For Online Publication: Appendix of "Breaking Bad: How Health Shocks Prompt Crime"

APPENDIX A: COMPARISON OF INDIVIDUALS DIAGNOSED IN 1 VERSUS 10 YEARS

To further validate our approach, we conduct an exact matching between pre-diagnosis individuals who, in the same (calendar) year, are of the same age and gender but are diagnosed as far apart as possible.[1] Specifically, Online Appendix Table I.III compares the averages of seven key covariates (pre-diagnosis crime, survival probability decline, total income, earnings, homeownership, financial wealth, marital status, and education) between people who will be diagnosed in 1 year and people who, in the same year, are of the same age and gender but will be diagnosed only 10 years later, which is the longest gap available by construction (as we retain only 10 years before cancer in our sample). Furthermore, Online Appendix Figure I.2 plots the average crime rate, total income, marital status, and education over time for the two groups (cancer in 1 and 10 years, respectively).

As can be seen from Online Appendix Table I.III and Online Appendix Figure I.2, the two samples show very similar averages and trajectories with age. While differences in several covariates are statistically significant, in economic terms the differences between the two groups are small. Importantly, we find no significant difference in the average crime rate before diagnosis. This evidence supports the argument that individuals diagnosed many

[1] Note that in our baseline specification these comparability requirements are addressed by including year-by-age and person fixed effects.

years apart are ex ante observationally equivalent in terms of the relevant covariates. And, therefore, the timing of the cancer diagnosis is as good as random for the purpose of our analysis.

APPENDIX B: NEVER-TREATED AS AN ALTERNATIVE CONTROL GROUP

B.1. *Differential criminal trends of treated and never-treated*

Unobservable differences between the treated and the never-treated group can lead the latter to follow a different trend in criminal activity. Online Appendix Figure I.3 shows raw crime rates in event time for three groups of individuals: i) treated, ii) yet-to-be treated, and iii) never treated. The treated group contains all treated individuals from 10 years before diagnosis to 10 years after. The yet-to-be-treated include individuals up to one year before diagnosis. The never-treated are people who never develop cancer in our data. Since neither the yet-to-be treated nor the never treated receive a cancer diagnosis in this sample, we set event date $\tau = 0$ at age 52 for these groups. We center the event study at 52, as this is the age when the average individual in the treatment group is diagnosed with cancer. This implies that yet-to-be-treated and never-treated individuals are 42 at $\tau = -10$ and 62 at $\tau = +10$. We observe that the criminal trajectories for treated and yet-to-be treated individuals are highly similar before event date $\tau = 0$, while the average crime rate across event years is less similar when comparing the treated and never-treated population. This finding suggests that never-treated individuals are to an extent on a different trajectory and, therefore, might not be a valid control group.

B.2. *Specification with never-treated group as control*

We test whether our results are similar when considering people who are never diagnosed with cancer as control group. To that end, we augment our sample by including a random selection of people who never develop cancer in our data. To make the treatment and control group comparable in size, we select a number of "never treated" individuals equal to the number of cancer patients in our main sample. We then re-estimate equation 1 in the paper. The treatment indicators $\mathbb{1}\{T_{i,t} = \tau\}$ are zero for all never-treated individuals in all years. Online Appendix Figure I.5 illustrates a similar pattern as the one that we find from our main analysis. Crime propensity falls at time $\tau = 0$, and then recovers shortly thereafter, rising significantly above pre-diagnosis levels at event year $\tau = +5$. The ATE and RTE are smaller than in our main analysis at 0.03 percentage points and 2.58%, respectively.

Parallel lead coefficients provide an indication of whether the control group is a suitable counterfactual. In the analysis that includes never treated individuals as control (Online Appendix Figure I.5), we find lead coefficients that are negative and jointly significant at the 10% level, which supports the conclusion that the crime rates of never-treated individuals are on a different trend.

APPENDIX C: ALTERNATIVE TESTS OF THE ASSUMPTION OF PARALLEL TRENDS

We propose a number of alternative tests of the hypothesis that crime rates run parallel for control and treated individuals before the latter are diagnosed. Following Borusyak, Jaravel and Spiess (2024) we test for anticipation on untreated observations only ($\tau < 0$), thereby explicitly separating testing from estimation. Specifically, we estimate lead coefficients for the five periods immediately before treatment, with periods more than five years before treatment serving as the reference group. Online Appendix Figure I.11 Panel b shows insignificant coefficients for each lead. We cannot reject the null hypothesis that the pre-event coefficients are jointly equal to zero (F-statistic $= 0.57$, p-value $= 0.75$). We also test for parallel trends violations recovering lead coefficients as linear combinations of different cohort effects (following Sun and Abraham 2021). Furthermore, we employ the methodology of De Chaisemartin and D'Haultfœuille (2020), De Chaisemartin and d'Haultfoeuille (2024) to estimate placebo effects. Also in these cases, we find no evidence of pre-trends (see Online Appendix Figure I.11, panel c and d).

APPENDIX D: STYLIZED FRAMEWORK

We present a simple framework that outlines how health shocks may induce criminal behavior. We consider three main channels that prompt crime through changes in (i) the ability to earn legal income, (ii) survival probabilities, and (iii) preferences.[2]

D.1. *Model setup*

An individual lives for a maximum of two periods, a working period, from $t = 0$ to $t = 1$, and a retirement period, from $t = 1$ to $t = 2$. At the start of the working period, the

[2]Our framework builds on the models by Dobkin et al. (2018) and Ehrlich (1973). As our focus is on examining the effect of health shocks on criminal activity, we remove several features from the Dobkin et al. (2018) model of health and add others from Ehrlich (1973). Specifically, we remove savings behavior and out-of-pocket medical expenses (as these are negligible in Denmark). By contrast, we add the decision to commit criminal activity and the possible consequences, as well as the change in survival probability due to the health shock.

individual receives information on her health state $J \in \{S, H\}$. We superscript the state of the world in which she is sick with an S, and the state in which she is healthy with an H. After observing the state of the world, she chooses to allocate a share, κ^J, of her labor supply to illegal activities, and the residual share, $1-\kappa^J$, to legal activities. For simplicity, we assume that the discount rate is zero, such that the individual maximizes lifetime utility defined as:

$$U(c_1^J, c_2|\kappa^J) \equiv g\big(c_1^J(\kappa^J)\big) + \rho^J g(c_2) - \rho^J b(\kappa^J), \tag{1}$$

where $g(\cdot)$ represents the per-period utility of consumption (c_1^J and c_2) which is increasing and concave in consumption (i.e., $g'(\cdot) > 0$ and $g''(\cdot) < 0$). The survival probability to the retirement period is denoted by ρ^J. The last term, $b(\kappa^J)$, is the expected disutility of crime, which we assume is globally increasing and weakly convex in crime to reflect that both the likelihood of getting caught and the size of the penalty increase with the share of labor supply allocated to crime (i.e., $b'(\kappa^J) > 0$ and $b''(\kappa^J) \geq 0$). We assume that the disutility of crime in the first period is zero. This simplifying assumption reflects that criminals are usually apprehended and convicted with a delay and implies that survival probabilities matter when choosing the fraction of labor supply to allocate to criminal activity.

To explore the tradeoff between illegal and legal activity, we define the income process as follows: the labor supplied to the legal market earns a wage w and the labor supplied to the illegal market earns a wage normalized to one. Importantly, we assume $w < 1$, which ensures compensation for the additional expected disutility of crime, and thus a positive supply of criminal activity (in line with Ehrlich 1973 and Freeman 1999).[3] A health shock reduces human capital, which translates into lower productivity, and, in turn, results in lower compensation for legal activity. More generally, reducing legal wages can be interpreted as a worse career trajectory due to illness. We model the fall in productivity by assuming that the legal wage declines by a fraction $\alpha \in]0, 1[$ in the sick state.

Furthermore, the welfare system only partially compensates for the reduction in legal earnings. We model sickness benefits by assuming that the welfare system compensates a fraction $\lambda \in [0, 1[$ of the legal wage decline, α. For simplicity, we assume that there is no possibility to save so that in each period the individual consumes her entire income. Fur-

[3] Notably, this setting can be easily extended to non-economic crimes by interpreting w as non-monetary utility from criminal activity.

thermore, we assume that, in the retirement period, the individual consumes exogenously fixed retirement benefits c_2. Consumption in the working period in the two different states is as follows

$$c_1^S = (1 - \kappa^S)\iota w + \kappa^S, \tag{2}$$

$$c_1^H = (1 - \kappa^H)w + \kappa^H, \tag{3}$$

where $\iota = [1 - \alpha[1 - \lambda]]$, $0 < \iota \leq 1$ reflects the fraction of legal income including sickness benefits maintained in the sick state.

To explore the impact of an adverse health event on criminal activity through changes in survival probabilities, we specify the probability of being alive in the second period for each health state. In a healthy state, a person's survival probability is $\rho^H = \rho$, while in a sick state a person's survival probability is lowered by ϱ, thus $\rho^S = \rho - \varrho$, where $\rho \in [0, 1]$ and $\varrho \in]0, \rho]$.

Maximizing lifetime utility with respect to κ^J yields the following indifference conditions, which equate the marginal benefit with the marginal cost of crime in each state:

$$\frac{\partial g(c_1^H(\kappa^H))}{\partial c_1^H} \times [1 - w] = \rho \frac{\partial b(\kappa^H)}{\partial \kappa^H}, \tag{4}$$

$$\frac{\partial g(c_1^S(\kappa^S))}{\partial c_1^S} \times [1 - \iota w] = [\rho - \varrho] \frac{\partial b(\kappa^S)}{\partial \kappa^S}, \tag{5}$$

with Equation (4) for the healthy and Equation (5) for the sick state. The left-hand side of each equation represents the marginal utility of obtaining extra income when replacing legal with illegal work—that is—the marginal benefit of crime, $MB(\kappa)$. The right-hand side of each equation represents the marginal disutility when replacing legal with illegal work—that is—the marginal cost of crime, $MC(\kappa)$. The framework allows us to explore how the incentive to commit crime changes as marginal costs and benefits differ between the healthy and the sick states.[4]

[4]Equations (4) and (5) are only necessary (first-order) conditions for maxima. To ensure a global utility maximum in the domain of interest, $0 < \kappa^J < 1$, we further assume $b(0) = 0$, $g(0) = 0$, and $\lim_{\kappa^J \to 1} b'(\cdot) = \infty$. A richer model could allow for corner solutions by, for example, adding present bias preferences or entry costs into the illegal labor market.

