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DP15899

BREAKING BAD: HOW HEALTH SHOCKS PROMPT CRIME

Steffen Andersen, Gianpaolo Parise and Kim
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LABOUR ECONOMICS



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Published 11 March 2021

Submitted 08 March 2021

Centre for Economic Policy Research
33 Great Sutton Street, London EC1V 0DX, UK
Tel: +44 (0)20 7183 8801
www.cepr.org

This Discussion Paper is issued under the auspices of the Centre's research programmes:

- Labour Economics

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Abstract

We explore the impact of health shocks on criminal behavior. Exploiting variations in the timing of cancer diagnoses, we find that health shocks elicit an increase in the probability of committing crime by 13%. This response is economically significant at both the extensive (first-time criminals) and intensive margin (reoffenders). We uncover evidence for two channels explaining our findings. First, diagnosed individuals seek illegal revenues to compensate for the loss of earnings on the legal labor market. Second, cancer patients face lower expected cost of punishment through a lower survival probability. We do not find evidence that changes in preferences explain our findings. The documented pattern is stronger for individuals who lack insurance through preexisting wealth, home equity, or marriage. Welfare programs that alleviate the economic repercussions of health shocks are effective at mitigating the ensuing negative externality on society.

JEL Classification: N/A

Keywords: Economics of crime, Health shocks, Human Capital, event study

Steffen Andersen - san.fi@cbs.dk
Copenhagen Business School and CEPR

Gianpaolo Parise - gianpaolo.parise@edhec.edu
EDHEC Business School and CEPR

Kim Peijnenburg - kim.peijnenburg@edhec.edu
EDHEC Business School and CEPR

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STEFFEN ANDERSEN

Copenhagen Business School and CEPR

GIANPAOLO PARISE

EDHEC Business School and CEPR

KIM PEIJNENBURG

EDHEC Business School and CEPR

MARCH 2021

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KEYWORDS: economics of crime, health shocks, human capital, event study.

Steffen Andersen: san.fi@cbs.dk

Gianpaolo Parise: gianpaolo.parise@edhec.edu

Kim Peijnenburg: kim.peijnenburg@edhec.edu

Corresponding author: Kim Peijnenburg. For comments, we thank Nicola Gennaioli, Leontine Goldzahl, Tobin Hanspal, Glenn Harrison, Luigi Iovino, John Peijnenburg, Julien Sauvagnat, Nicolas Serrano-Velarde, Enrique Schroth, Raman Uppal, and seminar participants at Aarhus University, BI Norwegian Business School, EDHEC Business School, Goethe University Frankfurt, HEC Paris, London Business School, 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 committed by adolescents and young adults. Yet, the demographics of criminals are gradually changing in most developed countries. For example, in the United States the share of arrestees above 35 years of age has increased from 21% in 1985 to 41% in 2019.¹ Hitherto, the main emphasis of the crime economics literature has been on factors that have an early but long-lasting impact on criminal paths, such as education, family background, and opportunities upon entering the labor market (see, e.g., [Cullen, Jacob, and Levitt 2006](#) and [Heckman, Pinto, and Savelyev 2013](#)). 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 one of the most impactful and widespread type of 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, 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

¹Based on our own computations using FBI arrest statistics. Similar patterns are common in other developed countries (see, e.g., “The Rise of the Geriatric Criminal,” [CBS News 2015](#), May 29). In our country of analysis, Denmark, the percentage of crimes committed by people over 35 climbed to 35% from less than 25% in 1985.

²For 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. 2018](#), [Fadlon and Nielsen 2021](#)).

a person's overall risk attitude or perception (e.g., [Decker and Schmitz 2016](#)). In Online Appendix A, we outline these channels in a simple theoretical framework that incorporates health shocks in a model of rational criminal behavior *à la* Ehrlich.³

Rather than considering all health shocks, we focus on cancer diagnoses for three reasons. First, cancer is widespread in the population 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, at least prior to its terminal stages, often affects a person's physical condition to a lesser extent than other serious diseases (e.g., a stroke). 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 will develop cancer in later years but have not yet been diagnosed. At the same time, we include a battery of fixed effects to account for the impact of age, time, and unobservable invariants at the individual level. Essentially, our identification strategy exploits that, conditional on age, time trends, 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 13% following a cancer diagnosis (from the annual baseline crime rate of 0.68%). 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 an increase in the crime propensity of (healthy) spouses of cancer patients. A challenge for interpreting these findings stems from the fact

³Notably, 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 et al. 2018](#)), as Danes benefit from universal health insurance that covers the costs of cancer treatment.

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 these concerns. Namely, we rely on an exogenous change in the Danish welfare programs, we control for proxies of physical ability, and we show that cancer does not seem to impact the length of time the criminal avoids apprehension.

In the second part of our analysis, we seek empirical confirmation for the channels that link health shocks to crime. In line with the presence of an economic channel, 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 do not self-insure via financial wealth, home-equity ([Gupta et al. 2018](#)), education, or marriage ([Fadlon and Nielsen 2021](#)). Our analysis also confirms the existence of a survival probabilities channel: 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 channel. Specifically, we rely on risk preference estimates from two lab experiments conducted between 2003 and 2010, which we match to a subset of the 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 shows the importance of social policies in mitigating the relation between illegal activities and health shocks, even when those policies are not designed to fight crime or improve healthcare.

Our study joins an extensive literature on the economics of crime and, in particular, the body of work that explores the rational incentives to commit crime.⁴ The largest part of this literature focuses on determinants that impact the individual during childhood, including education, family background, and neighborhood environment. While we acknowledge the importance of these factors, our paper emphasizes the role of trigger events during the life course to explain the incidence of crime among adults (in the spirit of [Sampson and Laub 1993](#)).

Particularly relevant for our work is the branch of the literature that investigates how legal labor markets influence crime. Several studies use aggregate data to show a relation between labor income and criminal offenses (e.g., [Dix-Carneiro, Soares, and Ulyssea 2018](#), [Gould, Weinberg, and Mustard 2002](#), [Machin and Meghir 2004](#)). Furthermore, a recent stream of papers exploits micro-level data to investigate the effect of job loss on crime ([Bennett and Ouazad 2020](#), [Grönqvist 2011](#), [Öster and Agell 2007](#), [Rose 2018](#), [Yang 2017](#)). Yet, an examination of the effect of labor markets on crime presents some intrinsic difficulties. First, local economic shocks directly induce firm closures and crime, without the former necessarily causing the latter. Second, local shocks (e.g., plant closures) are to a large extent predictable. Therefore, high-skilled individuals tend to move to a different job before the shock occurs. Identifying a relation between labor income and crime using

⁴Related papers explore the relationship between the propensity for crime and i) its economic returns ([Draca, Koutmeridis, and Machin 2019](#), [Levitt and Venkatesh 2000](#)), ii) the likelihood of getting caught ([Ayres and Levitt 1998](#), [Di Tella and Schargrotsky 2004](#), [Draca, Machin, and Witt 2011](#)), iii) the childhood neighborhood ([Damm and Dustmann 2014](#)), iv) education and youth-targeted employment programs ([Cullen, Jacob, and Levitt 2006](#), [Deming 2011](#), [Gelber, Isen, and Kessler 2015](#), [Heckman et al. 2010](#)), v) the presence of impediments or facilitators ([Dahl and DellaVigna 2009](#), [Quercioli and Smith 2015](#)), and vi) environmental conditions such as pollution ([Grönqvist, Nilsson, and Robling 2021](#)). Closely related to our work, [Schroeder et al. \(2011\)](#) use survey data to show a contemporaneous correlation between self-reported measures of health status and crime. Furthermore, [Corman et al. \(2011\)](#) show that men are more likely to commit a crime if they have a child born with Down syndrome, congenital heart malformations, or low birth weight. See [Draca and Machin \(2015\)](#) for an excellent survey of the field.

health shocks solves this set of issues, as cancer is unrelated to local economic conditions and is hard to predict, especially the timing of the diagnosis.

