term of less than one year, but more than 15 days. Short sentences or fines are crimes resulting in fines or a prison sentence shorter than 15 days.

¹²We further show the relation between cancer and the eight most common types of crime in Online Appendix Figure I.6. While all eight types of crime increase following cancer, we often lack sufficient statistical power to detect significant effects.

¹³Home-ownership and marriage can potentially provide a cushion against the adverse financial impact of crime (in line with Gupta et al. 2018).

¹⁴In our sample, most crimes are committed by men (71% versus 29% for women), which is in line with other studies conducted in different settings (see Kruttschnitt 2013 for a review).

from differences in coping mechanisms, access to support networks, and economic opportunities, all of which can influence how individuals, based on their gender, navigate and respond to the challenges posed by health shocks and other stressors.

Furthermore, we find significant results for both younger and older individuals, and similar effects for individuals who have and have not had previous exposure to crime through a family member who violated the law (parent, sibling, partner, child, or in-law).

We show that the individuals who have the strongest crime-cancer relation also tend to experience the largest decline in total income. In particular, Online Appendix Figure I.7 presents heterogeneous effects of cancer on total income for people with different socioeconomic and demographic characteristics. It is evident that the groups who experience the strongest effects on their income are more likely to commit crimes after cancer, suggesting that individuals whose human capital is affected the most seek additional revenues from the illegal labor market. For example, the results show that low-educated, single men face the largest decline in income. These characteristics also relate to a stronger increase in criminal propensity following cancer, as reported in Figure 5.

Overall, our results suggest that the decline in human capital following cancer is important 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.

While the impact on economic and property crimes is considerable, we also observe a substantial increase in non-economic crimes, especially violent offenses. This suggests that, while the economic motive is important, there are additional factors at play. In the following sections, we explore additional channels that are likely relevant.

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 D).¹⁵ 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.¹⁶ We then conduct our analysis separately on five subsets of individuals based on quintiles of their survival probability decline at diagnosis. Importantly, we rely on different thresholds by gender to define the quintiles, resulting in an equal share of men and women in each group. 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.¹⁷ Online Appendix Table I.IV shows that people in the first quintile face a decline in survival probability of 6% compared to an expected survival probability decline of 67% for those in the fifth quintile. We discuss the procedure for the estimation of survival probabilities in detail in Online Appendix E.

We estimate our main regression specification for each quintile of the survival probability decline. Figure 6 shows that impact of cancer on criminal behavior is stronger among individuals who experience a more substantial decrease in survival probability in the year of diagnosis. In particular, the estimated ATEs are small and statistically insignificant for quintiles 1 and 2, but 0.12 and 0.14 percentage points and significant for quintiles 3 and 4, respectively. The treatment effect for people who suffer the biggest decline in survival probability (quintile 5) is positive but smaller (0.08) and only significant at the 10% level. The latter result is not surprising, since individuals diagnosed with the deadliest cancer

¹⁵Lower quality of health care could lead to an increase in the expected punishment of crime after a cancer diagnosis. However, inmates within prison facilities in Denmark have the right to healthcare services similar to individuals in the broader society. The equal quality of health care is governed by the Danish law (Danish Health Act, § 2). Accordingly, we should not expect a prison sentence to influence the probability of dying from a cancer diagnosis.

¹⁶We consider a five-year period because this is standard in the medical literature.

¹⁷There are other differences in demographics between quintiles when we sort by survival probabilities. However, these differences are not large enough to explain our findings.

types typically remain hospitalized or sick for a long period, which naturally limits their ability to commit crimes.

In Online Appendix Figure I, we perform the same analysis on two subsamples split based on the median survival probability decline. We find that people with an above median decline in survival probability significantly increase crime (at the 1% level) while people who face a below median decline do not significantly increase crime. Overall, 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.

5.3. *Other mechanisms*

We explore two additional mechanisms both outlined in detail in Online Appendix F. First, we explore whether cancer leads to a change in risk attitudes. The analysis is conducted by linking registry data with experimental individual-level data from incentivized risk attitude experiments conducted in 2003 ([Andersen et al. 2008](#)), 2009 ([Andersen et al. 2014](#)), and 2020 ([Andersen et al. 2024](#)). Analyzing pooled cross-sectional data for individuals diagnosed with cancer (treatment group) and those yet to be diagnosed (control group), Online Appendix Table I.V reveals no significant relation between cancer and risk aversion among the 58 individuals considered.

Second, Online Appendix Table I.VI explores whether part of the uptick in crime could be explained by psychological distress. Namely, cancer could prompt crime through an effect on a person's mental health. In line with this hypothesis, 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 this section we explore whether welfare policies play a role in mitigating the impact of health shocks on crime. As we find substantial effects of cancer on labor outcomes, a natural question is whether compensating cancer patients for the loss of income can mitigate the impact on criminal behavior.

In Denmark, social policies are administered at the municipality level, whereas health treatment is organized at the regional level. Municipal authorities can provide cancer patients with sickness benefits, pay permanent disability subsidies, allow early retirement, and conduct policies to reintegrate people into the labor force. The decentralization of welfare policies implies that people who face the same health shock will experience economic hardship differently based on where they reside. However, as the choice of where to reside is itself endogenous, the presence of local heterogeneity is not sufficient in itself to identify whether welfare policies mitigate the adverse effect of cancer on crime.

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 1st, 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. The reform included local changes in domains such as social services, transportation, roads, and employment amongst others. We take advantage of the reallocation of decisional authority on social matters across the country to explore whether social policies can mitigate the effect of health shocks on crime.

We conduct the analysis in two steps. First, we measure the municipality-level change in income support to cancer patients induced by the reform, while acknowledging that this change should be interpreted as an instrument for other changes as well. Second, we explore how the sensitivity of crime to cancer differs depending on the change in income 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 G).

Overall, we find that 163 of 271 municipalities (60%) cut social support after the reform, while the remaining 108 (40%) left it unchanged or increased it. The average change in income replacement among patients experiencing a decline is -2%, while the 10th and 90th percentiles are -5% and -0.3%, respectively. The average change among patients experiencing an increase is 2%, while the 10th and 90th percentiles are 0.1% and 4%, respectively. Figure 7 illustrates the geography of the change in generosity across municipalities: we document a wide geographical dispersion.