We further allow for the possibility that the health state influences a person's preferences: in particular by assuming that, in the sick state, absolute risk aversion is lower. We define absolute risk aversion as $A^J(c_1^J(\kappa^J)) = -g^{J\prime\prime}(c_1^J(\kappa^J))/g^{J\prime}(c_1^J(\kappa^J))$ and consider the possibility that $A^S(\cdot) < A^H(\cdot)$. In words, we allow for the health shock to reduce the absolute risk aversion of the individual.[5] The propositions below follow:

D.2. *The impact of health shocks*

PROPOSITION D.1: *If $\iota < 1$ and $\varrho = 0$ then $\kappa^S > \kappa^H$. That is, a health shock that reduces total legal income generates an increase in the labor supply of illegal activities.*

Thus, individuals suffering a health shock that decreases their legal wage, which is not fully compensated by sickness benefits, will have a higher marginal benefit from committing crime. This will increase the labor supply to illegal activities.

PROOF: To understand how a lower legal wage in the sick state compared to the healthy state affects the supply of illegal activity κ, we assume that the survival probability across health states is constant, thereby fixing the marginal cost of criminal activity.

The optimal choice for κ^S is given by the following indifference condition which equates the marginal benefit of additional labor supplied to crime to its marginal cost:

$$\frac{\partial g(c_1^S(\kappa^S))}{\partial c_1^S} \times [1 - \iota w] = \rho \frac{\partial b(\kappa^S)}{\partial \kappa^S}. \tag{6}$$

The left-hand side presents the marginal utility of an additional unit of consumption multiplied by the wage differential between illegal (1) versus legal work ($\iota w < 1$). The right-hand side presents the probability of surviving to the second period times the marginal expected disutility of punishment for an additional unit of labor supply to crime.

The optimal choice for κ^H is given by

$$\frac{\partial g(c_1^H(\kappa^H))}{\partial c_1^H} \times [1 - w] = \rho \frac{\partial b(\kappa^H)}{\partial \kappa^H}. \tag{7}$$

[5] Note that we assume that health shocks decrease risk aversion to explain the increase in the propensity of committing a crime. Under more stringent conditions, it is possible to show that an increase in risk aversion can also lead to more crime (see Proposition D.3 below).

Note that the marginal cost per unit of criminal activity remains constant in both health states. However, the marginal benefit of criminal activity increases in the sick state, as $1 - \iota w > 1 - w$. Then the result follows from the fact that $g(\cdot)$ is increasing and concave in κ and $b(\cdot)$ is increasing and convex in κ. *Q.E.D.*

COROLLARY D.1: *If sickness compensation (λ) is smaller or if the negative impact on earnings (α) is larger, then the health shock increases the labor supply to criminal activity more.*

The marginal benefit of crime increases more if the health shock results in a larger decrease in the legal income including sickness benefits. This increases the labor supply to illegal activities.

PROPOSITION D.2: *If $\iota = 1$ and $\varrho \in]0, \rho]$ then $\kappa^S > \kappa^H$. That is, a health shock that reduces survival probabilities generates an increase in the labor supply of illegal activities.*

Individuals suffering from a health shock that decreases their survival probability face a lower marginal cost of crime. This is because the health shock increases the discount rate of future consumption and punishment. This, in turn, increases the labor supply of illegal activities.

PROOF: To understand how a decrease in survival probability affects the supply of illegal activity κ, we assume that the ability to generate income across health states is constant, thereby fixing the marginal benefit of criminal activity at identical levels across health states. The optimal choice for κ^H and κ^S are given by

$$\frac{\partial g(c_1^H(\kappa^H))}{\partial c_1^H} \times [1 - w] = \rho \frac{\partial b(\kappa^H)}{\partial \kappa^H}, \tag{8}$$

$$\frac{\partial g(c_1^S(\kappa^S))}{\partial c_1^S} \times [1 - w] = [\rho - \varrho] \frac{\partial b(\kappa^S)}{\partial \kappa^S}. \tag{9}$$

The health shock decreases the marginal cost per unit of crime, as $\varrho \in]0, \rho]$. A decline in survival probability corresponds to a downward shift of the marginal cost curve, as the cost of an additional hour of illegal work becomes comparatively lower since the probability of paying the penalty for crime is lower. Since $\rho - \varrho < \rho$, the result follows from the fact that $g(\cdot)$ is increasing and concave in κ and $b(\cdot)$ is increasing and convex in κ. *Q.E.D.*

PROPOSITION D.3: *If $\iota = 1$, $\varrho = 0$, and $A^S(\cdot) < A^H(\cdot)$ then $\kappa^S > \kappa^H$ when $|g^{S\prime\prime}(\cdot)| \geq |g^{H\prime\prime}(\cdot)|$. That is, under specific assumptions on the shape of the utility function, a health shock that reduces risk aversion generates an increase in the labor supply of illegal activities.*

Individuals suffering from a health shock may face changes in their preferences potentially resulting in a higher marginal benefit of crime. This increases the labor supply of illegal activities.

PROOF: To understand how a change in preferences elicits an increase in the labor supply of illegal activities, we fix the legal wage and the survival probabilities at identical levels across health states. In this way, we investigate the implications of a change in the curvature of the utility function $g(\cdot)$.

We rewrite the FOCs as:

$$-g^{H\prime\prime}(c(\kappa^)/A^H(c(\kappa^) \times (1-w) = \rho b'(\kappa^), \tag{10}$$

$$-g^{S\prime\prime}(c(\kappa^)/A^S(c(\kappa^) \times (1-w) = \rho b'(\kappa^), \tag{11}$$

then, we have two scenarios to consider:

1. If $|g^{S\prime\prime}(\cdot)| \geq |g^{H\prime\prime}(\cdot)|$ then, as $A^S(\cdot) < A^H(\cdot)$, function $g(\cdot)$ is concave and increasing in κ^J and $b(\cdot)$ is increasing and convex in κ^J, we have that the marginal benefit of crime is higher in the sick state and, therefore, $\kappa^S > \kappa^H$.
2. If $|g^{S\prime\prime}(\cdot)| < |g^{H\prime\prime}(\cdot)|$ then whether the marginal benefit of crime is higher or lower in the sick state depends on the functional form of $A^J(\cdot)$ and $g^J(\cdot)$.

Q.E.D.

APPENDIX E: 5-YEAR SURVIVAL RATE ESTIMATION

We estimate the decline in the 5-year survival probability due to cancer in three steps. We first use matching to select a set of cancer treated as well as undiagnosed control individuals. We then estimate the 5-year death probability for each individual in our sample. Finally, we define as having low survival probability those individuals who, in the diagnosis year, face an above-median increase in death probability conditional on their gender.

To select the set of control individuals, we rely on exact matching. Specifically, for each diagnosed individual in our baseline sample, we consider only those individuals in the

population who, in the same year (our reference year is the year before the diagnosis), are of the same age, have the same marital status, the same gender, and are in the same ventile of income and quintile of years of education. Treated and control observations without a match are dropped. Out of all available matches, we select ex post a maximum of ten control individuals who exhibit the smallest difference in total income.

We then estimate the 5-year death probability of individuals between the age of 18 and 62 in the years from 1980 to 2013. Notably, we do not include data from 2014–2018, as we cannot establish whether people will die within 5 years (as our sample ends in 2018). The estimation sample consists of people who are diagnosed with cancer between 1980 and 2013 and a control sample extracted from the entire population. We estimate a logit model whereby p is the probability that person i has 5 years or less left to live:

$$\log\left(\frac{p_{i,t}}{1-p_{i,t}}\right) = \beta_t + \sum_{\nu=0}^{15}\theta_\eta \mathbb{1}\{\eta_{i,t}=\nu\} + \sum_{\nu=0}^{15}\gamma_\eta \mathbb{1}\{\eta_{i,t}=\nu\}A_{i,t} + \zeta A_{i,t} \tag{12}$$

$$+ \vartheta \mathbb{1}\{Post\ cancer\} + \lambda Z_{i,t}, \tag{13}$$

where $\eta_{i,t}$ takes a value from 0 to 15, where every number from 1 to 14 corresponds to a different type of cancer diagnosed in year t, and 0 indicates that the person has not been diagnosed with cancer in year t. The index of 15 is reserved for individuals diagnosed with multiple types of cancer. $A_{i,t}$ is a vector containing a fourth-order polynomial of age. $\mathbb{1}\{Post\ cancer\}$ is an indicator variable that takes a value of 1 if a person has been diagnosed with cancer in the past, excluding the diagnosis year. $Z_{i,t}$ includes gender and married controls. β_t are calendar year fixed effects. We estimate this equation separately on three different periods, 1980–1992, 1993–2003, and 2004–2013, to account for the effect of advances in cancer treatment that can alter the coefficients above.

We then proceed in three steps. We first rely on the estimates above to predict the probability that treated observations will die within five years of the diagnosis ($\hat{p}$). Second, we predict the counterfactual probability of being dead in five years in the case the person had not been diagnosed with cancer (we impose $\eta_{i,t}=0$). We define the difference between these two probabilities as the decline in survival probability due to cancer. The average decline in survival probability for each cancer type are listed in Table I.IV.

Finally, we sort individuals into groups based on their survival probability decline, conditional on their gender. We begin by dividing the sample into two categories based on

whether the survival probability decline is above or below the median. As a second more nuanced categorization, we split the sample into five groups, each determined by the quintile of the survival probability decline. We then estimate the main specification separately for each subgroup. While the results from the quintile split are elaborated on in the main paper, the outcomes from the binary split can be found in Figure I.8. Both analyses confirm the presence of a survival probabilities mechanism: individuals for whom cancer induces a larger decline in survival probabilities increase criminal activity to a larger extent than individuals with better odds of surviving.

APPENDIX F: OTHER MECHANISMS

In this section we consider two other mechanisms that may explain in part the effect of cancer on crime.

Preference mechanism. 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, 2009/2010, and 2020. These 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 proxies for risk aversion a dummy that equals one if the person makes a risk-averse choice in more than half of the tasks and the fraction of risk-averse choices made in the tasks. These experiments form the basis of Andersen et al. (2008), Andersen et al. (2014), and Andersen et al. (2024) to which we refer the reader for a detailed description of the experimental design.