Finally, our paper complements the literature on the consequences of health shocks. The conventional approach in this literature is to consider the implications of health shocks for the affected individual and her close family (e.g., [Dobkin et al. 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 critical to the design of optimal welfare policies. For example, individualistic societies may decide against policies aimed at mitigating the detrimental (but private) effects of adverse health events. However, generous social policies may be in everyone's best interest if unmitigated private shocks generate higher social costs in the form of a rise in crime rates. 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 channels. Section 6 shows the presence of heterogeneity in the response to health shocks. Section 7 presents additional robustness results and Section 8 concludes.

2. INSTITUTIONAL SETTING 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 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. These features of the Danish system provide us with a unique testing ground. Out-of-pocket health expenses—though not zero—play little role, which allows us to eliminate an almost mechanical channel through which medical expenses force cancer patients into insolvency, which, in turn, leads them to perpetrate criminal offenses.

Income insurance against severe health shocks in Denmark consists broadly of three parts. First, there is short-term sick pay and, depending on the occupation, employer-based policies (lump sum payment for critical illness). Short-term coverage is 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.⁵ 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.

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

⁵The length of the period with full pay while still sick is tied to each specific employment contract and is not directly observable in the data. In general, the economic consequences of cancer only affect people with a lag. When we estimate the average treatment effect we conservatively include the year following the diagnosis, though this—both because of inability to commit crimes due to hospitalization and cancer treatment, as well as delayed economic hardship—biases the effect of cancer on crime against finding a positive effect.

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. We use this variation in Section 5.4 to assess the role of different welfare policies. Approved applicants 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.⁶

2.2. Administrative registry data

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

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.⁷

⁶At 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.

⁷Although health care is universal in Denmark, patients can choose where to be treated. Medical treatment is to a large degree provided by public hospitals but, in the case public hospitals lack capacity to treat new patients, they can be treated at private hospitals. Private hospitals are refunded through the same administrative system as public hospitals, and the treatments and diagnoses are recorded in our data. Privately funded treatments (for example, new exploratory treatments) and treatments outside of Denmark are, however, not recorded in the registers.

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 that year, 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 earns DKK 318,857 (\$51,429) 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 get 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 hospitalized for cancer excluding the year of initial diagnosis (6.33%) or in prison for more than half of the year (0.19%). In total, we have 4,897,472 observations for 357,043 distinct individuals who are diagnosed with cancer at different times 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. Our main dependent crime variable is $C_{i,t}$, which equals one if person i is convicted of violating the penal code or other special legislation in year t , where t corresponds to the year when the crime is committed regardless

TABLE I
SUMMARY STATISTICS^a

	Mean (1)	SD (2)
Crime (in %)	0.684	8.244
Economic crime (in %)	0.410	6.387
Non-economic crime (in %)	0.053	2.311
Sexual crime (in %)	0.014	1.172
Property crime (in %)	0.379	6.142
Violent crime (in %)	0.095	3.080
Other crimes (in %)	0.073	2.697
Crime charge (in %)	0.750	8.627
First crime (in %)	0.290	5.380
In hospital (in %)	6.330	24.350
In prison (in %)	0.188	4.332
Male	0.406	0.491
Married	0.643	0.479
Age	47.791	9.668
Education	12.680	3.124
Homeowner	0.462	0.499
Total income (in 1,000 DKK)	318.86	646.51
Financial wealth (in 1,000 DKK)	118.86	200.36
Mortgage-to-income ratio	0.745	1.094
Doctors' fees (in DKK)	2,017.58	2,699.75
Psychological treatment fees (in DKK)	85.63	667.98
Physiotherapy fees (in DKK)	199.49	1,445.73

^aThis table reports summary statistics for our main sample, which consists of 4,897,472 observations. *Crime* takes a value of one if a person commits a crime for which she is then convicted. *Economic crime* takes a value of one if a person is convicted of a crime that is economically motivated. *Non-economic crime* takes a value of one if a person is convicted of a crime that is not economically motivated (the classification method is presented in Online Appendix Table G.I). *Property crime*, *Sexual crime*, and *Violent crime* take a value of one if a person is convicted, respectively, of a property, sexual, or violent crime on the basis of the classification system used by Statistics Denmark. *Crime charge* takes a value of one when a person is charged for a crime (regardless of whether she is convicted or not). *In hospital* takes a value of one if a person is hospitalized for cancer after the year of initial cancer diagnosis. *In prison* takes a value of one if a person is imprisoned for at least half of the year. *Male* is an indicator for male. *Married* is an indicator for married. *Age* is age in years. *Education* is the length of education in years. *Homeowner* is an indicator for home ownership. *Total income* is the gross total income. *Financial wealth* is the sum of the values of checking accounts, savings accounts, stocks, and bonds. *Mortgage-to-income ratio* is the mortgage amount divided by total income. *Doctors' fees* are the annual fees that doctors have received for their services provided to the individual. *Psychological treatment fees* are the annual fees that doctors have received for the psychological treatment provided to the individual. *Physiotherapy fees* are the annual fees that doctors have received for the physiotherapy treatment provided to the individual. *Mortgage-to-income ratio* and *Financial wealth* are winsorized at the 5th and 95th percentile.

of the conviction year. Table I shows that the probability of being convicted of a crime in a given year for the people in our sample is 0.68%.

The richness of the data allows us to explore further the different channels 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 G.I illustrates how the different types of crimes map into these categories and reports the crime summary statistics. The most common crime in Denmark is store theft, constituting 9.5% of all offenses. After that, holding drugs, other theft, and minor violent offenses are the most frequent infractions. Furthermore, we use the classification framework of Statistics Denmark to create three additional categorizations of crime: *Property Crimes*, *Sexual Crimes*, and *Violent Crimes*.

2.5. *Classifying cancer diagnostics*

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.

3. EMPIRICAL IDENTIFICATION OF CAUSAL EFFECTS

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. Yet, if a person anticipates a decline in her health, she could adjust her criminal behavior in advance, thereby invalidating a difference-in-differences approach. 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 adopt an event study design in which we focus only on people who develop cancer. This alleviates the concern that people who are not diagnosed with cancer are different along a number of meaningful dimensions. Specifically, we restrict our sample to only individuals who develop cancer between the age of 18 and 62 and, therefore, reveal to be similar in terms of the (unknown) determinants of the health shock.⁸ Furthermore, we account for the impact of time trends, personal traits, and age by including a battery of fixed effects. Our identifying assumption is that the exact timing of the cancer diagnosis is unpredictable, conditional on age, year of comparison, invariant personal traits, and on developing cancer at some point. With this procedure, we compare individuals who are similar along the underlying characteristics that determine the future trajectory of criminal paths but have different realizations in terms of the timing of the health shock.

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.⁹ 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.¹⁰ In Online Appendix B, we compare observables in the (same) pre-diagnosis year t for individuals who are respectively diagnosed 1 and 10

⁸A 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 similar results in both subsamples.

⁹Consider 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.

¹⁰Comparison 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).

years later. We find these individuals to be observationally equivalent in terms of the distribution of key covariates, as long as we account for time trends, age, and gender (see Online Appendix Figure G.1). In Section 7, we also report results where we impose the time interval between the diagnoses of treated and control individuals to be exactly 6 years. Results remain qualitatively similar.