We explore how the change in welfare benefits impacts the incentive to commit crime. To that end, we separate municipalities based on whether we estimate a decrease in income replacement post-reform for cancer patients residing there. We then compare criminal behavior of diagnosed and yet-to-be diagnosed individuals in municipalities negatively impacted by the reform (“stingy municipalities”) with respect to municipalities that were positively or not impacted by the reform (“generous municipalities”). Importantly, our empirical design assumes that other policies that came into effect because of the reform (e.g., changes in child care support) affect in a similar way cancer patients and yet-to-be diagnosed individuals residing in the same municipality.

We rely on the following specification:

$$C_{i,t} = \alpha_i + \beta_{t,a,g} + \sum_{\substack{\tau=-6 \\ \tau \neq -1}}^{10} \theta_{\tau} (\mathbb{1}\{T_{i,t} = \tau\} \times S_{t,m}) + \delta S_{t,m} + \sum_{\substack{\tau=-6 \\ \tau \neq -1}}^{10} \gamma_{\tau} \mathbb{1}\{T_{i,t} = \tau\} + \lambda X_{i,t} + \epsilon_{i,t}, \quad (2)$$

where $S_{t,m}$ is an indicator variable that takes a value of one from 2007 onwards for stingy municipalities, defined as municipalities where the income replacement for cancer patients decreases after the reform. The municipalities are indexed by m . The coefficients γ_{τ} measure the average effect of being diagnosed with cancer on crime in untreated municipalities. The coefficients of interest, θ_{τ} , capture the *additional* effect of being diagnosed with cancer on crime for cancer patients residing in municipalities that decreased support. Notably, with this empirical approach we identify causal effects from variations in crime levels from both the time dimension (before versus after the reform) and the cross-sectional dimension (municipalities that reduced welfare versus municipalities that did not reduced welfare). $\beta_{t,a,g}$ are year–age–municipality’s generosity fixed effects, where g identifies whether municipalities are stingy or generous. These fixed effects allow for people of the same age to be on different crime rate trajectories depending on whether they live in municipalities that cut welfare support or not.

Figure 8 presents the coefficients θ_{τ} . The figure indicates that the effect of cancer on crime is larger for the individuals living in municipalities that cut social support. Specifically, the additional ATE is 0.08 percentage points. We find similar results when classifying

municipalities in four groups based on the change in income replacement rates due to the reform (see Online Appendix Figure I.9). 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 address the economic consequences of health shocks are important in mitigating the resulting impact on crime.

Overall, our analysis provides evidence for three channels through which cancer leads to crime. First, several results suggest a role for economic motives. In particular, the cancer-crime relation is stronger for people lacking spousal support and home equity, both factors that can provide a form of insurance. Furthermore, changes in social security benefits alter the propensity to commit crime following cancer. However, we find evidence for other channels as well. When examining the types of crimes committed, we find an increase in both economic and non-economic crimes. Furthermore, the adverse economic impact of cancer diminishes over time while the propensity to commit crime remains high.¹⁸ The second mechanism that emerges is a decline in survival probability, as people that face stronger declines are more likely to commit crime. However, it is difficult to assess the relative importance of these two channels, as the individuals who face the most severe economic consequences tend to also experience the largest decline in survival probability. Finally, we also find some evidence which is consistent with the presence of a psychological distress channel.

6. ROBUSTNESS

6.1. Attrition

Attrition from mortality is substantial in our study. Online Appendix Figure I.10 shows approximately 13% annual attrition due to death during the diagnosis year and the first year after diagnosis, declining to 3%-5% in subsequent years. This can lead to biased coefficients if mortality is correlated with crime. We address this concern in several ways.

First, by incorporating individual fixed effects in Equation (1), our analyses focus solely on variation *within* individuals, which ensures that the treatment estimates are not contaminated by differential attrition correlated with crime propensity. However, Figure 5 shows

¹⁸This pattern can also emerge because cancer patients undergo intense medical treatments, which may limit their ability to commit crime in the first few years after diagnosis, or because social support and accumulated savings may buffer the effect on income in the short term.

that the treatment effects vary with individual characteristics, and thus changes in the sample composition over time can bias our coefficients as the sample to identify coefficients at different event dates changes. To assess in which direction this biases our coefficients, we examine the changes in the composition of our sample due to attrition.

Online Appendix Table I.VII compares the crime propensity and socioeconomic characteristics at event date $\tau = -1$ for (1) people that survive and (2) people who die in the 10 years post diagnosis. We find that low-educated men and those with a higher crime propensity the year before diagnosis are more likely to exit the sample. These are characteristics related to a larger treatment effect. By contrast, people with higher survival probabilities—who are more likely to remain in our sample—tend to have smaller or statistically insignificant treatment effects. Therefore, the changing sample composition due to attrition is likely to lead to an underestimation of the average treatment effect.

Furthermore, Online Appendix Figure I.11 shows that our results are similar when using a balanced sample with a fixed time interval Δ between treated and control observations where Δ varies from 3 to 8 (following the approach of [Fadlon and Nielsen 2019](#)). Using this approach, the crime choices of individuals diagnosed with cancer at time s (treatment group) are compared to those who are diagnosed with cancer at time $s + \Delta$ (control group). Individuals in the control group are assigned a placebo shock at time s , since they are actually diagnosed with cancer only at time $s + \Delta$. We can then estimate the effect of cancer on crime for $\Delta - 1$ time periods using a difference-in-differences estimator (see details in Online Appendix Section H). This analysis allows us to assess the impact of changes in comparability between control and treated observations as well as that of attrition. We find that the coefficients remain similar when Δ increases, thereby limiting concerns that attrition and/or lack of comparability drive our estimates.