Using this pooled cross-sectional data, Table I.V shows the relation between health shocks and risk preferences for 58 individuals who have already been diagnosed with cancer (treatment group) or will be in the future (control group). $Post\ cancer$ equals one if a person has been diagnosed in any of the previous 10 years. We find no significant relation between cancer and risk aversion.[6] To confirm this result, we further examine the

[6] As Åkerlund et al. (2016) and Epper et al. (2022) find an important role for time discounting in explaining criminal behavior, we also examine the effect of health shocks on time preferences. However, we use a smaller sample as only the experiments in 2003/2004 and 2009/2010 contain questions to measure time preferences. 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.

effect of cancer on the likelihood of receiving speeding tickets—as an alternative proxy of decreased risk-aversion—in our baseline sample of 5,007,687 observations (see Panel a of Online Appendix Figure I.12). The results indicate no effect on speeding. Finally, we examine the relation between cancer and risk aversion in longitudinal data from the University of Michigan Health and Retirement Study (HRS) (University of Michigan 2022). This alternative dataset covers the years 1992-2018 and contains bi-annual self-reported cancer diagnoses and risk attitude measurements based on choices between pairs of jobs out of which one guarantees the current family income and the other is risky. We conduct a staggered adoption analysis analogous to our main specification (see Panel b of Online Appendix Figure I.12). Also in this exercise, we do not find empirical support for a preference channel in the data.

Psychological distress mechanism. We further explore whether the uptick in crime is in part driven by psychological distress. To that end, we link our dataset to data on medical visits. Individuals are defined to be (or not to be) in psychological distress if they have (not) received treatment by a psychologist or psychiatrist following the cancer diagnosis. We then conduct two analyses. First, we explore the effect of cancer on the propensity to receive psychological treatment. Second, we estimate our standard treatment effects on each sample separately (i.e., in/not in psychologically distress). The results in Table I.VI show that 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 seek help. Together with the finding that part of the additional crimes due to cancer are not economically motivated, this evidence suggests that there might be a psychological distress mechanism behind some of the crimes.

APPENDIX G: ESTIMATING THE GENEROSITY OF MUNICIPALITIES

The analysis on the effects of the 2007 Danish municipality reform presented in Section 5.4 is conducted in two steps. First, we estimate the 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 face the largest decrease in social support from their municipality due to the reform. In this section, we describe in detail the first step. Details on the second step and the results are presented in the paper.

Step 1: Estimating the change in municipalities' generosity. As we do not observe directly the social policies put in place by each municipality, we infer the variation in welfare's generosity from the data. We expect that more generous social policies will mitigate to a greater extent the adverse impact on total income (labor income and social transfers) and thus will be reflected into a lower income decline caused by cancer (Corollary D.1 of the model). Note that we also consider labor income besides social transfers because a number of welfare policies consist of re-integrating people with disabilities in a work environment. If those policies are prevalent, a municipality's "generosity" will be reflected into a relatively higher share of labor income preserved post cancer rather than into larger income transfers. In sum, our approach defines as generous those municipalities that minimize the average loss of income streams induced by the local population's health shock with respect to pre-cancer levels.

To estimate the practical implications of the municipality reform for income, we first run the following specification:

$$\Delta Income_{i,t}^{R} = K_{m}^{R} + \lambda Z_{i,t}^{R} + \beta_{t}^{R} + \epsilon_{i,t}^{R}, \tag{14}$$

where $\Delta Income_{i,t}^{R}$ is the percentage change in the sum of labor income and additional income transfers earned by person i in year t after the cancer diagnosis, with respect to the average over the five years before the diagnosis.[7] $\Delta Income_{i,t}^{R}$ is thus only defined in the years *after* the initial diagnosis. To generate this variable, we exclude individuals who have been diagnosed with cancer between 2004 and 2006, to remove the effect of the runup to the municipality reform. K_{m}^{R} are municipality fixed effects that capture the average change in income after cancer *at the municipality level* net of the effects of individual-specific characteristics ($Z_{i,t}$) and time trends (β_t). The vector $Z_{i,t}$ includes age fixed effects and a third order polynomial of income rank in the year before the diagnosis. We include controls based on income because welfare support in Denmark is allocated progressively. Therefore, high-net worth individuals will experience a comparatively higher decline in income (or, equivalently, will receive lower social support) regardless of the municipality's generosity. Notably, the superscript $R \in \{0, 1\}$ indicates whether the equation is estimated on the subsample that includes the calendar years before the reform (1987–2006) or after the reform

[7] The choice of a five-year window minimizes the effect of noise in our measurement of pre-cancer income levels. Results for alternative lengths are in any case qualitatively similar.

(2007–2018). Note that we only include years from 1987 onwards because, to generate the variable $\Delta Income_{i,t}$, we need five years of income data before the diagnosis. K_m^0 (K_m^1) are pre-reform (post-reform) municipality fixed effects.

We obtain the change in generosity induced by the reform as $\Delta G_m = K_m^1 - K_m^0$. Next, we categorize our sample into groups based on the change in support. In our main analysis, we sort municipalities into two groups based on whether their municipality of residence increased or decreased income replacement following the reform. In a robustness analysis presented in Online Appendix Figure I.9, we sort municipalities into four groups. We first split the sample as before based on whether their municipality of residence increased or decreased income replacement following the reform. Then we divide each group in two based on the within group median change in income replacement following the reform.

Step 2: Estimate the change in sensitivity of crime to cancer due to a change in welfare generosity. This step is presented in the paper.

APPENDIX H: DIFFERENCE-IN-DIFFERENCES APPROACH FADLON AND NIELSEN

We replicate our analysis employing a difference-in-differences approach that follows closely the methodology of Fadlon and Nielsen (2019, 2021). 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). We fix the time interval between treated and control observations to $\Delta = 6$ years. Individuals in the control group are assigned a placebo shock at time s, since they are actually diagnosed with cancer only at time $s+\Delta$. As in Fadlon and Nielsen (2019), the same individual can appear both in the treatment group and in the control group, but is never used as a control to himself.

We can then estimate the effect of cancer on crime for $\Delta - 1$ time periods using a difference-in-differences estimator. For more details, Druedahl and Martinello (2020) explicitly compare this approach to our baseline methodology in Online Appendix C of their paper. The regression specification is as follows:

$$C_{i,t} = \beta_{t,a} + \theta\, treat_i + \sum_{\tau \neq 1; \tau = -4}^{5} \eta_\tau \mathbb{1}\{T_{i,t} = \tau\} \tag{15}$$

$$+ \sum_{\tau \neq 1; \tau = -4}^{5} \gamma_\tau \mathbb{1}\{T_{i,t} = \tau\} \times treat_i + \lambda X_{i,t} + \epsilon_{i,t}, \tag{16}$$

where i indexes individuals, t the calendar year, a the age, 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 time relative to the year of diagnosis. $treat_i$ is an indicator that takes a value of one if the person is part of the treatment group. γ_τ therefore captures the effect of cancer on crime at event time τ. The vector $X_{i,t}$ includes $In\ prison$, and $Cancer\ recurrence$ fixed effects. Person fixed effects cannot be included since they would be collinear with the treatment variable. The standard errors are clustered at the person-treatment group level.

APPENDIX I: ADDITIONAL FIGURES AND TABLES

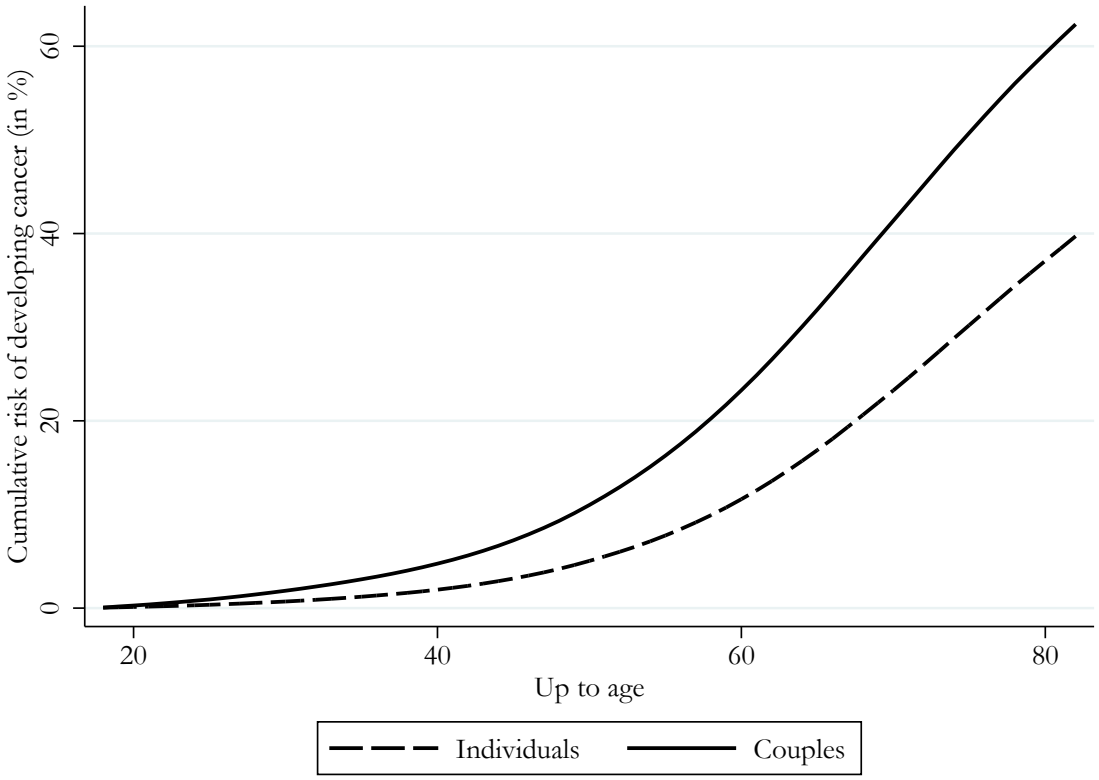


FIGURE I.1.—Risk of developing cancer by age. *Notes*: This figure reports the cumulative probability of developing cancer by age. The dashed line shows the risk of developing cancer over time for an individual and the solid line for either of the partners in a couple.