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 research setting involves a multitude of treatment events, the static specification recovers the *weighted* average of all treatment effects. However, recent research shows that these weights lack economic interpretability and could even be negative when different units of observation are affected at different times and with different intensity (see [Sun and Abraham 2021](#), [Athey and Imbens 2018](#), [Borusyak and Jaravel 2017](#), [De Chaisemartin and d’Haultfoeuille 2020](#), [Goodman-Bacon 2018](#)). To overcome this issue, we estimate a semi-dynamic specification with a full set of post-treatment variables. Under the assumption of lack of pre-trends (verified below), we recover the causal effect of health shocks on crime by estimating the following linear probability model:

$$C_{i,t} = \alpha_i + \beta_t + \sum_{\tau=0}^{10} \gamma_{\tau} \mathbb{1}\{T_{i,t} = \tau\} + \lambda X_{i,t} + \epsilon_{i,t}, \quad (1)$$

where i indexes individuals, 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.¹¹

¹¹Our reduced-form approach may raise concerns including potential misspecification of the choice model and attrition bias (which we examine in detail in Section 7). Using simple Monte Carlo simulations of different data-

We also add a number of controls. α_i are person fixed effects and β_t are calendar year 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).¹² Time fixed effects account for time trends. For instance, crime progressively declines over time (see, e.g., [Donohue and Levitt 2001](#)), whereas the number of people diagnosed with cancer increases, thereby inducing a spurious negative correlation between the two variables. The vector $X_{i,t}$ includes *Age*, *In prison*, and *In hospital* fixed effects. Age fixed effects account for the fact that 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). *In prison* and *In hospital* fixed effects account for circumstances that limit the possibility of committing crime. We exclude from our sample the last available year ($t = 2018$), as all observations are treated in the last period, and the first cohort diagnosed in 1980, since these people are always treated.¹³

In the above specification, we estimate one coefficient for each relative time period, assuming that the treatment effects are homogeneous across year-of-diagnosis cohorts. However, evolving medical research, variations in social assistance schemes, and changing macro-economic conditions may give rise to variations in the intensity of treatment. Heterogeneity in treatment effects across cohorts could produce causally uninterpretable results because non-convex weights are assigned to cohort-specific treatment effects. This is especially the case for coefficients on leads ([Sun and Abraham 2021](#)). In a second specification, we estimate a different treatment effect for each year-of-diagnosis cohort and

generating processes, we find that our linear probability model tends to slightly underestimate the true treatment effect, whereas logit and probit slightly overestimate the true treatment effect.

¹²For instance, personality and childhood experiences affect the probability of facing financial distress (see, e.g., [Parise and Peijnenburg 2019](#)), which, in turn, can increase the propensity to commit crime.

¹³Notably, the control group and treatment group vary for each event time τ due to the high mortality rate post health shock and the age restrictions on the sample. In Section 7, we confirm that the results are qualitatively similar when we run the main analysis on a balanced sample. Furthermore, in Section 7 we also show 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.

retrieve the effect for each event time by taking the weighted average across cohorts. Formally:

$$C_{i,t} = \alpha_i + \beta_t + \sum_e \sum_{\tau} \gamma_{e,\tau} (\mathbb{1}\{E_i = e\} \times \mathbb{1}\{T_{i,t} = \tau\}) + \lambda X_{i,t} + \epsilon_{i,t}, \quad (2)$$

for year-of-diagnosis cohort $e := 1981, \dots, 2017$ and event times $\tau = 0, \dots, +10$. $\mathbb{1}\{E_i = e\}$ are indicator variables for the different cohorts. We then recover semi-dynamic treatment effects as $\gamma_{\tau} = \sum_e w_{e,\tau} \times \gamma_{e,\tau}$, where the weights $w_{e,\tau}$ are the sample share of each cohort in the relevant period (following [Sun and Abraham 2021](#)).

Both our approaches rely 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 multicollinearities. We follow [Borusyak and Jaravel \(2017\)](#) and omit the event year before treatment ($\tau = -1$) and a number of leads distant from the treatment ($\tau < -6$).¹⁴ Figure 1 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 = 0.45). Given known concerns about bias in the estimation of lead coefficients (see [Sun and Abraham 2021](#)), we also estimate an analogous specification based on Equation (2), which allows the pre-treatment effects to vary with year-of-diagnosis cohort. Our results again reassure against the presence of pre-trends (see Online Appendix Figure G.2).

The findings above validate our empirical design and mitigate concerns that the people in our sample anticipate the health shock. Note that the coefficients in Figure 1 should only

¹⁴Alternatively, we omit leads $\tau = -2$ and $\tau < -6$, thereby explicitly estimating the relation between cancer and crime the year before diagnosis ($\tau = -1$). Our results mitigate concerns regarding anticipation (see Online Appendix Figure G.3).

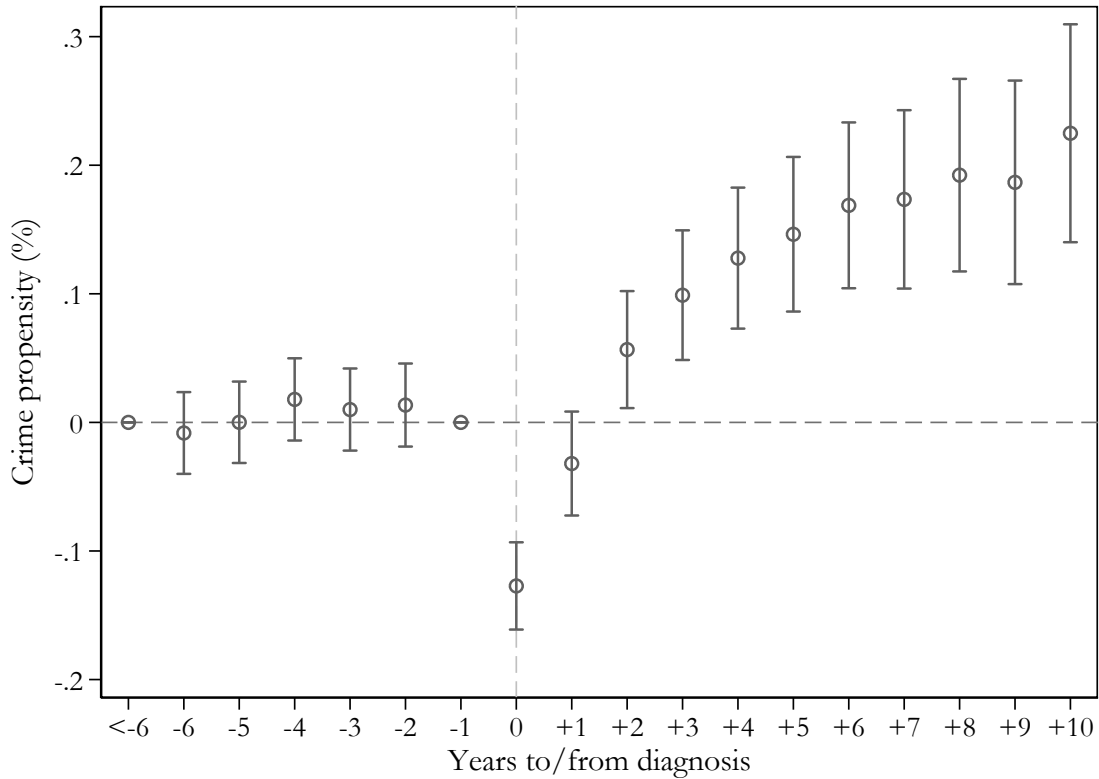


FIGURE 1.—Test for pre-trends in the relation between cancer and crime. *Notes:* This figure reports event study estimates for criminal activity changes in response to cancer diagnoses. The figure plots the estimated coefficients along with their 95% confidence interval. The x-axis denotes time with respect to the year of diagnosis. The y-axis denotes crime propensity in percentage points. The empirical model includes person, year, age, in prison, and in hospital fixed effects. The number of observations is 4,897,472.

be used to evaluate the absence of pre-trends, as they do not estimate the treatment effects efficiently. 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. The post-diagnosis coefficients are re-estimated and interpreted in Section 4 below.

