Decomposing Causal Mechanisms in Duration Models with Unobserved Heterogeneity

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Abstract

This paper develops a new econometric framework for optimal policy design which identifies the causal effects of a treatment policy regime and of the actual implementation of treatment when the treatment and outcome of interest are duration variables. We consider a situation in which, upon entering an initial state, agents are randomized to a policy regime that prescribes a stochastic propensity to future treatment. Thereafter, at different moments in time and depending on their policy regime, surviving agents are randomized to actually receive treatment. Our dynamic potential outcomes framework provides non-parametric identification of: the ex-ante effect of the policy regime on the probability of exit, which may include placebo-type information effects, the ex-post baseline effect of actually receiving treatment on the probability of exit within a given policy regime, and the additional ex-post interaction effect of the policy regime and actually receiving treatment. The paper considers settings with and without unobserved intermediate variables and presents an estimation procedure. We illustrate the framework using data from the US National Job Corps Study.

Keywords: Dynamic treatment evaluation, optimal policy, mediation analysis, non-parametric identification, survival models, crime economics.

1   Introduction

Experiments and quasi-experiments form the cornerstone of microeconometric evaluations. These methods use some exogenous variation to analyse the ex-post effect of a policy intervention, or treatment, on one or several outcomes of interest. However, ex-post evaluations may not be fully informative to a policy-maker interested in rolling out or changing a treatment. After expanding a treatment, parts or all of the assignment mechanism may become public knowledge. Forward-looking agents can take the information on the assignment mechanism into account and change their expectations or behaviour already prior to receiving the treatment (Rosenzweig and Wolpin, 2000). These pre-treatment changes can alter the composition of individuals receiving treatment but also the ex-post effect of the treatment (Chassang, Padro i Miquel, Snowberg, 2012). As a result, the causal effect measured in a randomized trial or a quasi-experimental evaluation may incompletely predict the full scope of effects were that treatment to be rolled out. Static treatment evaluation methods are well developed but poorly accommodate these types of dynamic situations in which there is some time between an initial randomization and the actual treatment intervention.

Problems of dynamic selection are particularly pronounced for duration settings in which the treatment is assigned at different moments in time and treatment is only observed if the duration to treatment is shorter than the duration to the outcome. The main dynamic selection problem is that agents who survive up until a given time are not a random subset of the population at time 0. As a result, the group of surviving agents who are treated at any time greater than 0 is also not random. Furthermore, the process of dynamic selection may differ across policy regimes if the latter interacts with unobserved variables or individual expectations determining exit outcomes (Ham and Lalonde, 1996). These dynamic selection problems are in addition to the usual selection problems often discussed in the static treatment evaluation literature. In this paper we mainly focus on dynamic selection problems in duration models but our discussion pertains to any outcome associated to a duration (e.g. accepted wages or health score).

This paper presents a new econometric framework for optimal policy design which identifies the causal decomposition effects of a treatment policy regime and of the actual implementation of treatment when the treatment and outcome of interest are both duration variables. More specifically, we consider a situation in which individuals are randomized to a *policy regime* upon entering a state at time 0. The policy regime dictates a stochastic propensity to future treatment among agents. Thereafter, at different moments in time and depending upon their policy regime, surviving agents are randomized to actually receive treatment. Our interest is in formulating a meaningful decomposition which allows us to compare how the policy regime and the treatment influence the probability that an individual exits the initial state within a fixed amount of time. Our dynamic potential outcomes framework develops non-parametric identification of: (i) the ex-ante effect of the policy regime on the probability of exit prior to treatment, (ii) the ex-post baseline effect of actually receiving treatment on the probability of exit within a given policy regime, and (iii) the additional ex-post interaction effect of the policy regime and actually receiving treatment on the probability of exit from the initial state.

The framework is particularly relevant for implementing social experiments in which one

wishes to study pseudo-placebo effects of the expectation of future treatment (Duflo, Glennerster, and Kremer, 2007; Bulte, et. al., 2014). The usual setup of randomized trials is to apply a treatment on an unsuspecting group of agents who do not anticipate the treatment. In many situations, the effect found in such a setup may be a poor predictor of the full set of effects once the treatment is expanded to a wider audience of people making forward looking decisions. Following the framework proposed in this paper, a two step experimental setup, the first in which agents are randomly assigned and informed to some degree of their propensity to future treatment, the second in which they are randomized to actually receive the treatment, can project different possible responses for when the treatment is rolled out, thereby providing additional information to a policymaker.