Online Appendix Figure I.12 shows how the estimates change when we restrict our sample to individuals who are alive at least x years post-diagnosis, where we vary x from one to ten. In this analysis, we estimate all event-time coefficients but present only those for which everyone is still alive. Notably, by restricting our main sample to individuals who survive cancer, we bias the analysis against finding an effect, as we exclude the majority of individuals who contribute to the rise in crime (since the treatment effect is driven by individuals experiencing a high decline in survival probability; see Section 5.2). To counterbalance this effect, we run this analysis exclusively on individuals who face an above second-quintile survival probability decline at diagnosis, that is, on individuals that are relevant in our con-

text. In this way, we decrease the noise in our estimations by excluding people who are ex-ante unlikely to increase their criminal propensity but are overrepresented in the data when we impose the condition that everyone in the sample is still alive at event time x . Online Appendix Figure I.12 shows that the estimated coefficients remain consistent across specifications, further alleviating concerns that our results are due to attrition.

Finally, we evaluate the impact of attrition on the magnitude of our estimates by comparing Online Appendix Figure I.12 panel (j), which is estimated on a fully balanced sample, to Online Appendix Figure I which is estimated on the main (unbalanced) sample. The treatment effects on the fully balanced sample are large and statistically significant, with an estimated ATE of 0.09 and a RTE of 16%. The treatment effects on the unbalanced sample are however larger in magnitude, with an estimated ATE of 0.14 and a RTE of 21%. All in all, these results suggest that attrition has a moderate impact on the estimated effects.

6.2. *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.

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 9). Second, we test if there is a 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 I.VIII). We include only observations in the year of offense and estimate an insignificant effect of cancer on time to apprehension both jointly and for each event time separately. Third, we compute the percentage of reported crimes that remain 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 disproportionately more likely to be apprehended (see Online Appendix Table I.IX).

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 Massenkoff and Rose 2024). Figure 9 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.

We also estimate a specification using never-treated individuals as an alternative control group. Online Appendix Figure I.13 shows a statistically significant but economically smaller treatment effect. This smaller effect can be explained by considering the patterns in Online Appendix Figure I.3 which displays raw crime rates for treated, yet-to-be-treated, and never-treated individuals. The figure illustrates that crime declines more rapidly with age for treated individuals compared to those who are never treated. In the specification using never treated as controls, treatment effects are calculated relative to the crime trend of the never-treated group. Consequently, the estimated treatment effects (calculated as $ATE = ATT - ATC$) are smaller when using never-treated individuals as the control group, rather than using individuals who are yet-to-be treated as controls. This evidence suggests that never-treated individuals are on a different crime trajectory pre-event date and are, therefore, less suitable as a control group in our setting.

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 0.11 percentage points more likely to be charged with a crime (an increase of 17%). This RTE is slightly larger than the RTE we estimate for convictions, which could indicate some leniency in the judicial system when sentencing cancer patients.

6.4. *Heterogeneous treatment effects by year-of-diagnosis cohorts*

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 I.14 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’Haultfoeuille (2020), De Chaisemartin and d’Haultfoeuille (2024). This methodology recovers the event study coefficients as weighted averages of DID estimators.¹⁹ Results remain qualitatively similar (see Online Appendix Figure I.14 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 the average treatment effect of the (placebo) cancer diagnosis on crime in each random sample. We find no statistically significant effect for placebo cancer diagnoses (p-value: 0.90).

¹⁹Note that this methodology does not allow to estimate all the relative time periods included in our baseline.

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 re-offend. The documented effect is subdued in the short run but increases over time as the individual recovers from medical treatment. 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 and no home equity. When examining the types of crimes committed, we find an increase in both economic and non-economic crimes, suggesting other mechanisms are also likely to be important. Second, we find evidence that the increase in criminal activity is in part driven by individuals whose survival probabilities are impacted the most by the health shock and thus face lower expected cost of punishment. Finally, we find evidence of a psychological distress mechanism. Importantly, we provide evidence that the adverse effects of health shocks on society can be mitigated through welfare policies.