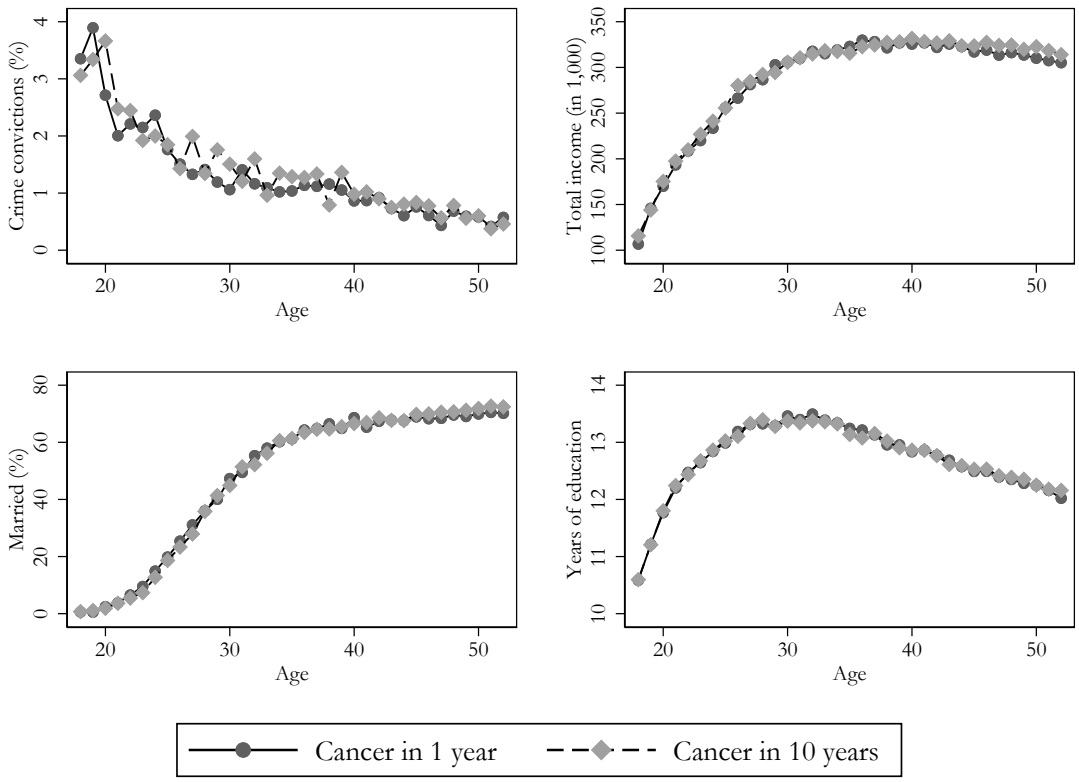


FIGURE I.2.—Comparison of individuals diagnosed in 1 versus 10 years. *Notes*: This figure compares average crime convictions (top left), total income (top right), marital status (bottom left), and education (bottom right) of individuals who will be diagnosed with cancer in 1 and 10 years, respectively. Averages are plotted against the ages of individuals.

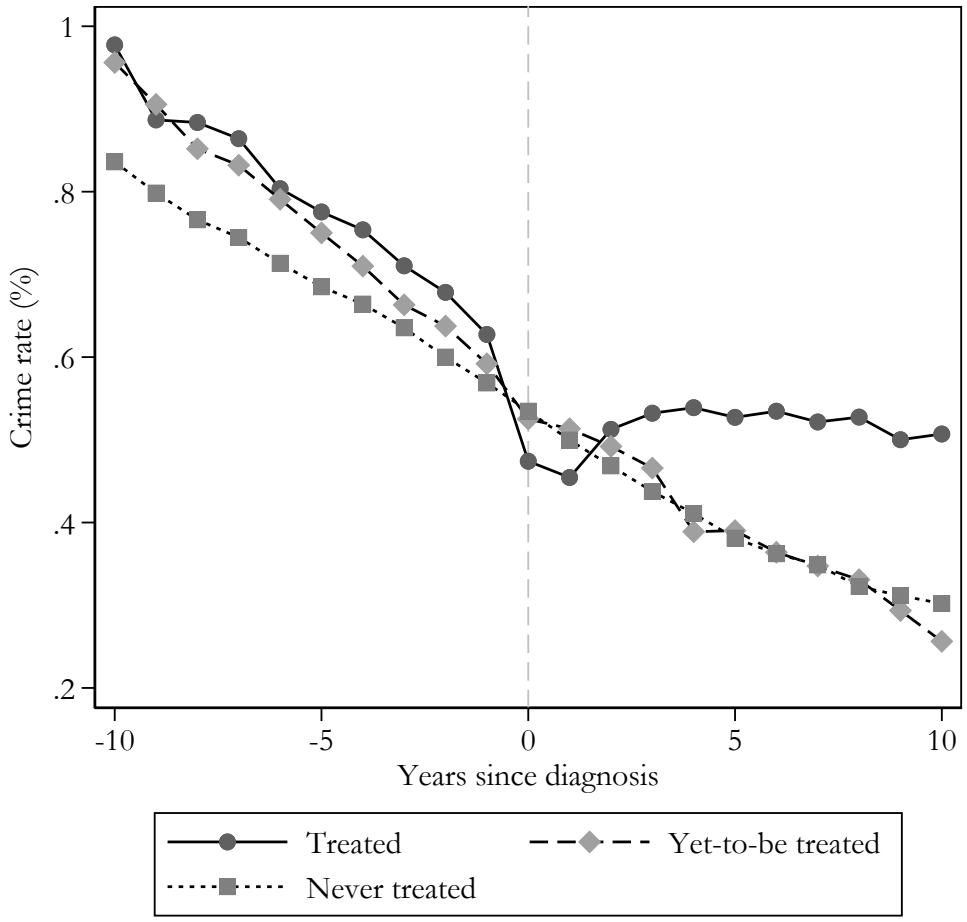


FIGURE I.3.—Raw crime rates across event years. *Notes*: This figure illustrates the average crime rates for three groups: the treated, yet-to-be-treated, and never treated populations. "Never treated" includes individuals who are never diagnosed with cancer. "Yet-to-be treated" includes individuals in our treated population up to the year before they are diagnosed with cancer. Event time 0 corresponds to the year of cancer diagnosis for the treated group and to the average age when people are diagnosed with cancer in our sample (the age of 52) for the yet-to-be-treated and never treated groups.

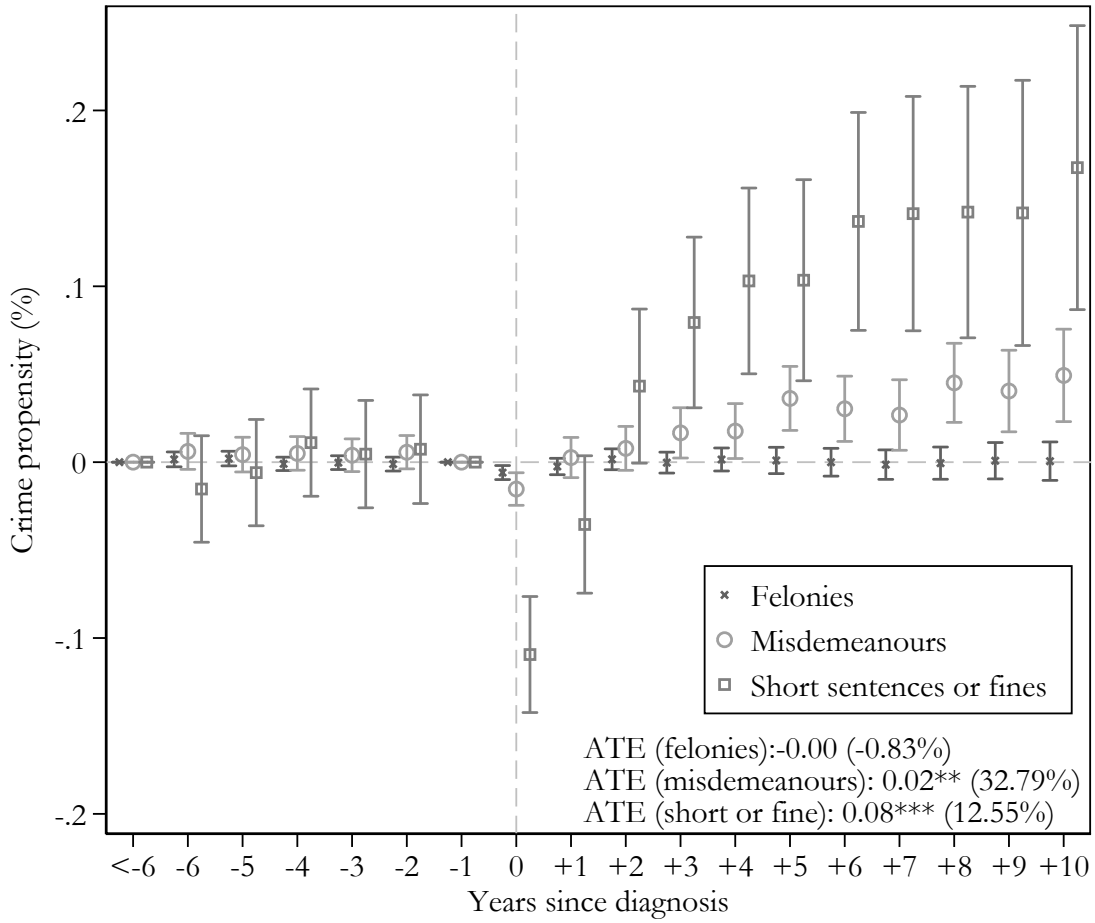


FIGURE I.4.—Effect on cancer on felonies, misdemeanours, and short sentences and fines. *Notes*: This figure reports event study estimates for criminal activity changes for different categories of crime in response to cancer diagnoses. Felonies are crimes for which the potential jail or prison sentence 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. 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 (ATE) is reported alongside the relative treatment effect (RTE) 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.