The dynamic potential outcomes framework we develop draws on several strands of literature in econometrics and statistics. It mainly builds on the discrete time dynamic treatment effect g-computation identification strategy of Robins (1986; 1997) and first introduced in economics by Lechner (2009) and Lechner and Miquel (2010). Although we employ a different identification strategy, our framework also finds some parallels with the model in Abbring and van den Berg (2005 section 4) which extends the approach of Abbring and van den Berg (2003a) to model ex-ante and ex-post effects when the treatment and outcome are duration variables, but does not present effects within a larger optimal policy framework and only allows point-identification under semi-parametric restrictions.[1]

More generally, although we employ a different nomenclature, our methods and discussion relate to the mediation analysis literature with roots in epidemiology.[2] The classical mediation analysis literature focuses on decomposing the direct and indirect effects of an initial treatment and a later mediator on an outcome of interest (Robins and Greenland, 1992; Pearl, 2001; Imai, Keele and Yamamoto, 2010).[3] Recent contributions have pushed the field further by allowing these models to account for selection on unobservables (Heckman, Pinto and Savelyev, 2013; Imai, et. al., 2011; Frölich and Huber, 2017; Wunsch and Strobl, 2018; Huber et. al., 2019). There are two main problems with trying to transpose the causal decompositions from classical mediation analysis to our setting. First, the decomposition used for natural effects in mediation analysis relies on counterfactual potential outcomes that by definition can never factually exist and which therefore can not offer any policy relevant interpretation. Second, papers using the other decomposition for controlled effects will, due to dynamic selection, not be able to separate effects of policy regime vs. treatment when the treatment and outcome are duration variables.

To overcome these limitations, our paper invokes a dynamic variation of a rank invariance assumption (Matzkin, 2003; Chernozhukov and Hansen, 2005) on the timing of exit. Our new dynamic rank invariance assumption is mainly of interest not in its formulation but in its

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[1] For an overview of the econometrics of dynamic treatment effects see Abbring and Heckman, 2007.

[2] Our nomenclature differs from the mediation analysis literature wherein the policy regime would be referred to as 'the treatment' and treatment in our case would be referred to as 'the mediator'. Our change in nomenclature reflects a different point of departure when decomposing causal effects but is for expositional purposes only.

[3] See Huber (2019) for an overview. For settings with mediation analysis using a duration outcome variable but a non-duration mediator see for instance VanderWeele (2011), Lange and Hansen (2011), Tchetgen Tchetgen (2013) in epidemiology, and Bijwaard and Jones (2018) in economics.

application, which generates causal effects within a novel optimal policy framework. Drawing from the IV-LATE and essential heterogeneity literature (Imbens and Angrist, 1994; Heckman and Vytlacil, 2005) we show that our dynamic rank invariance assumption characterises the sub-populations of interest in the causal decomposition.

The first set of identification results focuses on the setting in which the 'best' agents as ranked by a scalar index for initial unobserved characteristics will always exit first. This dynamic rank invariance assumption implies that, given a duration to treatment in one policy regime, we can find another treatment point in the alternative policy regime such that the distribution of time 0 unobserved heterogeneity is the same at both points conditional on observed baseline variables. This assumption ensures that we can decompose causal mechanisms for the untreated survivors at any time of treatment. Identification further calls upon dynamic unconfoundedness and overlapping support extensions of the static treatment effects literature. One of the special features of our dynamic rank invariance assumption is that it requires a necessary condition on the data, and this necessary condition is testable.

We then extend our framework to allow for time-varying unobserved intermediate variables which can dynamically influence agent exit decisions (VanderWeele,Vansteelandt, Robins, 2014). The introduction of unobserved intermediate variables has ambiguous behavioural implications for the interpretation of our 'best exit first' rank invariance assumption. To shed light on these implications we first present an assumption under which unobserved intermediate variables can be redefined as intermediate shocks from the point of view of the agent. We then propose a new assumption which allows us to integrate out the effect of unobserved intermediate variables by assuming positive and negative intermediate shocks have symmetric effects on the probability of survival for the not yet treated. Under this assumption we can still preserve non-parametric identification even in a model with unobserved intermediate variables. Since this new assumption is somewhat opaque, we further explain that the dynamic rank invariance assumption is satisfied if the underlying model for the untreated survivors follows a mixed proportional hazard structure. This implies again identification of all causal effects of interest when assuming there is dynamic information accumulation based on the history of unobserved intermediate variables.