4. BASELINE RESULT: THE EFFECT 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 decre-

ment is intuitive: undergoing cancer treatment is physically strenuous and forces a cancer patient to visit or remain at the hospital for long periods. Furthermore, economic support in the form of sick leave and sickness benefits may be granted for an extended period after the diagnosis, thereby delaying any adverse economic repercussion. 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 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 0.09 to 0.20 percentage points (a 12.4% to 29.7% increase compared to the baseline crime rate of 0.68 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 0.087 percentage points (significant at the 1% level): cancer patients are thus 13% more likely to commit a crime after they are diagnosed with cancer with respect to the baseline of 0.68 percentage points. This indicates that health shocks are trigger events that foster criminal behavior. We present several robustness analyses in Section 7.

Column 2 reports the coefficients estimated using Equation (2), which explicitly considers that the treatment effects may vary in intensity amongst year-of-diagnosis cohorts. We uncover an analogous pattern: both the estimates' economic magnitude and their statistical significance remain virtually unchanged compared with Column 1 of Table II. For the sake of brevity, we therefore only report results estimated using Equation (1) in the remainder of the paper.

5. WHY DOES CANCER PROMPT CRIME?

Guided by the theoretical framework proposed in Online Appendix A, we conjecture that a number of different mechanisms concur in explaining the effect of health shocks on

TABLE II
EFFECTS OF CANCER ON CRIME^a

Years from diagnosis	Homogeneous effects (1)	Heterogeneous effects (2)
0	-0.137 (0.015)	-0.132 (0.014)
+1	-0.043 (0.018)	-0.044 (0.018)
+2	0.044 (0.020)	0.043 (0.020)
+3	0.085 (0.023)	0.084 (0.022)
+4	0.113 (0.025)	0.111 (0.024)
+5	0.130 (0.027)	0.127 (0.027)
+6	0.151 (0.029)	0.149 (0.029)
+7	0.155 (0.032)	0.154 (0.031)
+8	0.173 (0.034)	0.169 (0.034)
+9	0.166 (0.036)	0.151 (0.035)
+10	0.203 (0.039)	0.197 (0.039)
ATE	0.087 (0.019)	0.084 (0.018)
Observations	4,897,472	4,897,472

^aThis table reports event study estimates for criminal activity changes in response to cancer diagnoses. Column (1) reports the coefficients obtained estimating Equation (1) (homogeneous treatment effects), and Column (2) estimating Equation (2) (heterogeneous treatment effects by diagnosis-cohort). At the bottom of each column the average treatment effects (ATEs) are reported. 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 empirical models include person, year, age, in prison, and in hospital fixed effects. All coefficients are multiplied by 100. Standard errors are clustered at the person level and presented in parentheses.

crime. A financial motive may induce individuals to mitigate the loss in human capital by seeking illegal revenues (economic channel). Furthermore, decreased survival probabilities might increase time discounting and therefore reduce the expected cost of future punish-

ment (survival probabilities channel). Finally, cancer may alter risk preferences (preference channel). 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 channel*

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 finding 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 III, Panel A document an increase of both economic and non-economic crime. Of the additional crimes prompted by cancer, economic crimes are three times more prevalent: we find a 0.05 percentage point increase in economic crimes vs. a less than 0.02 percentage point increase in non-economic crimes. 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 and violent offenses increase significantly, while the incidence of sexual offenses does not change. These results suggest that the economic channel explains an important part of the surge in crime post health shocks. However, we document a substantial and statistically significant increase in non-economic crime as well.

Next, we explore the response heterogeneity based on the economic background of cancer patients. Panel (a) of Figure 2 shows that there is no large difference in crime rates between people who have above- and below-median income *levels* in the year before treat-

TABLE III
ECONOMIC CHANNEL—EFFECTS OF CANCER ON DIFFERENT TYPES OF CRIME^a

Years from diagnosis	Panel A: Economic crime?		Panel B: Property, sexual, or violent crime?		
	Economic (1)	Non-economic (2)	Property (1)	Sexual (2)	Violent (3)
0	-0.098 (0.011)	-0.004 (0.004)	-0.086 (0.011)	-0.001 (0.002)	-0.017 (0.006)
+1	-0.018 (0.014)	0.003 (0.005)	-0.014 (0.014)	0.001 (0.003)	-0.010 (0.007)
+2	0.032 (0.016)	0.012 (0.006)	0.032 (0.015)	0.004 (0.003)	-0.000 (0.008)
+3	0.053 (0.018)	0.009 (0.006)	0.052 (0.017)	0.001 (0.003)	0.016 (0.009)
+4	0.069 (0.019)	0.014 (0.007)	0.069 (0.019)	0.001 (0.003)	0.017 (0.009)
+5	0.088 (0.021)	0.013 (0.007)	0.085 (0.021)	0.003 (0.004)	0.024 (0.011)
+6	0.079 (0.023)	0.017 (0.008)	0.072 (0.022)	0.003 (0.004)	0.033 (0.011)
+7	0.092 (0.025)	0.033 (0.009)	0.082 (0.024)	0.010 (0.005)	0.029 (0.012)
+8	0.089 (0.026)	0.025 (0.010)	0.090 (0.026)	0.009 (0.005)	0.031 (0.013)
+9	0.085 (0.028)	0.036 (0.010)	0.082 (0.027)	0.006 (0.005)	0.025 (0.014)
+10	0.126 (0.031)	0.039 (0.011)	0.125 (0.030)	0.010 (0.006)	0.048 (0.015)
ATE	0.053 (0.015)	0.016 (0.006)	0.052 (0.015)	0.004 (0.003)	0.015 (0.007)
Observations	4,897,472	4,897,472	4,897,472	4,897,472	4,897,472

^aThis 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). At the bottom of each column the average treatment effects (ATEs) are reported. 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 empirical models include person, year, age, in prison, and in hospital fixed effects. All coefficients are multiplied by 100. Standard errors are clustered at the person level and presented in parentheses.

ment. In Panel (b), we instead 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.¹⁵ Although crime rates fall in a comparable way in the diagnosis year, individuals who experience a more severe loss of income are more likely to commit crime after cancer compared to those who do not (ATE of 0.098, statistically significant at the 1% level, versus 0.017, statistically insignificant).¹⁶ This finding supports the existence of an economic channel and suggests that the individuals whose human capital is affected the most seek additional revenues in the illegal labor market.

This result also allows us to estimate the additional societal benefit of income subsidies in terms of the reduction in the cost of crime. In particular, if cancer patients were fully compensated for their income losses, then such income subsidies would be recuperated in a range between 2% and 18% through a lower negative externality of crime.¹⁷

Panel (c) reports that the increase in criminal activity is driven by individuals who do not own a home before the diagnosis, thereby suggesting that home equity provides a cushion (in line with [Gupta et al. 2018](#)). By contrast, homeowners do not significantly alter their criminal activity regardless of whether they are highly leveraged or not. The finding that highly indebted households do not alter their crime supply suggests that the relation is not driven by previous expense commitments. Finally, Panel (d) shows that the effect is mostly driven by people with below-median financial wealth pre-cancer.

Taken together, these results speak to the fact 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 (e.g., [Lochner 2004](#), [Mocan, Billups, and Overland 2005](#)). Furthermore, our

¹⁵This 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.

¹⁶In Online Appendix Table G.II, we show that the effect of cancer on income is large and heterogeneous (consistent with previous findings; see [García-Gómez et al. 2013](#)).

¹⁷For our back-of-the-envelope calculation, we assume that the total cost of crime ranges between \$1,300 and \$10,700 per capita based on estimates from the UK and US ([Anderson 2012](#), [Brand and Price 2000](#), [United States Government Accountability Office 2017](#)). Furthermore, we assume that when everyone is compensated for their income loss following cancer that the incentive to commit crime drops as predicted by the average treatment effect estimates in Figure 2 (0.098 and 0.017). For more details, see Online Appendix C.