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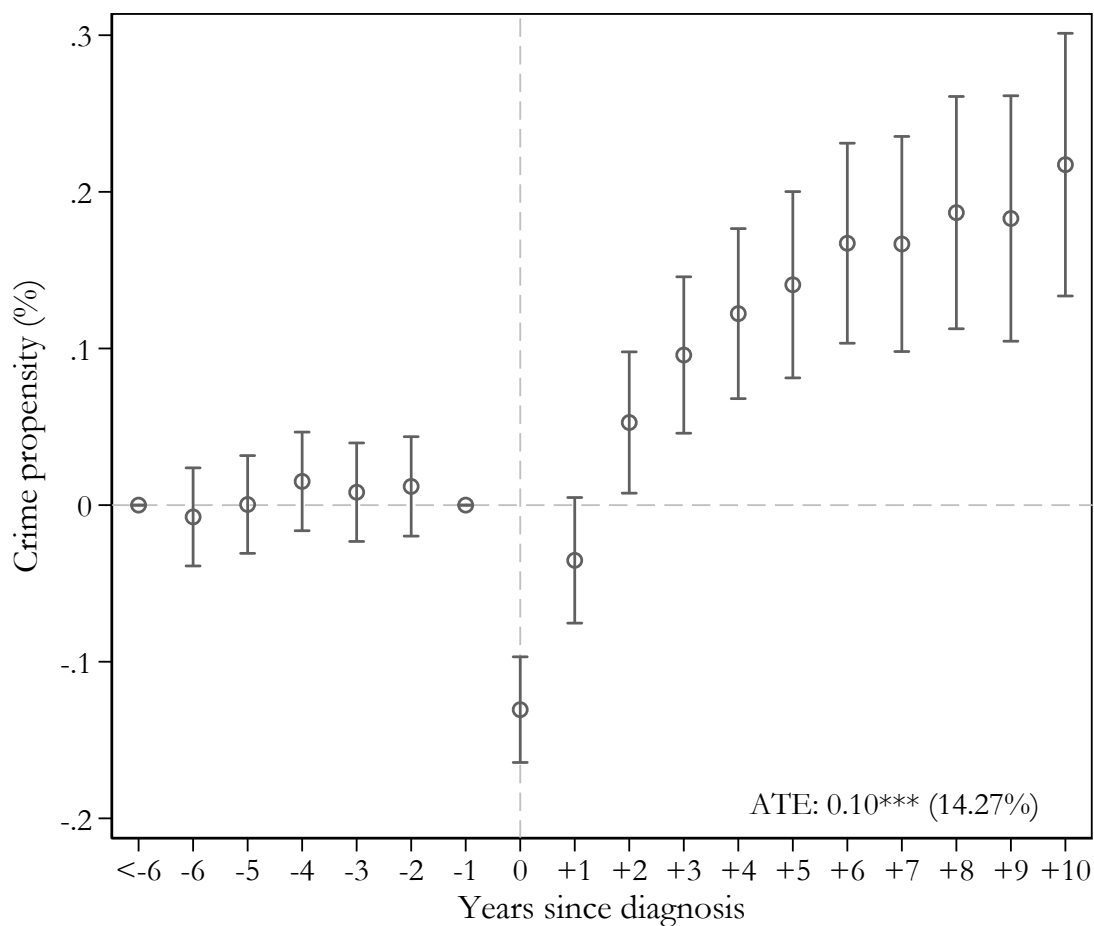
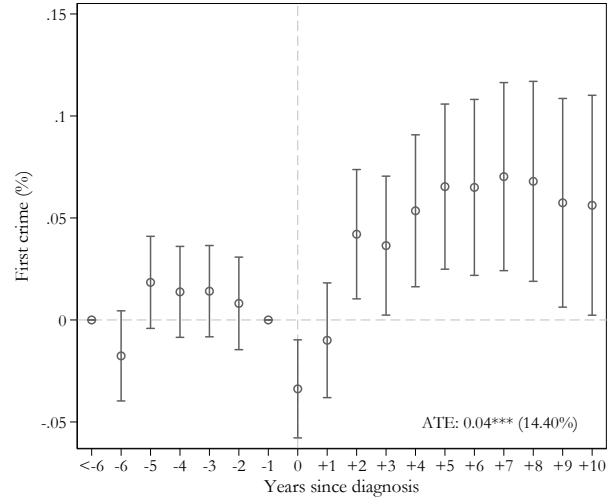
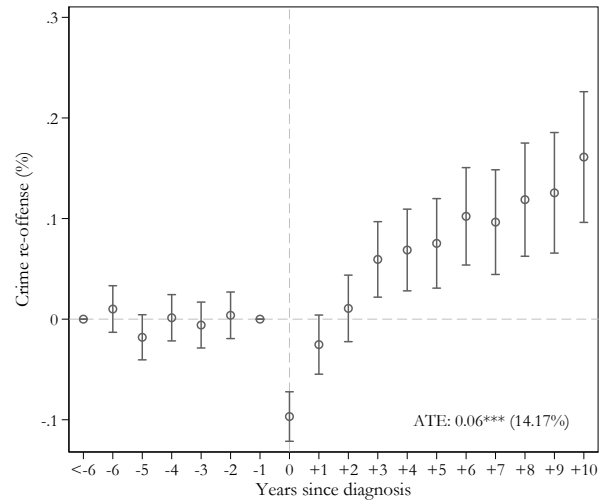


FIGURE 1.—Effect of cancer on crime. *Notes:* This figure reports event study estimates for criminal activity changes in response to cancer diagnoses. The figure plots the estimated coefficients along with their 95% confidence interval. The y-axis denotes crime propensity in percentage points. The x-axis denotes time with respect to the year of diagnosis. The average treatment effect (ATE) is reported alongside the relative treatment effect (RTE) in parentheses in the bottom right corner of the figure. The ATE is obtained as a linear combination of the treatment effects for each event year post-diagnosis, weighted by the relative size of the treatment group. The RTE is obtained as the ATE divided by the average crime rate. The empirical model includes person, year-by-age, in prison, and cancer recurrence fixed effects. The number of observations is 5,007,687. ***, **, * indicate statistical significance at the 1%, 5%, and 10% levels, respectively.