For estimation we propose a straightforward and easy to implement continuous-time semi-parametric mixed proportional hazard model, and present some simulation results to assess the performance of our estimators. The methods in this paper are relevant to researchers interested in optimal policy design who seek to decompose causal mechanisms in duration models with minimal functional form assumptions about agent utilities, expectations and underlying search processes. Topics of application include health interventions, development evaluations, active labor market programs, or education reforms, among others.[4]

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[4]The literature on pre-treatment effects has touched on several fields of public policy. Examples include threat effects of active labor market policies (Black, Smith, Berger and Noel, 2003; Rosholm and Svarer, 2008), announcement effects of tax policy reform on female labor force decisions (Blundell, Francesconi and Van der Klaauw, 2010), or sorting in the housing market to evaluate the value of school facility investments (Cellini, Ferreira and Rothstein, 2010). Anticipation effects of treatment have also been studied using structural models as, for instance, Attanasio, Meghir, and Santiago (2012) who study them in a randomized experiment in Mexico (Progresa).

Finally, we provide a brief illustration of our framework and discuss optimal policy design using data from the US National Job Corps Study, which follows youths from disadvantaged backgrounds who were eligible for the Job Corps program and contains monthly information on whether these individuals find a job or are arrested. Within the context of our methods, the randomization of youths to the Job Corps program or control group serves as the policy regime and the time until a person finds a job takes the role of the treatment. The latter is assumed to be random for survivors conditional on the rich set of observed baseline covariates. We then apply our estimation model to separate: (i) the ex-ante effect of Job Corps on the probability of arrests, (ii) the ex-post baseline effect on arrests of obtaining a job while part of the Job Corps control group, (iii) the additional ex-post interaction effect on arrests of obtaining a job while part of the Job Corps program group.

The remainder of the paper proceeds as follows. We begin in the next section by presenting the potential outcomes framework, describe causal effects of interest, compare them to those from mediation analysis, and present the assumptions for non-parametric identification of ex-ante effects and ex-post effects. Section 3 explains how the non-parametric identifying assumptions can be preserved when allowing for unobserved intermediate shocks, provides our estimation model and discusses some simulation results. Section 4 presents our empirical application and results.

2 Evaluation framework

In this section we introduce the potential outcomes notation, define causal effects and develop the causal decomposition framework. As a running example, we consider a drug which is tested in a randomized trial and then rolled out. This example provides a simple introduction to our assumptions and causal framework, and illustrates the salient problems at hand. Our empirical application in section 4 complements this example and illustrates the broad application of our framework with microeconomic data from the Job Corps study.

Consider a setting in which a researcher is interested in evaluating the effect of a new drug to treat a viral disease, for instance a new antiretroviral drug to combat HIV. The usual approach to such an evaluation is to gather a group of sick individuals, randomize them to a treatment or control group, and compare their outcome(s) at some later time. Now let's say the randomized trial shows strong effects of the drug on reducing the probability of death within 3 years after administering the drug. Usually this is the point at which the researcher stops and leaves the wider implementation of the drug to a policymaker. However, once the drug is rolled out, sick individuals may change their behaviour already prior to receiving the drug given knowledge of its availability and positive effect in the randomized trial. They may, for instance, be more reckless in their general behaviour or may instead benefit from a reinforced psychological or emotional state in hope of recovery. As a result of these behavioural changes, the probability of death for sick individuals may be higher or lower already prior to receiving the drug just due to being in a world in which the drug is available as opposed to the randomized trial world in which the drug is administered unexpectedly. In addition, these changes in behaviour may interact with the drug itself and lead to changes in the effectiveness of the drug once it is

administered.

Altogether, the baseline effect measured in the randomized controlled trial may be a poor predictor of the overall effect of rolling out a new drug. The question that remains is how to compare and contrast the different causal mechanisms in a manner that helps a policymaker in designing an optimal policy with that particular drug. To this end, we develop a new potential outcomes framework.

2.1 Potential Outcomes and Treatment Effects

Our potential outcomes framework combines the dynamic presentation of Abbring and Heckman (2007) with the more familiar notation in static treatment and mediation analyses (Angrist, Imbens and Rubin, 1996; Imai, Keele and Yamamoto, 2010). We follow each agent from the moment of entering an initial state which is set as $t = 0$. This may be for instance the moment of contracting an illness, becoming unemployed, or leaving prison. Each agent type $u \in \mathcal{U}$ represents the initial conditions at $t = 0$ of a single agent, it is the realization of a one-dimensional non-negative unobserved random variable U. Since identification is non-parametric, we leave the conditioning on observed baseline ($t = 0$) covariates implicit for notational convenience. However, we do refer to covariates when relevant for the topic of selection on observables and when instructive on addressing the problem of time-varying covariates. As such, U is a single index scalar representing all variables unobserved by the econometrician which are relevant to the duration to exit outcome of an agent. The ordering of realizations u among agents is relevant and will be discussed further in relation to our dynamic rank invariance assumption.