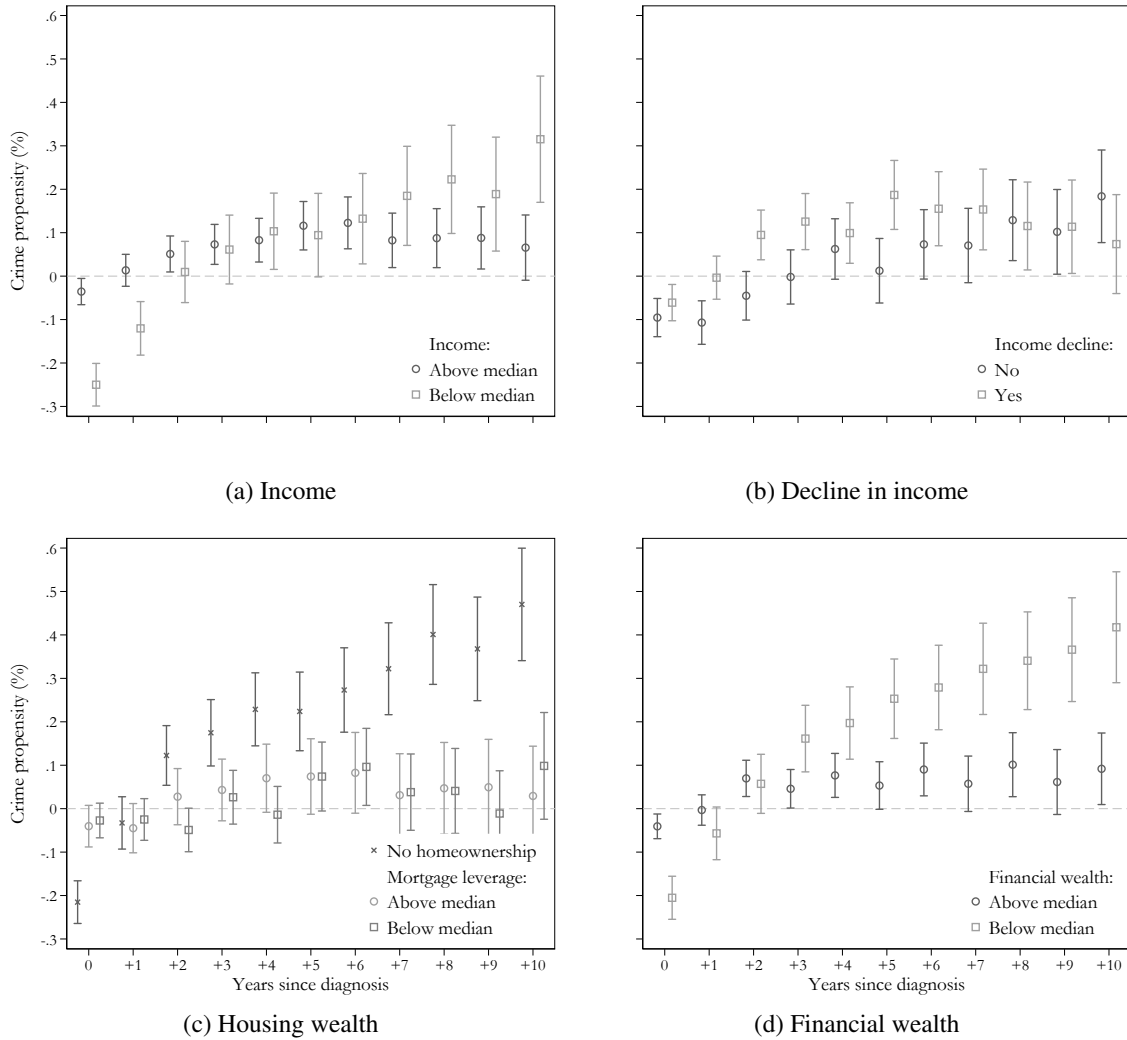


FIGURE 2.—Economic channel: personal finances. *Notes:* These figures report event study estimates for criminal activity changes in response to cancer diagnoses. The figures plot the estimated coefficients along with their 95% confidence interval. The x-axes denote time with respect to the year of diagnosis. The y-axes denote crime propensity in percentage points. Individuals are sorted in different subsets according to financial variables. Panel (a) shows the coefficients for people with above- (respectively below-) median income level in the year before the cancer diagnosis. Panel (b) shows the coefficients for people who face (respectively do not face) an income decline in the 6 years following the cancer diagnosis with respect to the year before the cancer diagnosis. Panel (c) shows the coefficients for people with no home equity, high mortgage-to-income ratio, and low mortgage-to-income ratio in the year before the cancer diagnosis. Panel (d) shows the coefficients for people with above (respectively below) median financial wealth in the year before the cancer diagnosis. The empirical models include person, year, age, in prison, and in hospital fixed effects. Standard errors are clustered at the person level.

results indicate that having ex ante a financial buffer to absorb the human capital loss can prevent or, at the very least, mitigate the surge in offenses. 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 and explore the effect of a change in social assistance schemes on the cancer–crime relationship.

5.2. *Survival probabilities channel*

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 A). 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.¹⁸ 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 to define the two groups on the basis of gender, resulting in an equal share of men and women in both groups. 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, in Online Appendix Figure G.4, we control for the effect of income in our specifications to ensure that we are not capturing the fact that more severe types of cancer are associated with a stronger income decline. We discuss the procedure for the estimation of survival probabilities in detail in Online Appendix D.

Figure 3 shows that crime reduction is larger for those individuals whose survival probabilities are affected the most in the year of diagnosis.¹⁹ Yet, in the long run, these are

¹⁸We consider a five-year period because this is standard in the medical literature.

¹⁹In Figure 3 standard errors are clustered at the person level. In Online Appendix Figure G.5, we present results obtained with bootstrapping to account for the fact that estimates are based on a two-stage procedure.

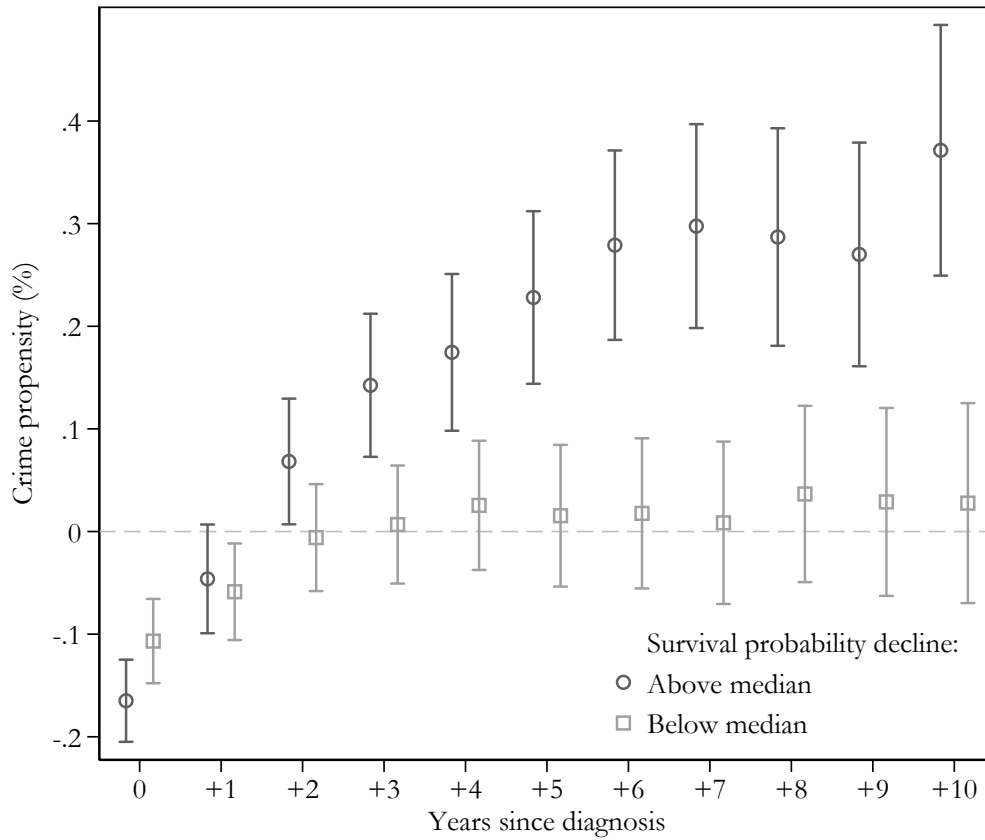


FIGURE 3.—Survival probabilities channel: decline in survival probabilities and the relation between cancer and crime. *Notes:* This figure reports event study estimates for criminal activity changes in response to cancer diagnoses. The figure plots the estimated coefficients along with their 95% confidence interval. The x-axis denotes time with respect to the year of diagnosis. The y-axis denotes crime propensity in percentage points. 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 person, year, age, in prison, and in hospital fixed effects. Standard errors are clustered at the person level.