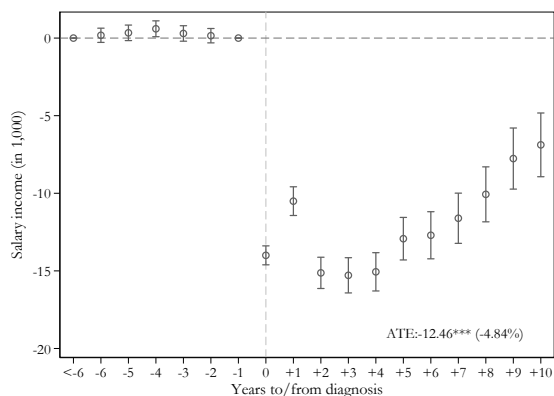


(a) First crime

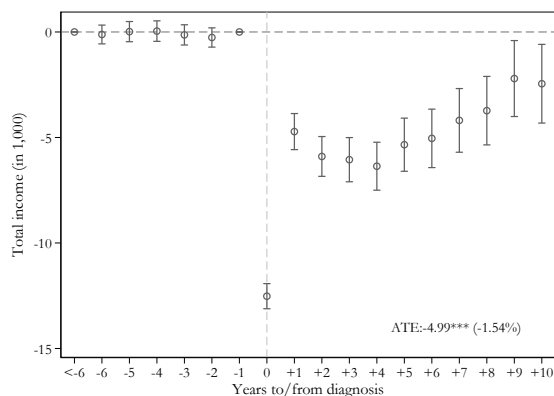


(b) Re-offenses

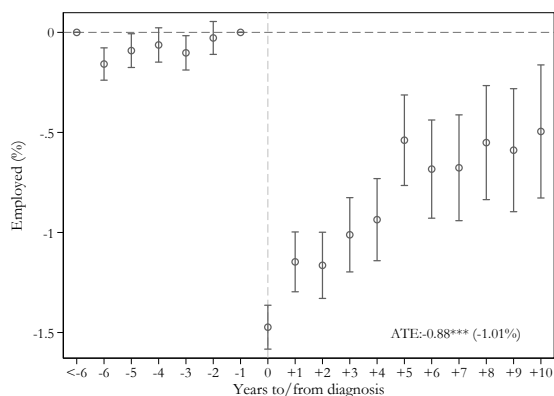
FIGURE 2.—Effect of cancer on first crimes and re-offenses. *Notes:* This figure reports event study estimates for the effect of cancer on first crimes (panel a) and re-offenses (panel b). Both figures plot the estimated coefficients along with their 95% confidence interval. The x-axis denotes time with respect to the year of diagnosis. The average treatment effect (ATE) is reported alongside the relative treatment effect (RTE) in parentheses in the bottom right corner of the figure. The ATE is obtained as a linear combination of the treatment effects for each event year post-diagnosis, weighted by the relative size of the treatment group. The RTE is obtained as the ATE divided by the average respective crime rate. The empirical models include person, year-by-age, in prison, and cancer recurrence fixed effects. The number of observations is 5,007,687. ***, **, * indicate statistical significance at the 1%, 5%, and 10% levels, respectively.



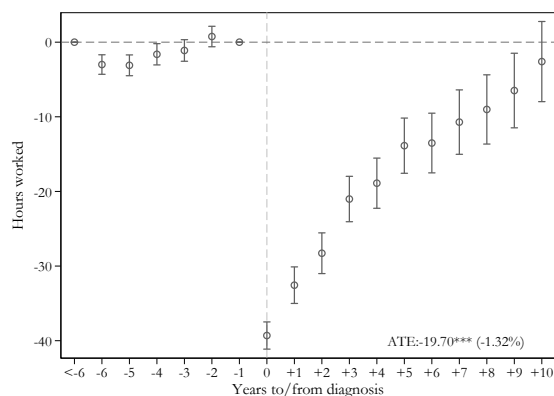
(a) Salary income



(b) Total income

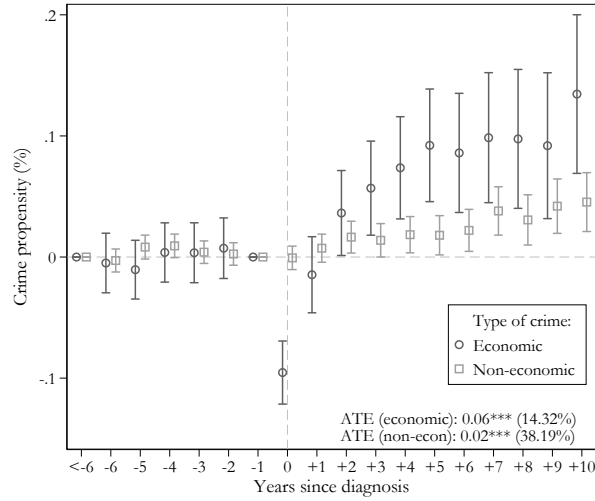


(c) Employed

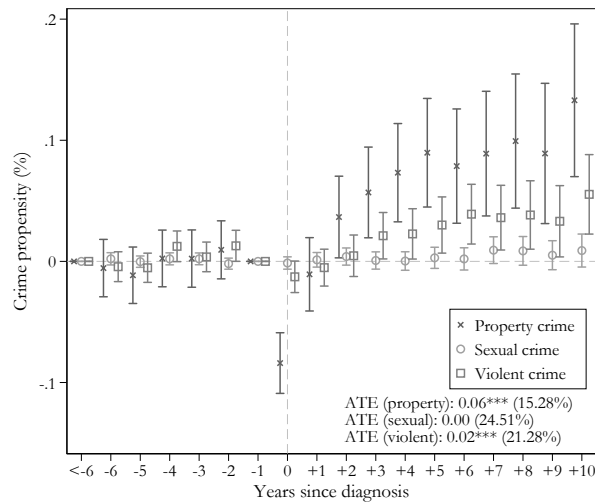


(d) Hours worked

FIGURE 3.—Effect of cancer on labor market outcomes. *Notes:* This figure reports event study estimates for the effect of cancer on labor market outcomes. The dependent variables are salary income (panel a), total income (panel b), employed (panel c), and hours worked (panel d). 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 average treatment effect (ATEs) is reported alongside the relative treatment effect (RTE) in parentheses in the bottom right corner of each figure. The ATE is obtained as a linear combination of the treatment effects for each event year post-diagnosis, weighted by the relative size of the treatment group. The RTE is obtained as the ATE divided by the average of the dependent variable. The empirical models include person, year-by-age, in prison, and cancer recurrence fixed effects. Standard errors are clustered at the person level. ***, **, * indicate statistical significance at the 1%, 5%, and 10% levels, respectively.



(a) Economic/Non-economic crimes



(b) Property/Sexual/Violent crimes

FIGURE 4.—Effect of cancer on different types of crime. *Notes:* This figure reports event study estimates for criminal activity changes for different categories of crime in response to cancer diagnoses. Panel a classifies crimes into economic/non-economic. Panel b classifies crimes into property, sexual, and violent crimes. Both figures plot the estimated coefficients along with their 95% confidence interval. The x-axis denotes time with respect to the year of diagnosis. The average treatment effect (ATE) is reported alongside the relative treatment effect (RTE) in parentheses in the bottom right corner of each figure. The ATE is obtained as a linear combination of the treatment effects for each event year post-diagnosis, weighted by the relative size of the treatment group. The RTE is obtained as the ATE divided by the respective average crime rate. The empirical models include person, year-by-age, in prison, and cancer recurrence fixed effects. Standard errors are clustered at the person level. The number of observations is 5,007,687. ***, **, * indicate statistical significance at the 1%, 5%, and 10% levels, respectively.