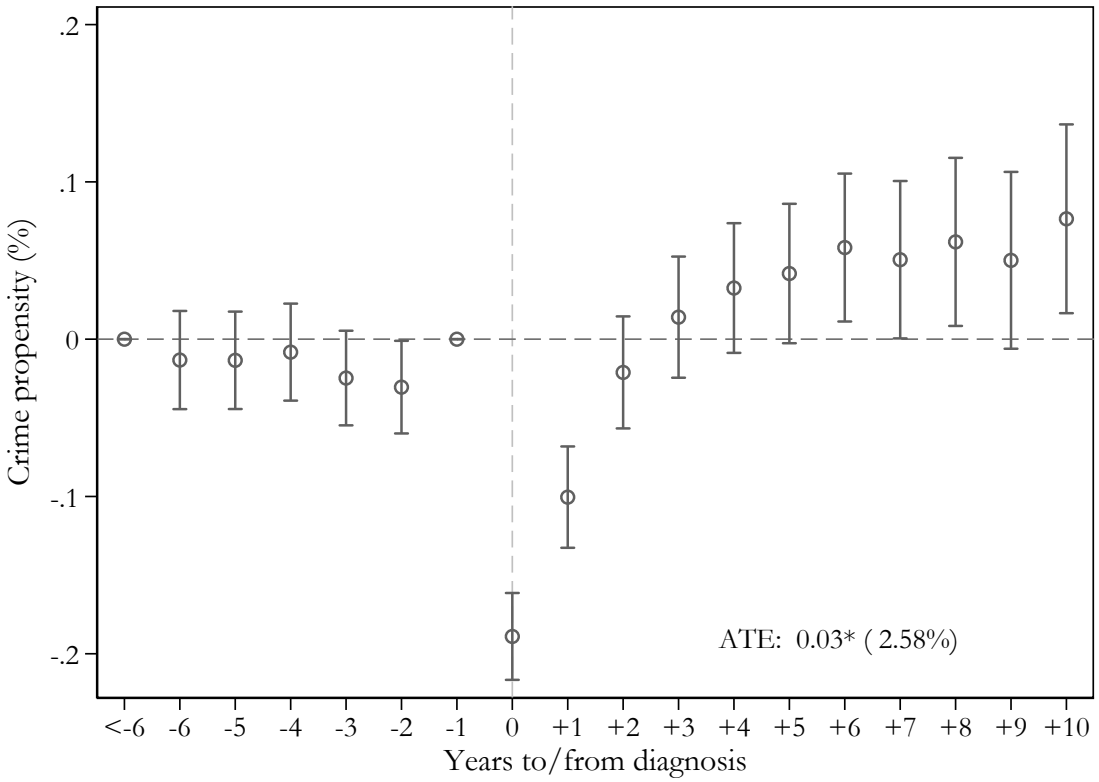


FIGURE I.5.—Effect of cancer on crime - Robustness using using never treated as control group. *Notes*: This figure reports event study estimates for criminal activity changes in response to cancer diagnoses. The sample includes both cancer patients and never-treated individuals. The figure plots the estimated coefficients along with their 95% confidence interval. The y-axis denotes crime propensity.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 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 person level. The number of observations is 12,043,684. ***, **, * indicate statistical significance at the 1%, 5%, and 10% levels, respectively.

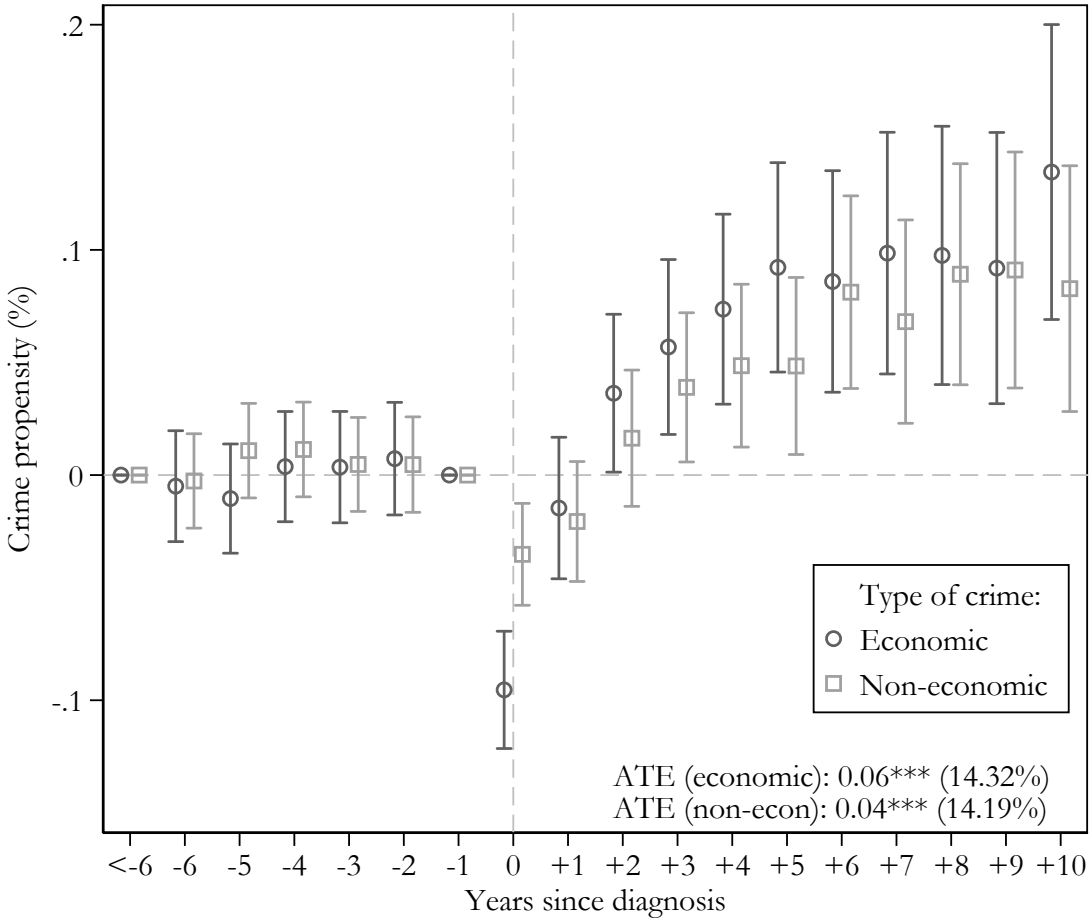


FIGURE I.6.—Effect of cancer on economic and non-economic crimes using alternative classification. *Notes*: This figure reports event study estimates for changes in economic and non-economic crime in response to cancer diagnoses, using an alternative classification than in the paper. In this classification, crimes that are labelled "Unclassified" in Online Appendix Table I.I are included in the "Non-Economic" crimes category. 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 (ATE) is reported alongside the relative treatment effect (RTE) 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 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.

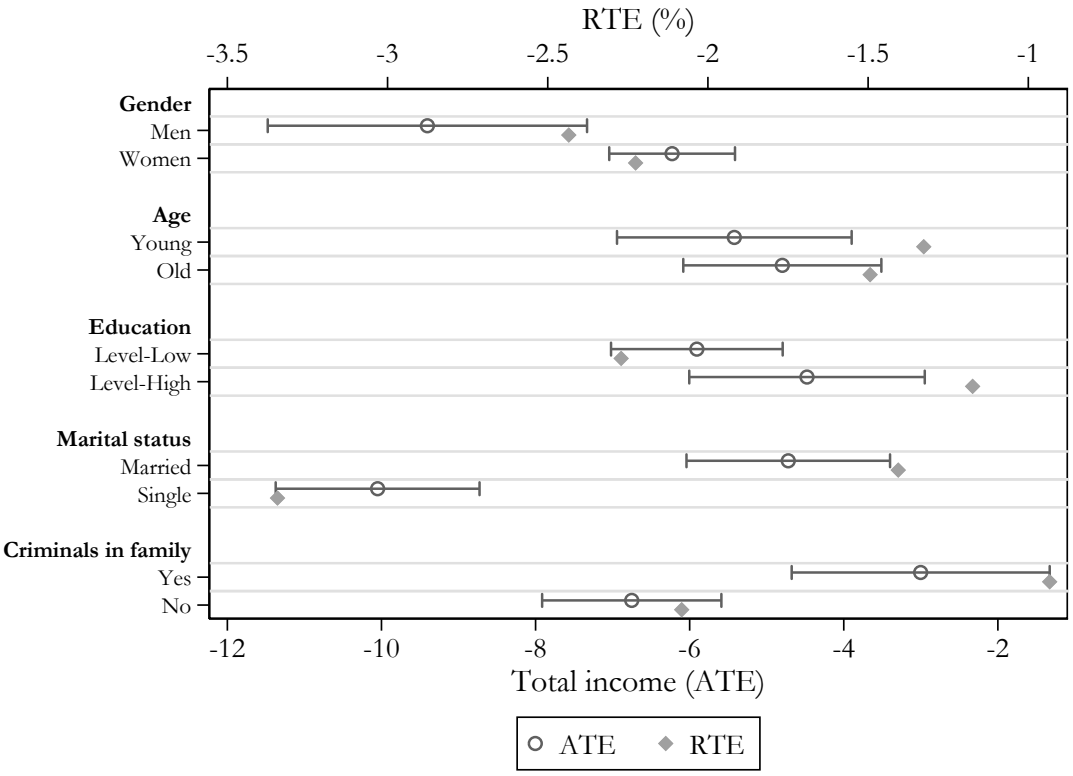


FIGURE I.7.—Heterogeneous effects of cancer on total income. *Notes*: This figure reports average treatment effects for changes in total income in response to cancer diagnoses for different subgroups. Individuals are sorted by 1. gender; 2. above- (respectively below-) median age in the year before diagnosis; 3. above- (respectively below-) median length of education in the year before diagnosis; 4. married (single) in the year before diagnosis; and 5. (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.