At $t = 0$, agents are randomized to one of two policy regimes denoted by the random variable Z. Z is observed by the econometrician and may be partially or fully known by the agents. Agents can either be assigned to a baseline regime $Z = 0$ or to a new regime $Z = 1$. The policy regime is a special type of randomization which influences the timing of a future treatment by introducing constraints, incentives, and/or information of future treatment to agents. These factors can influence the timing of treatment directly through administrative constraints and indirectly by changing agent behaviour in the case of selection on observed baseline covariates. In our health science example, $Z = 0$ is the experimental setup in which sick people are not previously informed about the existence of a drug, $Z = 1$ is the world in which sick people are informed of the existence and effect of a drug after is has been rolled out.

To define durations to treatment we use a potential outcomes notation. Let the random variable S^z be the potential duration to treatment, or equivalently the treatment time, had an agent been subject to policy regime $Z = z$. The potential treatment time can take on any value $s > 0$. We consider the simplified setting in which there is only a single binary treatment which once allocated remains permanently thereafter. In our example, this would be the moment a sick individual begins the antiretroviral drug treatment. We further define the potential outcome $T^{z,s}$ as the duration to exit had the agent been subject to policy regime $Z = z$, and had he been potentially treated at $S^z = s$. In our example, $T^{1,12 \text{ months}}$ would be the potential time to death for a sick person in the world in which they knew the availability and effect of the drug ($Z = 1$), and first received the drug after 12 months of sickness ($S^1 = 12$

months).

Several comparisons can be made based on this notation. In this paper, our object of interest is the probability of exit within a certain time t given treatment at $S^1 = s$. Any comparison therefore reduces to formulating counterfactual potential outcomes to the following,[5]

$$\Pr(T^{1,s} \leq t) = \int_{u \in \mathcal{U}} \Pr(T^{1,s} < t | U = u) f_U du \tag{1}$$

The usual problem with formulating causal comparisons with this potential outcome is that counterfactuals are never observed. In static settings this is because an individual can only be observed in one of two states of the world: treated, or non-treated. In our dynamic setting there are a possibly infinite number of unobserved counterfactuals, one for each treatment time s in each of the policy regimes z. Furthermore, there is dynamic selection in the sense that U will influence the timing of exit, and some agents will exit before ever receiving treatment. This means the population treated at $S^1 = s$ is not necessarily the same as the population treated at any other time s^* nor as the full population. These factors combined imply that for any comparison $\Pr(T^{z,s} < t) - \Pr(T^{z,s^*} < t)$ of treated at two points $s, s^* < t$ in a given regime z, we cannot know whether a difference in effect is due to an actual change in the causal effect of treatment, or due to a change in the composition of treated due to dynamic selection (Heckman and Singer, 1984). Such comparisons are therefore of limited policy relevance.

To formulate potential outcomes in dynamic settings which mimic the usual idea of a non-treated counterfactual we elicit the following axiom,

Axiom A.I: Causal Arrow

$$T^{z,s} = T^{z,s^*} \qquad \text{if} \ \ T^{z,s}, T^{z,s^*} < s, s^*$$

This axiom is fundamental to the concept of causality. It says that an individual in two potential worlds, one in which he receives the treatment at future time s and one in which it is at some other later time $s^* > s$, will always make the same exit decisions prior to time s had he been subject to policy regime z and experienced the same life events up until time s in both worlds. The Causal Arrow Axiom follows a common tenet of causal inference studies which states that there is a uni-directional arrow of information on which a cause must precede its effect.[6] The Causal Arrow Axiom does not relate to observed variables and implies no

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[5]In this paper our outcome of interest is the probability of exit instead of the expected exit duration $\mathbb{E}[T^{z,s}]$. Focusing on the expected duration to exit may be hampered by the fact that in many duration settings a large fraction of exit outcomes are censored so the right tail of the exit distribution will be poorly approximated. Also, the expected potential exit duration outcomes can all be expressed as functions of the probability to exit. In discrete time we can write

$$\mathbb{E}[T^{z,s}] = \sum_{t=1}^{\infty} t \cdot \Pr(T^{z,s} \leq t | T^{z,s} \geq t) \cdot \Pr(T^{z,s} \geq t)$$

Alternatively, some studies focus on the relative effects of the potential hazard $\theta_t^{z,s} = \Pr(T^{z,s} = t | T^{z,s} \geq t)$ as causal effects. The drawback of focusing directly on the hazard to infer about causal effects is that its magnitude is difficult to interpret for cost-benefit analyses without transforming it into a survivor function.