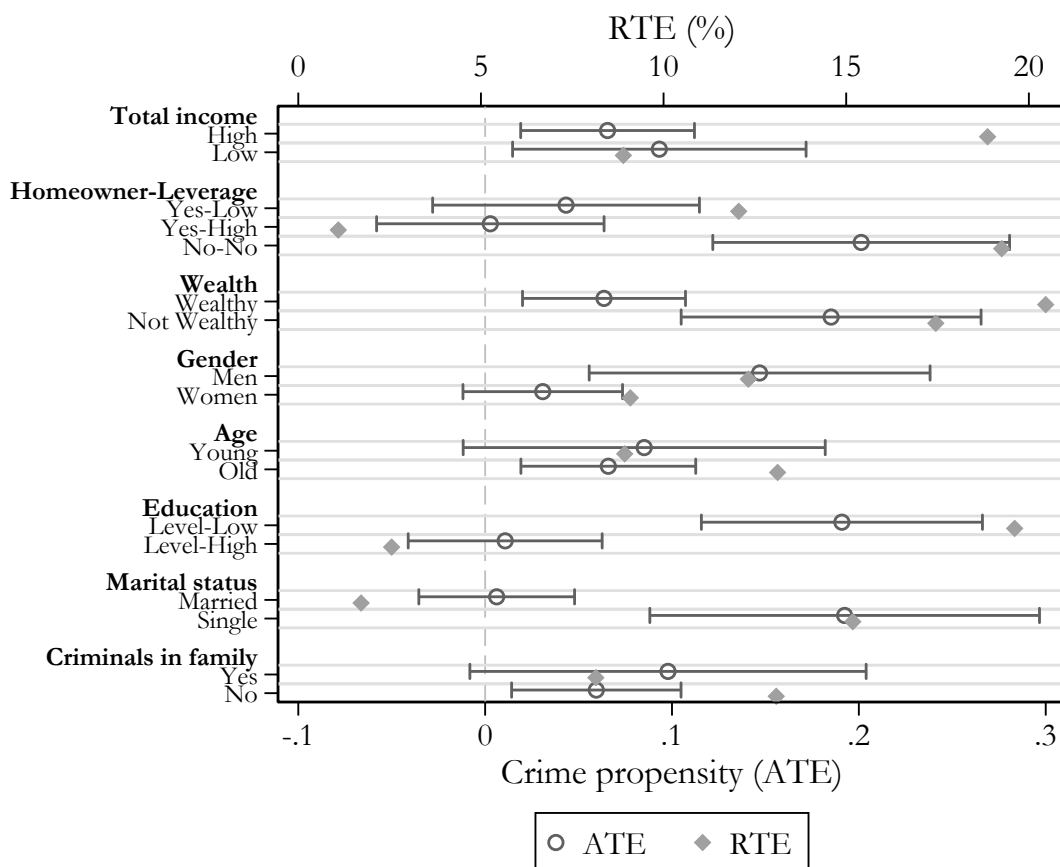


FIGURE 5.—Heterogeneous effects of cancer on crime. *Notes:* This figure reports average treatment effects for criminal activity changes in response to cancer diagnoses for different subgroups. Individuals are sorted into 1. above- (respectively below-) median income level in the year before the cancer diagnosis; 2. low mortgage-to-income ratio, high mortgage-to-income ratio, or no home equity in the year before the cancer diagnosis; 3. above- (respectively below-) median financial wealth in the year before the cancer diagnosis; 4. gender; 5. above- (respectively below-) median age in the year before diagnosis; 6. above- (respectively below-) median length of education in the year before diagnosis; 7. married (single) in the year before diagnosis; and 8. (no) criminals in the family in the year before diagnosis. The average treatment effects are indicated with circles and the relative treatment effects with diamonds. The lower x-axis denotes the ATEs and the upper x-axis the RTEs. The ATEs are obtained as linear combinations of the treatment effects for each event year post-diagnosis, weighted by the relative size of the treatment group. The ATEs are reported along with their 95% confidence interval. The RTEs are obtained as the ATE divided by the average sub-group crime rate. The empirical models include person, year-by-age, in prison, and cancer recurrence fixed effects. Standard errors are clustered at the person level.