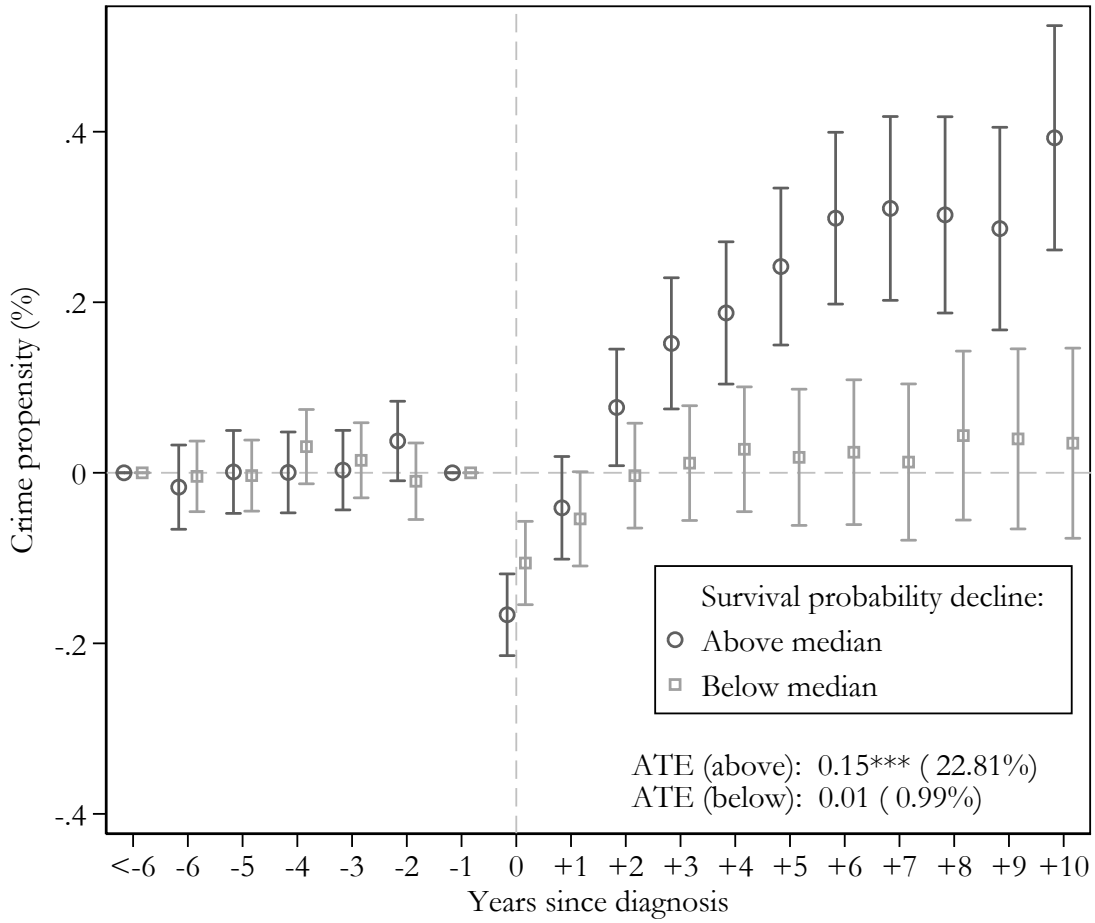


FIGURE I.8.—Survival probabilities mechanism - Robustness using two groups. *Notes*: This figure reports average treatment effects for criminal activity changes in response to cancer diagnoses for two groups based on their survival probability. Individuals are sorted on the basis of whether they face an above- (respectively below-) median decline in survival probability due to cancer. 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. ***, **, * indicate statistical significance at the 1%, 5%, and 10% levels, respectively.

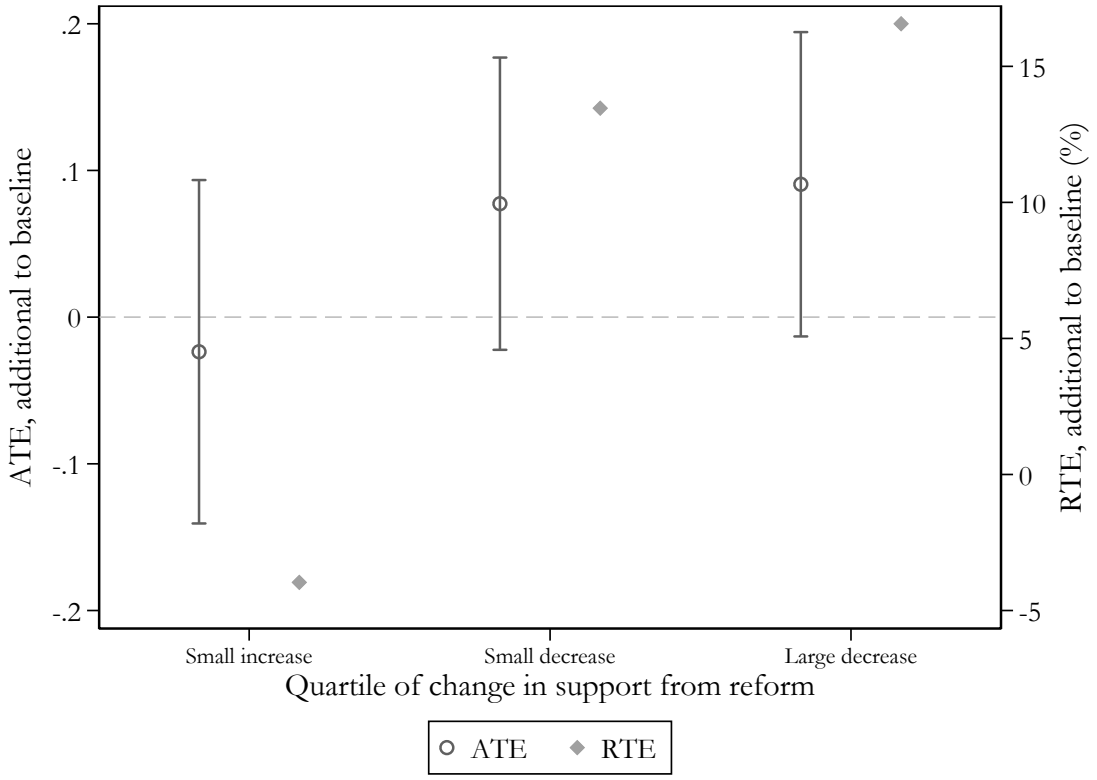


FIGURE I.9.—Change in welfare generosity and the effect of cancer on crime - Robustness using four groups. *Notes*: This figure reports average treatment effects for criminal activity changes in response to cancer diagnoses using a modification of the specification in Equation (2) in the paper. Instead of sorting municipalities in two groups, they are sorted in four groups. In step 1, municipalities are split into two groups based on whether they increased or decreased income replacement following the reform. In step 2, each group is further divided in two based on the within group median change in income replacement following the reform. The average treatment effects are indicated with circles and the relative treatment effects with diamonds. The x-axis denotes three categories of municipalities, where "Large increase" is the baseline category. 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 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.

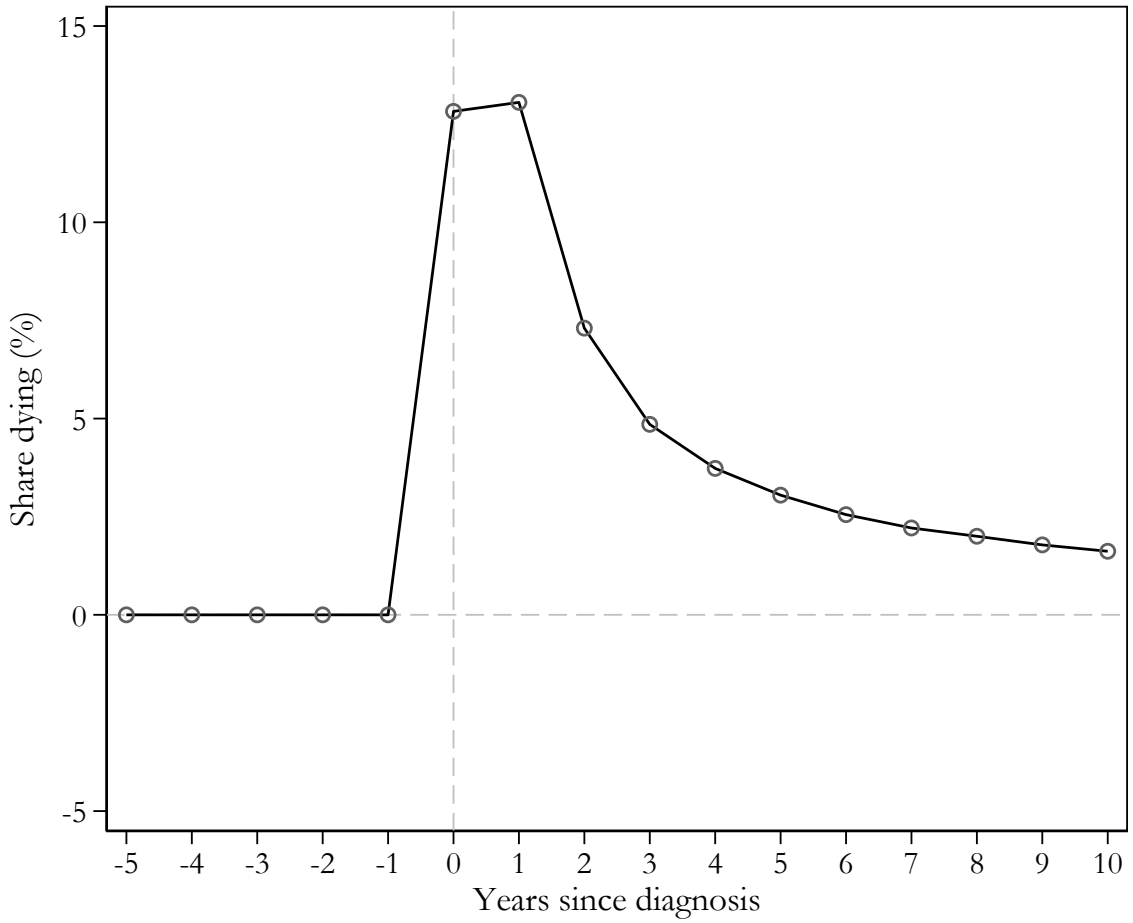
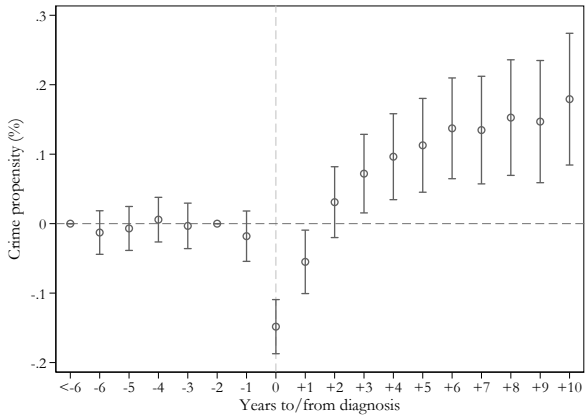
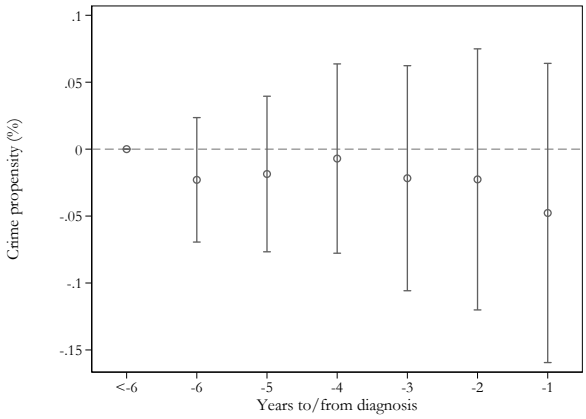