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

5.3. Preference channel

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 et al. 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 et al. (2008) and Andersen et al. (2014), to which we refer the reader for a detailed description of the experimental design.

Table IV shows the relation between health shocks and risk preferences. *Post-cancer* equals one in the year of diagnosis and the following 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 channel in our setting. In Online Appendix Figure G.6, we also show the effect of cancer on the likelihood of receiving speeding tickets but do not find evidence of increased risky behavior. Overall, we do not find empirical support for a preference channel in our data.²⁰

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

²⁰We 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.

TABLE IV
PREFERENCE CHANNEL—EFFECTS OF CANCER ON RISK ATTITUDES^a

	(1)	(2)	(3)
Post-cancer	0.229 (0.173)	0.242 (0.173)	0.425 (0.168)
Male		-0.0974 (0.162)	0.0518 (0.186)
Post-cancer × Male			-0.388 (0.170)
Observations	38	38	38

^aThis table reports cross-sectional estimates for the relation between risk aversion and having had a cancer diagnosis. Our baseline sample is matched with risk aversion measures obtained from lab 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.

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, 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 via the economic channel.

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 faced 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 E). We define as “stingy” (“generous”) the municipalities with below (above) median income-replacement after cancer. Figure 4 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 the sensitivity of crime to cancer changes for those who face a large decrease in generosity as follows:

$$C_{i,t} = \alpha_i + \beta_t + \sum_{\tau} b_{\tau} (\mathbb{1}\{T_{i,t} = \tau\} \times \textit{Stingy muni}_{t,M}) + \textit{Stingy muni}_{t,M} + \sum_{\tau} \gamma_{\tau} \mathbb{1}\{T_{i,t} = \tau\} + \lambda X_{i,t} + \epsilon_{i,t}, \quad (3)$$

where $\textit{Stingy muni}_{t,M}$ is a variable that takes a value of one during the post-reform period for people residing in an area that became part of a stingy municipality while not being part of a stingy municipality before the reform. The coefficient b_{τ} therefore captures the additional effect of the reduction in generosity on the sensitivity of crime to cancer. We do not include municipality fixed effects as they are collinear with the person fixed effects.²¹

Table V 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

²¹This is true only in the absence of “movers,” i.e., individuals relocating from one municipality to another. However, including municipality fixed effects does not alter our results. In Online Appendix Table G.III, we address the possibility that cancer patients strategically relocate to a better municipality by excluding movers from the analysis.

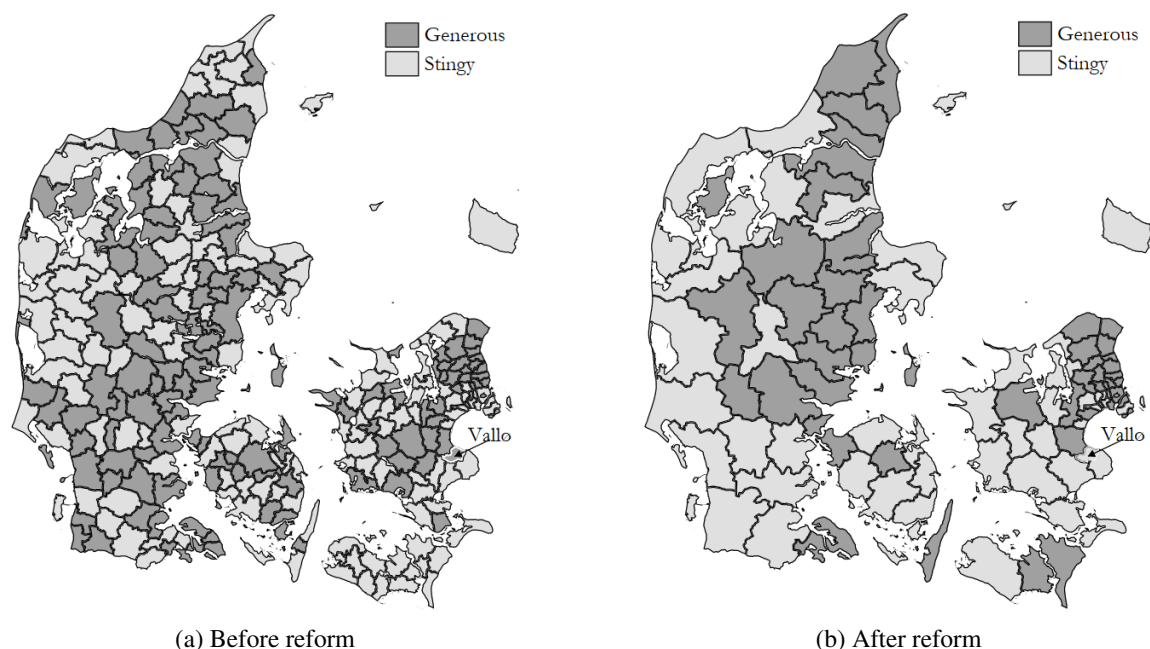


FIGURE 4.—Welfare generosity before and after the municipality reform. *Notes:* This figure illustrates the generosity of Danish municipalities before and after the implementation of the January 1, 2007 municipality reform. A generous (stingy) municipality is a municipality with above- (below-) median 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 E.

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 average effect of cancer on crime in our sample is 0.07 percentage points, a reduction in social support policies fosters a further increase by 0.09 percentage points.²²

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

²²A potential concern with our methodology is that less generous social policies may also affect people who have not yet been diagnosed with cancer (for instance, through a reduction of other subsidies that do not affect cancer patients). To sharpen our estimates, in Online Appendix Table G.IV, we replicate our analysis, retaining in our sample exclusively individuals after they have been diagnosed with cancer. Specifically, we run a difference-in-differences analysis whereby we compare the crime rate among cancer patients before and after a municipality is downgraded to stingy with the crime rate among cancer patients who reside in municipalities that are not downgraded to stingy. This analysis yields analogous conclusions: cancer patients residing in municipalities that are treated with the generosity shock increase their propensity for violating the law relative to those residing in untreated municipalities.

TABLE V
CHANGE IN WELFARE GENEROSITY AND THE EFFECT OF CANCER ON CRIME^a

Years since diagnosis	Years from diagnosis indicator (1)	Years from diagnosis indicator \times <i>Stingy muni</i> (2)
0	-0.149 (0.023)	0.095 (0.045)
+1	-0.046 (0.019)	0.029 (0.042)
+2	0.031 (0.023)	0.096 (0.053)
+3	0.083 (0.022)	0.024 (0.055)
+4	0.094 (0.026)	0.124 (0.061)
+5	0.125 (0.027)	0.040 (0.059)
+6	0.125 (0.034)	0.164 (0.064)
+7	0.123 (0.031)	0.194 (0.077)
+8	0.145 (0.034)	0.161 (0.079)
+9	0.135 (0.036)	0.172 (0.073)
+10	0.180 (0.040)	0.113 (0.086)
ATE	0.072 (0.019)	0.093 (0.040)
Observations	4,897,472	

^aThis table reports event study estimates for the effect of the 2007 municipality reform on the relation between cancer and crime using Equation (3). *Stingy muni* takes a value of one for people residing in an area that became part of a stingy municipality in 2007, while not being part of a stingy municipality before 2007. 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 the variable *Stingy muni*. At the bottom of each column the average treatment effects (ATEs) are reported. 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 empirical model includes person, year, age, in prison, and in hospital fixed effects. All coefficients are multiplied by 100. Standard errors are clustered at the post-reform municipality level and presented in parentheses.

system appears to play a large 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. HETEROGENEOUS RESPONSE TO HEALTH SHOCKS

6.1. *Heterogeneous responses: Demographic variables*

Our baseline sample includes a broad cross-section of individuals who have *ex ante* a different propensity to engage in criminal behavior. In the following, we consider alternative subsets of the Danish population, sorted along a number of relevant socio-demographic dimensions measured the year before treatment. Our aim is to identify differences in how people react to health shocks.