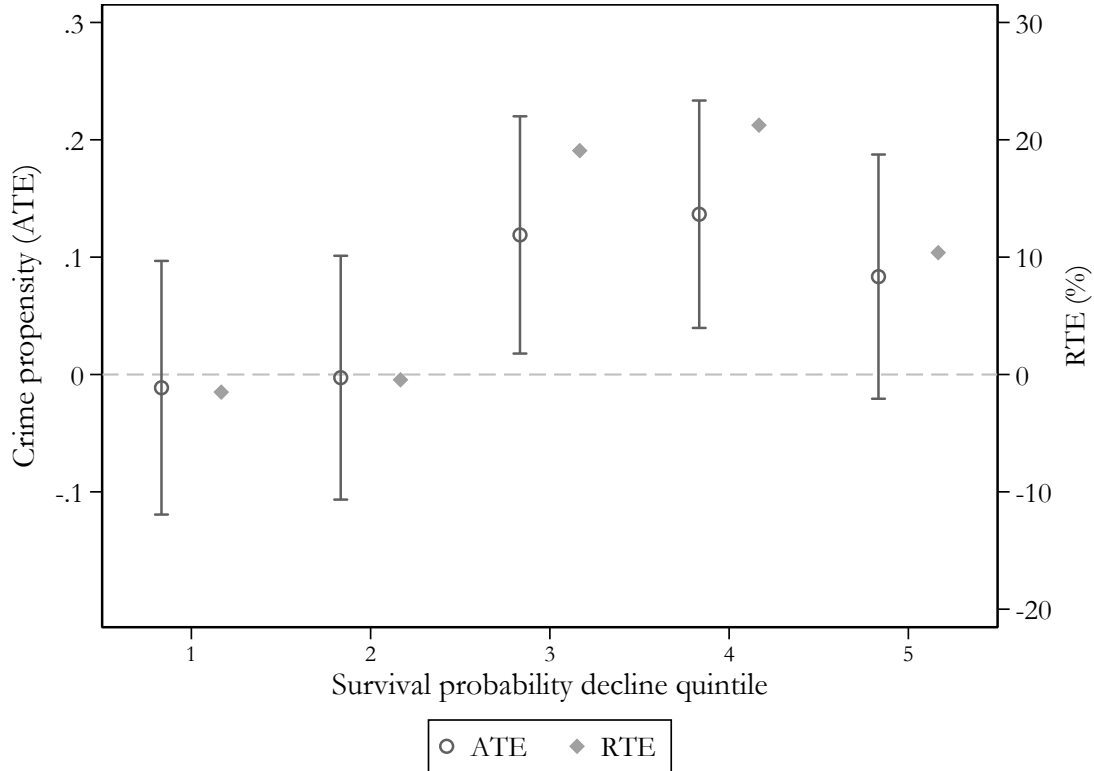


FIGURE 6.—Survival probability mechanism. *Notes:* This figure reports average treatment effects for criminal activity changes in response to cancer diagnoses for different quintiles of survival probability. The average treatment effects are indicated with circles and the relative treatment effects with diamonds. The x-axis denotes quintiles of survival probability decline due to cancer. The left hand side y-axis denotes the ATEs and the right hand side y-axis denotes the RTEs. The ATEs are obtained as linear combinations of the treatment effects for each event year post-diagnosis, weighted by the relative size of the treatment group. The ATEs are reported along with their 95% confidence interval. The RTEs are obtained as the ATE divided by the average sub-group crime rate. The empirical model is estimated separately for each group and 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.

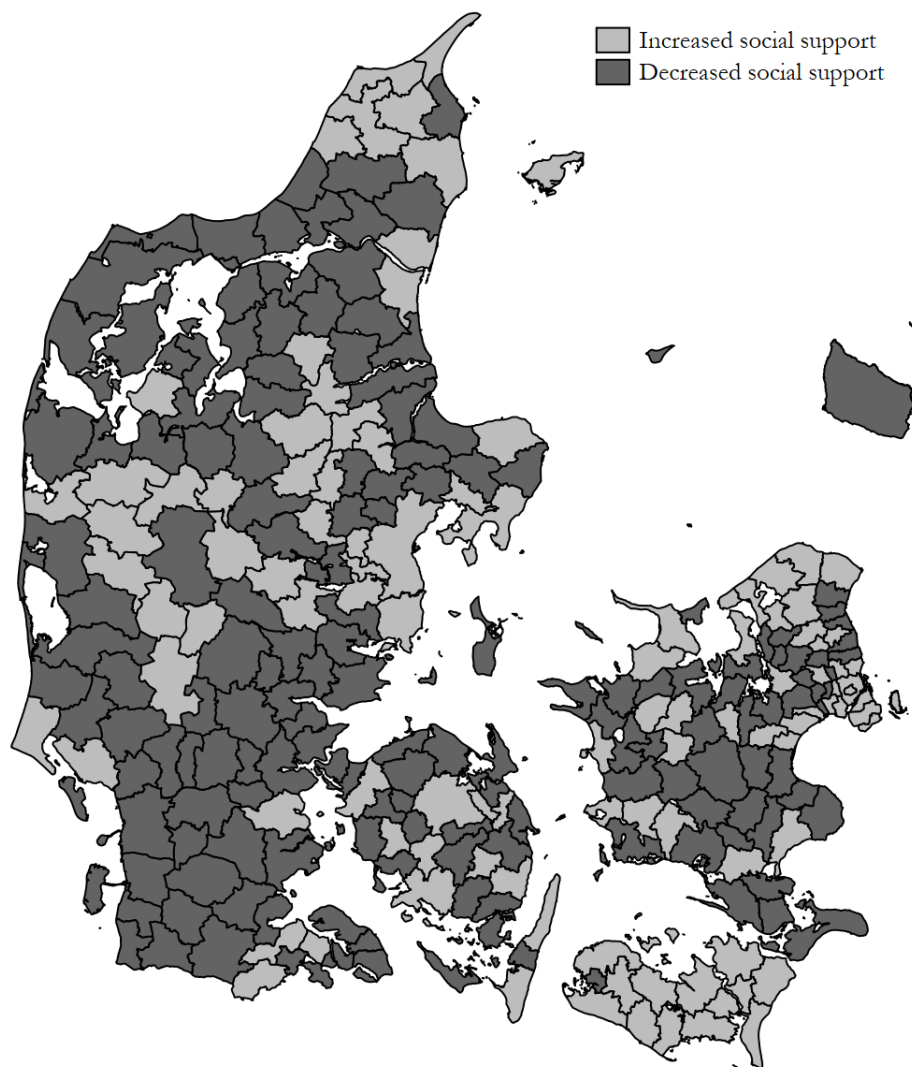
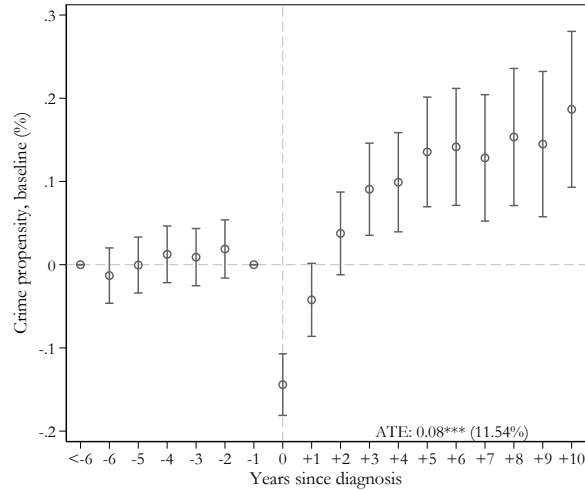
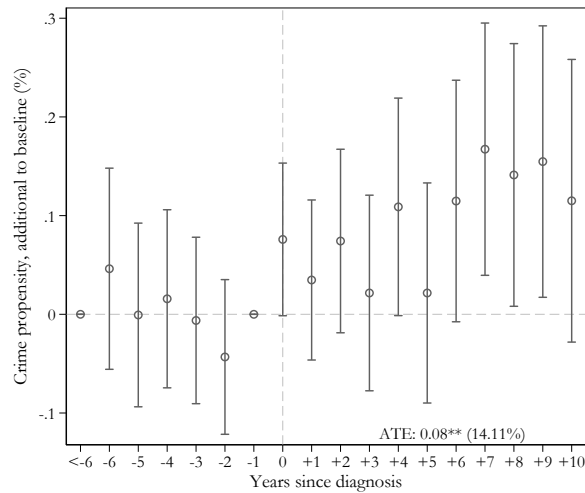


FIGURE 7.—Change in welfare generosity due to the municipality reform. *Notes:* This figure illustrates the change in generosity of Danish municipalities due to the implementation of the January 1, 2007 municipality reform. Values are obtained by estimating the change in the average income replacement for cancer patients in each municipality by comparing income replacement before and after the reform. Municipalities are colored dark (light) gray if they decreased (increased) social support for cancer patients. Details are presented in Online Appendix G.