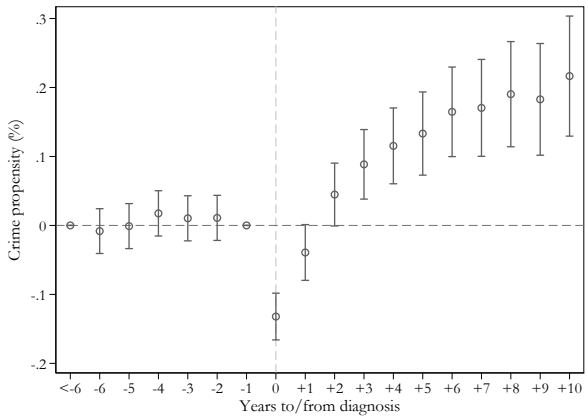
FIGURE I.10.—Attrition by event year. *Notes*: This figure reports the percentage of individuals who have died within each event year.



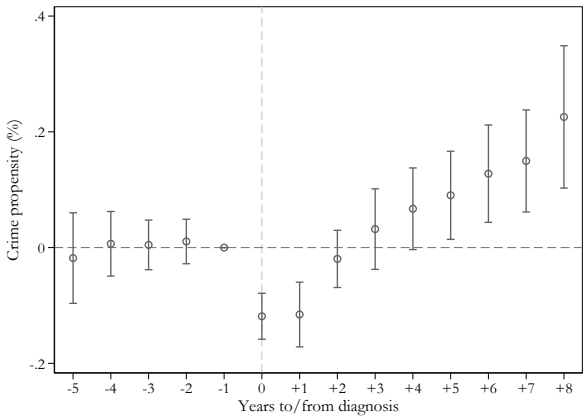
(a) Pre-trend anticipation



(b) Pre-trend Borusyak, Jaravel, and Spiess (2021)

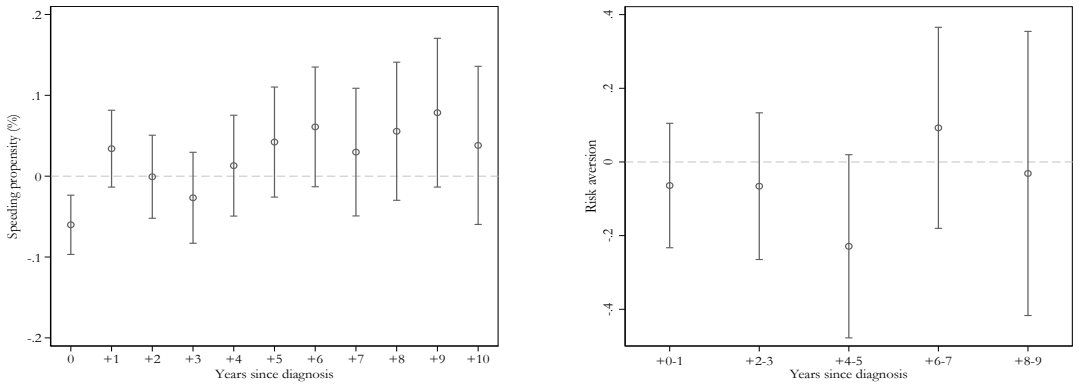


(c) Pre-trend Sun and Abraham (2021)



(d) Pre-trend Chaisemartin and D'Haultfœuille (2020,2021)

FIGURE I.11.—Robustness tests for pre-trends. *Notes*: This figure reports event study estimates for criminal activity changes in response to cancer diagnoses. In panel a, event time $\tau = -2$ (rather than event time $\tau = -1$) is excluded. In panel b, the event study estimates are obtained using the methodology developed by Borusyak, Jaravel and Spiess (2024), which uses only observations before treatment. In panel c, the event study estimates are obtained using the methodology developed by Sun and Abraham (2021). In panel d, the event study estimates are obtained using the methodology developed by De Chaisemartin and D'Haultfœuille (2020), De Chaisemartin and d'Haultfoeuille (2024). The figures plot the estimated coefficients along with their 95% confidence interval. The x-axis denotes time with respect to the year of diagnosis. The y-axis denotes crime propensity in percentage points. The empirical model includes person, year-by-age, in prison, and cancer recurrence fixed effects. Standard errors are clustered at the person level.



(a) Speeding tickets (b) HRS data

FIGURE I.12.—Preference mechanism—Robustness. *Notes*: Panel a reports event study estimates for speeding violations in response to cancer diagnoses using the main sample. The x-axis denotes time with respect to the year of diagnosis. The y-axis denotes speeding propensity in percentage points. The empirical model includes person, year-by-age, in prison, and cancer recurrence fixed effects. Standard errors are clustered at the person level. Panel b reports event study estimates for risk aversion changes in response to cancer diagnoses using data from the University of Michigan Health and Retirement Study (HRS). The HRS is a bi-annual panel survey of a representative sample of approximately 20,000 Americans over the age of 50 for the period from 1992 to 2018. The dependent variable ranges from 1 to 4, where 1 is least risk averse and 4 is most risk averse. This variable is based on the respondent's choice between pairs of jobs out of which one guarantees the current family income and the other offers a chance to increase it but also carries the risk of a loss of income. The sample includes only survey respondents that have had cancer and that we observe both before and after the diagnosis. The sample contains 4,205 observations. 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 risk aversion. The empirical model includes person and year-by-age fixed effects. Standard errors are clustered at the person level.

TABLE I.I

TYPES OF CRIME[a]

Type of crime	% of total crime (1)	Classification economic/ non-economic (2)	Classification property/ sexual/violent (3)
Forgery	0.634	Economic	Property
Forgery with check	0.397	Economic	Property
Burglary of bank/business	2.162	Economic	Property
Burglary of house	1.039	Economic	Property
Burglary of uninhabited building	0.324	Economic	Property
Theft from car, boat, etc.	0.665	Economic	Property
Store theft	9.458	Economic	Property
Theft other	3.598	Economic	Property
Theft indoor vehicle	1.600	Economic	Property
Theft of motorcycle	0.401	Economic	Property
Theft of bicycle	0.691	Economic	Property
Other theft	0.254	Economic	Property
Illegal handling of lost goods	0.419	Economic	Property
Embezzlement	0.315	Economic	Property
Fraud	1.870	Economic	Property
Check fraud	0.211	Economic	Property
Breach of trust	0.125	Economic	Property
Extortion and usury	0.058	Economic	Property
Fraud against creditors	0.094	Economic	Property
Possession stolen goods	1.577	Economic	Property
Robbery	0.709	Economic	Property
Serious tax fraud	0.061	Economic	Property
Negligent possession of stolen goods	0.102	Economic	Property
Wealth offenses, such as insider trading, bribery	0.210	Economic	Property
Counterfeiting money	0.141	Economic	Unclassified
Sale of drugs	0.385	Economic	Unclassified
Drug smuggling	0.128	Economic	Unclassified
Illegal occupation	0.053	Economic	Unclassified
Taxes and excise laws	0.550	Economic	Unclassified
Sales drugs	0.385	Economic	Unclassified
Prostitution, fornication	0.063	Economic	Sexual
Incest	0.033	Non-economic	Sexual
Rape	0.179	Non-economic	Sexual
Heterosexual offenses with children under 12	0.059	Non-economic	Sexual
Sexual crime against children under 12	0.012	Non-economic	Sexual
Heterosexual offenses otherwise	0.078	Non-economic	Sexual
Sexual crime against children under 15	0.014	Non-economic	Sexual
Sexual crime otherwise	0.011	Non-economic	Sexual

[a]Continues on next page

TABLE I.I

TYPES OF CRIME—CONTINUED[a]

Type of crime	% of total crime (1)	Classification economic/ non-economic (2)	Classification property/ sexual/violent (3)
Homosexual offenses with children under 12	0.008	Non-economic	Sexual
Homosexual offenses otherwise	0.009	Non-economic	Sexual
Unwanted touching	0.116	Non-economic	Sexual
Indecent exposure	0.082	Non-economic	Sexual
Indecency other	0.147	Non-economic	Sexual
Crimes against life and body, such as help with suicide	0.130	Non-economic	Violent
Violence against homeless	0.517	Non-economic	Violent
Arson	0.237	Non-economic	Unclassified
Vandalism	2.395	Non-economic	Unclassified
Family crimes	0.027	Non-economic	Unclassified
Involuntary manslaughter accident	0.220	Non-economic	Unclassified
Unknown	0.009	Unclassified	Unclassified
Violence against official authority	0.873	Unclassified	Violent
Riot/disturbance against official authority	0.032	Unclassified	Violent
Murder	0.034	Unclassified	Violent
Attempted murder	0.053	Unclassified	Violent
Simple violence	3.472	Unclassified	Violent
Serious violence	0.689	Unclassified	Violent
Particularly serious violence	0.018	Unclassified	Violent
Intentional bodily harm	0.199	Unclassified	Violent
Severe intentional bodily harm	0.017	Unclassified	Violent
Negligent manslaughter/bodily harm	0.019	Unclassified	Violent
Crimes against personal freedom	0.099	Unclassified	Violent
Threats	0.759	Unclassified	Violent
Terrorism, spying, treason, etc.	0.520	Unclassified	Unclassified
Offense by public employee	0.020	Unclassified	Unclassified
Perjury	0.088	Unclassified	Unclassified
False statement, withholding information	0.601	Unclassified	Unclassified
Public harm	0.088	Unclassified	Unclassified
Received request, received, or violated order	0.154	Unclassified	Unclassified
Data exploitation, defamation, etc.	0.369	Unclassified	Unclassified
Arms act	2.027	Unclassified	Unclassified
Health and social law	0.422	Unclassified	Unclassified
Building and housing law	0.053	Unclassified	Unclassified
Environmental law	0.584	Unclassified	Unclassified
Employment protection act, etc.	0.844	Unclassified	Unclassified
Company law	0.175	Unclassified	Unclassified
Military law	0.774	Unclassified	Unclassified
Electricity and gas law, etc.	0.087	Unclassified	Unclassified
Other special legislation	0.022	Unclassified	Unclassified
Holding drugs	6.681	Unclassified	Unclassified

[a]This table shows crime statistics and classifications. Column (1) reports the percentage of each type of crime out of total crime. Column (2) reports the classification of crime into economic and non-economic crime. Column (3) reports the classification of crime into property, sexual, and violent crime based on the system used by Statistics Denmark. The total number of crimes in the population is 4,732,529 from 1980 to 2018.