Figure 5, Panel (a) shows that the impact of cancer on crime is, on average, greater for men than for women. Note that we estimate larger confidence intervals for the subsample of men, as the incidence of cancer is lower among the male population. Panel (b) reports that younger and older individuals react similarly to health shocks. Furthermore, Panel (c) shows that the increase in crime is significantly higher for lower-educated individuals. Panel (d) indicates that the effect is driven by singles, whereas being part of a family prevents a direct effect of cancer on crime. 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). We explore the effect on the criminal behavior of the spouse in Section 6.3. Finally, Panel (e) shows that the effect is stronger for those individuals who have previous connections to crime through a family member (parent, sibling, partner, child, or in-law) who has violated the law before diagnosis. 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. Taken together, the results presented in Figure 5 indicate that there are heterogeneous effects of cancer on crime. In particular, the individuals most likely to "break bad" are single, low-educated men who have a family member with a criminal record.

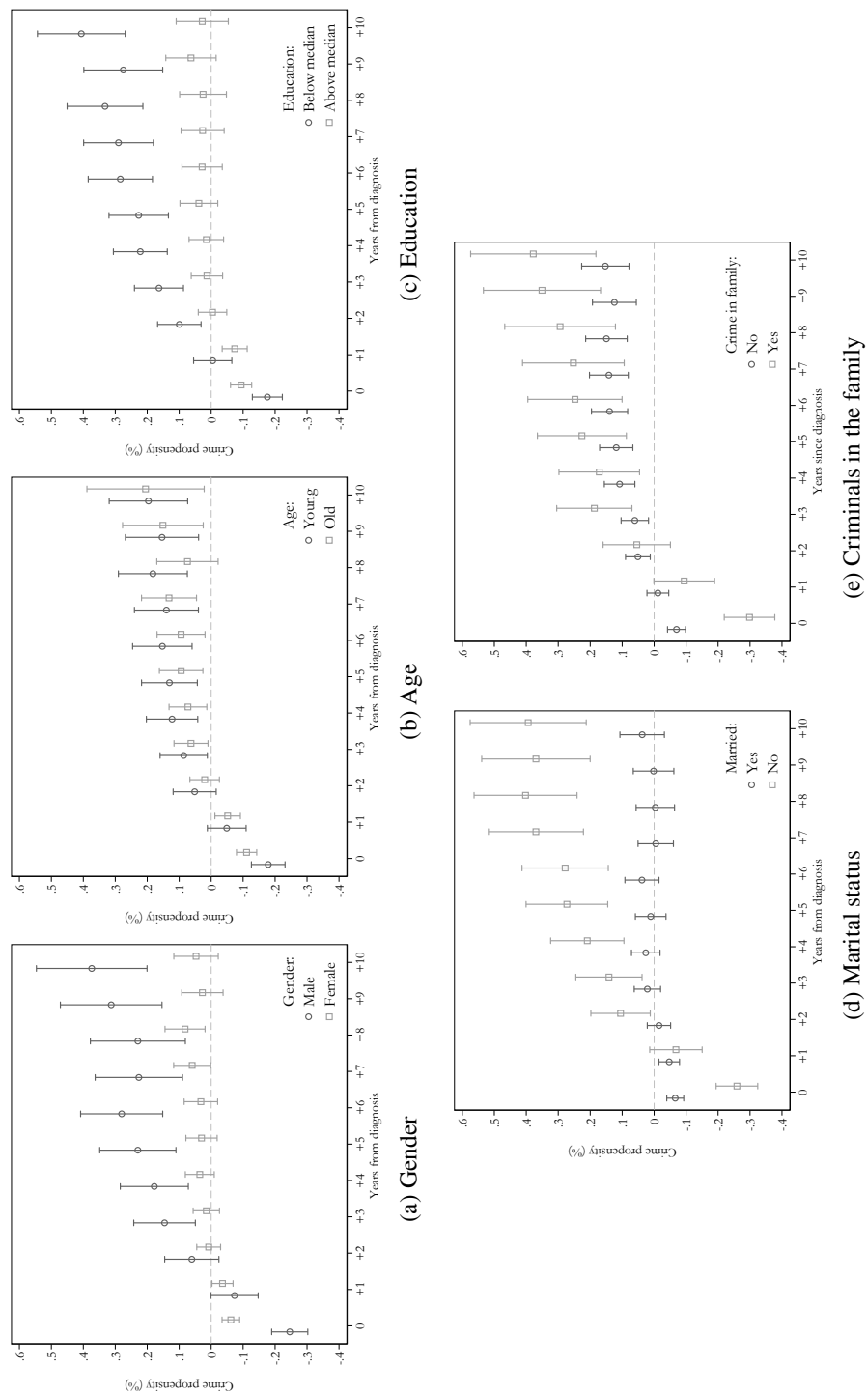


FIGURE 5.—Heterogeneous crime responses to cancer. *Notes:* These figures report event study estimates for criminal activity changes in response to cancer diagnoses. The figures plot the estimated coefficients along with their 95% confidence interval. The x-axes denote time with respect to the year of diagnosis. The y-axes denote crime propensity in percentage points. Individuals are sorted into different subsets according to demographic characteristics. Panel (a) shows the coefficients for women versus men. Panel (b) shows the coefficients for people above (respectively below) median age in the year before cancer diagnosis. Panel (c) shows the coefficients for people who achieved above (respectively below) median length of education in the year before cancer diagnosis. Panel (d) shows the coefficients for married versus unmarried people. Panel (e) shows the coefficients for people who have (respectively have not) at least one family member (parent, sibling, partner, child, or in-law) who committed crime in the past. The empirical models include person, year, age, in prison, and in hospital fixed effects. Standard errors are clustered at the person level.

6.2. Extensive and intensive margin

Do individuals with a clean record start committing crime because of cancer? The answer to this question speaks directly to the trigger event hypothesis and has relevant policy implications. If we found an effect at the intensive margin only (i.e., an increase in crime rates by previous criminals), it would indicate that health shocks are not pivotal in determining whether individuals become criminals in the first place. By contrast, the presence of an effect at the extensive margin (i.e., by individuals who were not criminals before the diagnosis) would identify health shocks as trigger events. From a policy standpoint, the latter result would imply that some individuals may be prevented from becoming criminals by mitigating the adverse effects of health shocks.

To estimate the extensive margin effect, we run a specification that replaces our baseline crime variable with a first crime indicator variable (*First Crime*). Column 1 of Table VI shows that the cancer diagnosis increases the probability that individuals with a clean record are convicted for the first time. In particular, after the cancer diagnosis, individuals are 0.028 percentage points more likely to commit their first infraction (10% 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).²³ 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 VI reports the coefficients for the average impact of cancer. We find an effect of 0.46 percentage points: 8% more than the average rate among criminals of 6.06 percentage points. Overall,

²³Notably, 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 bias our coefficient of interest against finding a positive effect. 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 different 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 only commit crime after cancer).