(a) Baseline effect



(b) Additional effect from decreased social support

FIGURE 8.—Change in welfare generosity and the effect of cancer on crime. *Notes:* This figure reports event study estimates for criminal activity changes in response to cancer diagnoses using the specification in Equation (2). Panel a illustrates the baseline treatment estimates for the effect of cancer on crime. Panel b shows the additional effect of decreased social support. The figure plots the estimated coefficients along with their 95% confidence interval. The y-axis denotes crime propensity in percentage points. The x-axis denotes time with respect to the year of diagnosis. The average treatment effect (ATE) is reported alongside the relative treatment effect (RTE) in parentheses in the bottom right corner of the figure. The ATE is obtained as a linear combination of the treatment effects for each event year post-diagnosis, weighted by the relative size of the treatment group. The RTE is obtained as the ATE divided by the average crime rate. The empirical model includes person, year-by-age, in prison, and cancer recurrence fixed effects. Standard errors are clustered at the municipality level.

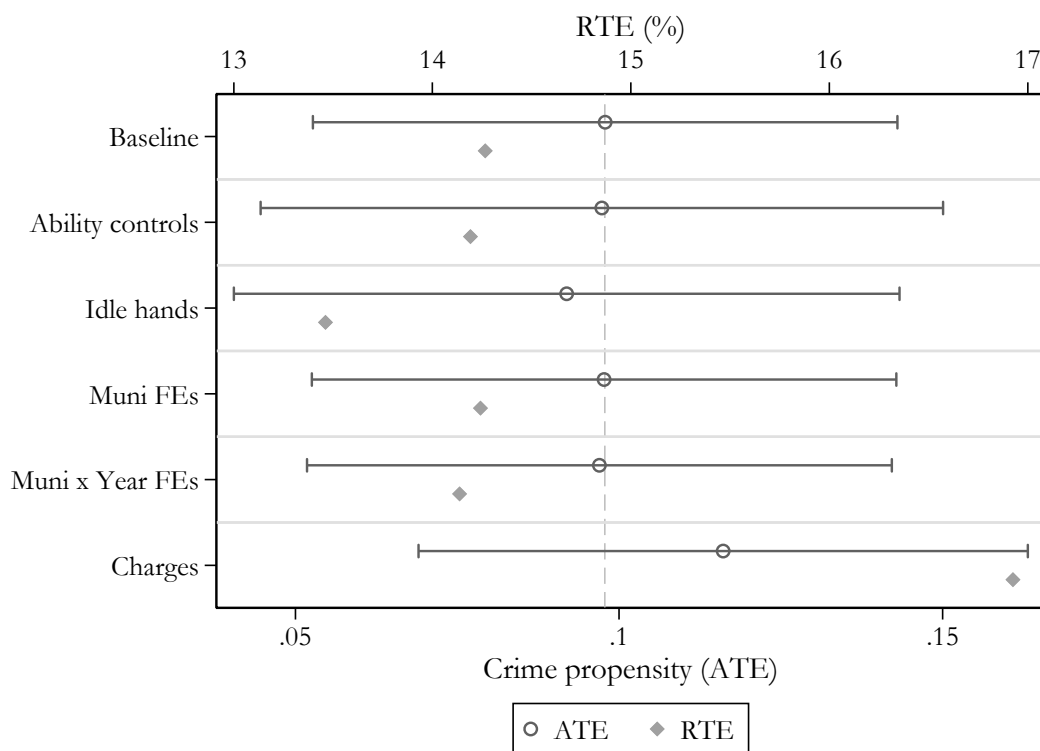


FIGURE 9.—Robustness tests for the effect of cancer on crime. *Notes:* This figure reports average treatment effects for criminal activity changes in response to cancer diagnoses. The light gray dotted line denotes the baseline ATE and RTE estimated in Figure 1, which is shown with confidence intervals 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. *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. *Muni FEs* further includes municipality fixed effects, and *Muni x Year FEs* includes municipality \times year fixed effects. *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. The average treatment effects are indicated with circles and the relative treatment effects with diamonds. The lower x-axis denotes the ATEs and the upper x-axis the RTEs. The ATEs are obtained as linear combinations of the treatment effects for each event year post-diagnosis, weighted by the relative size of the treatment group. The ATEs are reported along with their 95% confidence interval. The RTEs are obtained as the ATE divided by the average crime rate in that analysis. Standard errors are clustered at the person level.

TABLE I
SUMMARY STATISTICS^a

	Mean (1)	SD (2)
Crime (in %)	0.686	8.253
Economic crime (in %)	0.408	6.376
Non-economic crime (in %)	0.053	2.308
Sexual crime (in %)	0.014	1.170
Property crime (in %)	0.377	6.126
Violent crime (in %)	0.096	3.093
Crime charge (in %)	0.751	8.636
First crime (in %)	0.289	5.368
Re-offense (in %)	0.397	6.288
Cancer recurrence (in %)	6.191	24.098
In prison (in %)	0.193	4.391
Male	0.406	0.491
Married	0.641	0.480
Age of individual	47.763	9.656
Education in years	12.708	3.119
Home-owner	0.464	0.499
Total income (in 1,000 DKK)	313.255	185.165
Financial wealth (in 1,000 DKK)	155.668	361.051
Mortgage-to-income ratio	0.857	1.472
Doctors fees	1953.401	2695.868
Psychological fees	83.210	660.037
Physiotherapy fees	193.494	1428.462
Observations	5,007,687	
Number of individuals	368,317	

^aThis table reports summary statistics for our main sample. *Mortgage-to-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.