TABLE I.II

PREDICTING THE TIMING OF THE DIAGNOSIS[a]

	Diagnosis in 1 year (1)	Diagnosis in 2 years (2)	Diagnosis in 3 years (3)
F-statistic	1.528	0.575	0.301
P-value	(0.164)	(0.750)	(0.937)
Observations	2,926,697	2,926,697	2,926,697

[a]This table reports *F*-statistics for the hypothesis that is possible to predict the timing of the cancer diagnosis using the variables $Total\ income$, $Financial\ wealth$, $Mortgage\text{-}to\text{-}income\ ratio$, $Homeowner$, $Married$, and $In\ prison$. The dependent variable is a dummy that takes a value of one if a person is diagnosed with cancer within one, two, or three years, respectively. Only people who will develop cancer within 10 years are included. The empirical model includes person and year-by-age fixed effects. P-values are reported in parentheses.

TABLE I.III

COMPARISON OF INDIVIDUALS DIAGNOSED IN 1 VERSUS 10 YEARS[a]

	Cancer in 1 year (1)	Cancer in 10 years (2)	Difference (3)
Crime rate (%)	0.86	0.93	0.07
High mortality (%)	46.20	45.53	-0.66***
Total income (in 1,000)	309.23	314.67	5.44***
Labor income (in 1,000)	256.52	264.71	8.19***
Financial wealth (in 1,000)	146.93	159.99	13.07
Homeownership (%)	42.42	43.39	0.97***
Married (%)	62.48	63.25	0.77***
Education (years)	12.60	12.61	0.01
Observations	132,880	132,880	265,760

[a]This table reports observable characteristics for two sub-samples of individuals who will be diagnosed with cancer in 1 year (column 1) and in 10 years (column 2), respectively. Individuals are matched on year, gender, and age. Column 3 reports the results from a *t*-test on the difference in averages between the two groups. ***, **, * indicate statistical significance at the 1%, 5%, and 10% levels, respectively.

TABLE I.IV

CANCER TYPES BY QUINTILES OF 5-YEAR SURVIVAL PROBABILITY DECLINE[a]

	(1) Q1: 6%	(2) Q2: 14%	(3) Q3: 23%	(4) Q4: 37%	(5) Q5: 67%
Lip, oral cavity, pharynx	1	1	5	7	3
Esophagus and stomach	0	0	0	1	10
Small intestine and colon	0	1	6	10	8
Rectum	0	4	5	7	5
Liver, gallbladder and pancreatic	0	0	0	0	10
Respitory system and intrathoracic organs	0	1	1	5	42
Breast	39	33	29	16	2
Female genital organs	3	7	9	20	5
Male genital organs	26	4	4	2	0
Bladder, urinary organs except bladder	0	2	12	8	2
Eye, brain, central nervous system	0	0	1	5	7
Lymphoma	8	12	5	2	2
Leukemia	0	3	5	7	4
Others and ill defined	25	36	24	17	19

[a]This table reports the percentage of cancer types in each quintile (Q1 to Q5) of the 5-year survival probability decline variable. Note that each column sums up to 100. The average decline in survival probability for each quintile is reported at the top of each column.

TABLE I.V

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

	Risk aversion dummy (1)	Risk aversion fraction (2)
Post cancer	0.138	-0.005
	(0.132)	(0.050)
Observations	58	58

[a]This table reports cross-sectional estimates for the relation between risk aversion and having had a cancer diagnosis. Our main sample is matched with risk aversion measures obtained from experiments conducted in 2003/2004, 2009/2010, and 2020. The dependent variable *Risk aversion dummy* is a dummy that equals one if the respondent makes a risk-averse choice in more than half of the tasks (average of *Risk aversion dummy* is 0.5). The dependent variable *Risk aversion fraction* is the fraction of risk-averse choices out of total tasks (average of *Risk aversion fraction* is 0.49). *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 (average of *Post cancer* is 0.52). Standard errors are presented in parentheses.

TABLE I.VI

PSYCHOLOGICAL DISTRESS CHANNEL[a]

	Panel A	Panel B	
Dependent variable:	Psychological distress	Crime Subsample: in psych. distress	Crime Subsample: not in psych. distress
Years from diagnosis	(1)	(1)	(2)
0	0.019***	0.088	-0.162***
	(0.000)	(0.056)	(0.018)
+1	0.029***	0.094	-0.071***
	(0.001)	(0.062)	(0.021)
+2	0.015***	0.105	0.022
	(0.001)	(0.067)	(0.024)
+3	0.007***	0.173**	0.074***
	(0.001)	(0.076)	(0.027)
+4	0.004***	0.227***	0.083***
	(0.001)	(0.085)	(0.029)
+5	0.003***	0.096	0.111***
	(0.001)	(0.087)	(0.032)
+6	0.002**	0.120	0.145***
	(0.001)	(0.093)	(0.034)
+7	0.001	0.320***	0.097***
	(0.001)	(0.109)	(0.036)
+8	0.000	0.297**	0.133***
	(0.001)	(0.117)	(0.040)
+9	-0.000	0.229*	0.140***
	(0.001)	(0.124)	(0.042)
+10	-0.000	0.105	0.200***
	(0.001)	(0.128)	(0.045)
ATE	0.010***	0.166**	0.063***
	(0.000)	(0.068)	(0.024)
Observations	3,969,869	444,061	3,525,808

[a]Panel A reports event study estimates for changes in psychological distress in response to cancer diagnoses. Panel B reports event study estimates for criminal activity changes in response to cancer diagnoses for two distinct groups. Column 1 (column 2) shows the coefficients for people in (not in) psychological distress during one of the years following the cancer diagnosis. Psychological distress is proxied by having received treatment by a psychologist or psychiatrist. average treatment effects (ATEs) are reported at the bottom of each column. 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-by-age, in prison, and cancer recurrence fixed effects. All coefficients in Panel B are multiplied by 100. Standard errors are clustered at the person level and presented in parentheses. ***, **, * indicate statistical significance at the 1%, 5%, and 10% levels, respectively.

TABLE I.VII

COMPARISON OF INDIVIDUALS WHO REMAIN IN THE SAMPLE OR EXIT THE SAMPLE DUE TO DEATH[a]

	Remain in sample (1)	Exit sample (2)	Difference (3)
Crime rate (%)	0.54	0.80	-0.26***
High mortality (%)	35.39	76.91	-41.53***
Total income (in 1,000)	335.22	285.29	49.93***
Labor income (in 1,000)	272.06	208.81	63.25***
Financial wealth (in 1,000)	219.81	173.33	46.48***
Homeownership (%)	50.09	41.05	9.05***
Married (%)	64.68	62.90	1.78***
Male (%)	38.54	50.17	-11.63***
Age	49.43	51.11	-1.68***
Education (years)	12.91	11.81	1.10***
Observations	239,780	126,480	366,260

[a]This table reports average values for two sub-samples based on whether individuals pass away during the 10 years following diagnosis (column 2) or remain alive (column 1). All variables, with the exception of *high mortality*, are measured at event date $\tau = -1$. *High mortality* equals one if the decline in survival probability from diagnosis is greater than the gender-specific sample median. ***, **, * indicate statistical significance at the 1%, 5%, and 10% levels, respectively.

TABLE I.VIII

TIME TO APPREHENSION[a]

Years since diagnosis	(1)
0	0.079
	(0.091)
+1	-0.023
	(0.112)
+2	-0.137
	(0.122)
+3	-0.015
	(0.125)
+4	0.084
	(0.138)
+5	-0.048
	(0.148)
+6	0.036
	(0.153)
+7	0.020
	(0.168)
+8	-0.220
	(0.183)
+9	-0.031
	(0.205)
+10	0.197
	(0.212)
ATE	-0.035
	(0.088)
Observations	21,369

[a]This table reports event study estimates for changes in the time from offense to apprehension as a response to cancer diagnoses. The dependent variable is the time in days between when a crime is committed and when the perpetrator is apprehended. If a person commits multiple crimes in a year, the variable equals the median time. Only observations in the year of the offense are included. The ATE is 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-by-age, and cancer recurrence fixed effects. Standard errors are clustered at the person level and presented in parentheses.

TABLE I.IX

DO CANCER RATES RELATE TO THE RATE OF CRIMES SOLVED IN A MUNICIPALITY?[a]

	Rate of crimes convicted (1)	Rate of property crimes convicted (2)	Rate of crimes charged (3)	Rate of property crimes charged (4)
Rate of cancer	0.711	0.358	0.634	0.264
	(0.439)	(0.279)	(0.676)	(0.684)
Observations	5,587	5,587	5,587	5,587

[a]This table reports estimates for the relation between the rate of cancer by municipality and the rate of crimes solved by municipality. The analysis is conducted at the municipality-year level. The independent variable is the rate of cancer diagnoses in the population by municipality. The dependent variables are the *Rate of crimes convicted* out of all crimes reported (column 1), the *Rate of property crimes convicted* out of all crimes reported (column 2), the *Rate of crimes charged* out of all crimes reported (column 3), and *Rate of property crimes charged* out of all crimes reported (column 4). The empirical model includes municipality and year fixed effects. Standard errors are clustered at the municipality level and presented in parentheses.

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