TABLE VI
NEW CRIMINALS VS RE-OFFENDERS^a

Years from diagnosis	First crime (1)	Re-offenders (2)
0	-0.041 (0.010)	-1.288 (0.132)
+1	-0.020 (0.012)	-0.522 (0.166)
+2	0.031 (0.014)	0.176 (0.186)
+3	0.024 (0.015)	0.487 (0.207)
+4	0.041 (0.017)	0.690 (0.226)
+5	0.050 (0.018)	0.795 (0.247)
+6	0.048 (0.019)	0.963 (0.264)
+7	0.053 (0.021)	0.942 (0.282)
+8	0.050 (0.022)	1.065 (0.303)
+9	0.035 (0.023)	0.948 (0.318)
+10	0.034 (0.024)	1.230 (0.342)
ATE	0.028 (0.013)	0.464 (0.169)
Observations	4,897,472	552,696

^aThis 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 have committed a criminal offense between 1980 and 2018. In Column (2), the dependent variable is *Crime*. At the bottom of each column the average treatment effects (ATEs) are reported. 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 empirical models include person, year, age, in prison, and in hospital fixed effects. All coefficients are multiplied by 100. Standard errors are clustered at the person level and presented in parentheses.

we conclude that health shocks elicit a response both at the intensive and the extensive margin. Yet, relative to the baseline, the effect is more substantial for people who have never

violated the law before. This evidence supports the claim that health shocks are trigger events.

6.3. *Partner's criminal behavior*

We further explore how health shocks impact the criminal behavior of the spouse or registered partner of the diagnosed individual. Specifically, we rely on the health shock indicator variables for (sick) person i to explain the criminal behavior of her (healthy) partner j . Only partners of individuals diagnosed with cancer are considered for this analysis. We find that a 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 estimates for people with cancer. This result is presented in Table VII. Together with our result that cancer patients in a relationship do not break the law (see Figure 5 Panel (d)), this finding suggests that, in households affected by a health shock, the healthy partner carries out the criminal offense.

7. ROBUSTNESS

7.1. *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.

TABLE VII
EFFECTS OF CANCER ON CRIME COMMITTED BY PARTNER^a

Years from diagnosis	Partner crime
0	0.038 (0.017)
+1	0.015 (0.020)
+2	0.031 (0.022)
+3	0.017 (0.025)
+4	0.049 (0.027)
+5	0.042 (0.030)
+6	0.089 (0.033)
+7	0.087 (0.035)
+8	0.125 (0.038)
+9	0.094 (0.040)
+10	0.072 (0.043)
ATE	0.049 (0.021)
Observations	3,682,447

^aThis table reports event study estimates for criminal activity changes of the partner of cancer patients in response to cancer diagnoses. At the bottom of the column the average treatment effect (ATE) is reported. 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 empirical model includes person, year, age of cancer patient, age of partner, in prison, and in hospital fixed effects. All coefficients are multiplied by 100. Standard errors are clustered at the person level and presented in parentheses.

We further run a battery of tests to attenuate concerns of a change in criminal ability. First, we reproduce our main results controlling for proxies of criminal ability based on the diagnosed individuals' physical and psychological condition (see Column 1, Table VIII).

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 G.V). Third, we compute the percentage of reported crimes that remains unsolved in each municipality and show that this quantity 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 G.VI).

7.2. *Further robustness checks*

We conduct a number of additional tests. We 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 (Table VIII, Columns 2 and 3, respectively). Furthermore, we consider that attrition may pose a threat to our identification if correlated with crime. This would be the case, for instance, if mortality rates are different between criminals and non-criminals. We tackle this concern by re-estimating our analysis on a balanced sample. Using this balanced sample reduces our statistical power due to the mortality rate post-cancer and the smaller event window. However, the ATE coefficient's economic magnitude remains similar to that estimated on the non-balanced sample (see Column 4). Column 5 shows that our results are similar when we impose that treatment and control observations are diagnosed precisely 6 years apart (following the approach of [Fadlon and Nielsen 2019](#)).²⁴ We explain this stacked difference-in-differences analysis in detail in Online Appendix Section F. Finally, in Column 6, 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 15% relative to the sample average of 0.75 percentage points). Overall, all specifications produce qualitatively similar results.

²⁴The number of observations using the stacked difference-in-differences approach is larger than in the balanced panel, as the same individual can appear both in the treatment and in the control group.

TABLE VIII
ROBUSTNESS TESTS^a

Years from diagnosis	Crime ability controls (1)	Muni FEs (2)	Muni × year FEs (3)	Balanced panel (4)	Stacked diff-in-diff (5)	Charges (6)
0	-0.150 (0.017)	-0.137 (0.015)	-0.137 (0.015)	-0.005 (0.034)	0.001 (0.036)	-0.136 (0.015)
+1	-0.058 (0.020)	-0.044 (0.018)	-0.045 (0.018)	0.031 (0.040)	0.023 (0.036)	-0.044 (0.019)
+2	0.029 (0.023)	0.044 (0.020)	0.044 (0.020)	0.046 (0.045)	0.061 (0.036)	0.058 (0.021)
+3	0.091 (0.026)	0.085 (0.023)	0.084 (0.023)	0.113 (0.052)	0.099 (0.037)	0.103 (0.024)
+4	0.116 (0.028)	0.112 (0.025)	0.111 (0.025)	0.155 (0.058)	0.125 (0.036)	0.133 (0.026)
+5	0.121 (0.031)	0.130 (0.027)	0.130 (0.027)	0.128 (0.065)	0.118 (0.037)	0.161 (0.029)
+6	0.139 (0.033)	0.152 (0.029)	0.150 (0.029)	0.112 (0.072)		0.192 (0.031)
+7	0.148 (0.035)	0.155 (0.032)	0.154 (0.032)			0.178 (0.033)
+8	0.171 (0.038)	0.172 (0.034)	0.172 (0.034)			0.200 (0.036)
+9	0.165 (0.040)	0.166 (0.036)	0.166 (0.036)			0.193 (0.038)
+10	0.194 (0.043)	0.203 (0.039)	0.202 (0.039)			0.228 (0.041)
ATE	0.080 (0.022)	0.087 (0.019)	0.086 (0.019)	0.098 (0.050)	0.085 (0.029)	0.106 (0.020)
Observations	3,707,408	4,897,472	4,897,472	1,058,681	3,892,220	4,897,472

^aThis table reports robustness tests. In Columns (1) to (5), the dependent variable is the standard crime indicator, *Crime*. Column (1) includes additional controls proxying for the ability to commit crime: doctors' fees, psychological treatment fees, physiotherapy fees, and the logs of these controls. These are payments made by the state to the doctor(s). In Column (4), the analysis is performed on individuals who have survived cancer for at least 6 years, and for whom we have data for at least 6 years before the cancer diagnosis. The analysis only includes data from 6 years before the diagnosis until 6 years after the diagnosis. Column (5) reports estimates of a stacked difference-in-differences approach, which imposes that treatment and control observations are diagnosed exactly 6 years apart (as in [Fadlon and Nielsen \(2019\)](#); details are in Online Appendix F). In Column (6), the dependent variable is *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). At the bottom of each column the average treatment effects (ATEs) are reported. 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. All empirical models include person, year, age, in prison, and in hospital fixed effects. Column (2) further includes municipality fixed effects, and Column (3) municipality × year fixed effects. All coefficients are multiplied by 100. Standard errors are clustered at the person level and presented in parentheses.

8. CONCLUSION

In this paper, we provide evidence that health shocks elicit criminal behavior. Exploiting the random timing of cancer diagnosis, 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 and welfare support runs out. Overall, we document that health shocks have negative externalities that lie outside of the private sphere.

We further examine the channels 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, low financial wealth, and fewer years of education. 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 channel in our data. These results are in line with a simple rational framework showing that health shocks may induce crime, which builds on the seminal work by Isaac Ehrlich (1973). Notably, the adverse effects of health shocks will presumably be even stronger in institutional settings in which people bear larger financial costs from health shocks.

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