[6]The Causal Arrow Axiom draws mainly from the so-called 'no-anticipation' assumption in Abbring and van den Berg (2003a), although our formulation follows more that of Abbring and Heckman (2007). Its initial

empirical restrictions. It simply helps formally define the non-treated counterfactual outcome for the treated at $S^1 = s$ in equation 1 as $T^{1,S^1 \geq t}$. This is the exit outcome duration had the individual not received treatment prior to the evaluation cutoff time t.

Under axiom $A.I$ we can already formulate some policy relevant causal effects. The first is the ex-post effect of the treatment within a given policy regime z,

$$
\begin{aligned}
\Pr(T^{z,s} < t) &- \Pr(T^{z,S^z \geq t} < t) \\
&= \Pr(T^{z,s} < t | T^{z,s} \geq s) \Pr(T^{z,s} \geq s) + \Pr(T^{z,s} < s) \\
&\qquad - \Pr(T^{z,S^z \geq t} < t | T^{z,S^z \geq t} \geq s) \Pr(T^{z,S^z \geq t} \geq s) - \Pr(T^{z,S^z \geq t} < s) \\
(A.I) &= \big[\Pr(T^{z,s} < t | T^{z,S^z \geq s} \geq s) - \Pr(T^{z,S^z \geq t} < t | T^{z,S^z \geq s} \geq s) \big] \Pr(T^{z,S^z \geq s} \geq s)
\end{aligned}
\tag{2}
$$

This effect compares those intended to be treated at s, 'intended' because some agents exit before actually receiving treatment, to the treated after t. Due to $A.I$, we also see from the above that $[\Pr(T^{z,s} \leq t) - \Pr(T^{z,S^z \geq t} \leq t)]/\Pr(T^{z,S^z \geq s} \geq s)$ is the effect of treatment at s on survivors. This is the usual effect of interest in dynamic treatment evaluations (Fredriksson and Johansson, 2008; Vikström, 2017). The second causal effect is the policy regime effect given by,

$$
\Pr(T^{1,S^1 \geq s} < s) - \Pr(T^{0,S^0 \geq s} < s)
\tag{3}
$$

This effect compares the probability of exit prior to receiving treatment for agents exposed to $Z = 1$ compared to those exposed to $Z = 0$.

While each of the above causal effects may be of interest on its own, the drawback is that they cannot be compared nor contrasted for optimal policy design purposes. For instance, we cannot simply compare ex-post effects for the treated at s in $Z = 0$ to the treated at s in $Z = 1$. This is because in general the distribution of unobservables for the surviving treated at s will be different across regimes when dynamic selection depends on the policy regime assignment (Ham and Lalonde, 1996; Eberwhein, Ham, Lalonde, 1997). There is also no clear policy relevant comparison to make between the ex-post effect for the (surviving) treated at s which compares treated and non-treated within a policy regime $Z = z$ and the policy regime effect which compares non-treated across policy regimes.

If we want a meaningful comparison of effects for optimal design purposes, we need to formulate an assumption which relates the treated at $S^1 = s$ to the same sub-population in terms of unobservables in policy regime $Z = 0$. In this paper, we propose a dynamic rank invariance assumption which draws from the quantile treatment effect literature (Chernozhukov and Hansen, 2005) to satisfy this comparability condition. We impose that agents exit in finite time[7], $T^{z,s} < \infty$, and that the following rank invariance assumption on the exit sequence holds,

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formulation was presented at a time when potential and observed variables were not clearly distinguished, leading to much confusion about its interpretation and empirical content. While we acknowledge the important contribution of Abbring and van den Berg (2003a), the present authors prefer a formulation and name that do not incorrectly presuppose empirical restrictions. The seminal work on dynamic treatment effects in the statistics literature by Robins (1986;1997), Gill and Robins (2001) takes the Causal Arrow Axiom as given and therefore never states it explicitly.

[7]Without this assumption, we can still define the rank invariance assumption at any time s for the set of agents who would have exited before time s under $Z = 0$ and $Z = 1$.

Assumption A.II: Dynamic Rank Invariance

For any two agents u_j, $u_k \in \mathcal{U}$,

$$T_j^{0,S^0=\infty} < T_k^{0,S^0=\infty} \quad \text{iff} \quad T_j^{1,S^1=\infty} < T_k^{1,S^1=\infty}$$

$$\text{and} \quad T_j^{0,S^0=\infty} = T_k^{0,S^0=\infty} \quad \text{iff} \quad T_j^{1,S^1=\infty} = T_k^{1,S^1=\infty}$$

This is a new application of a rank invariance assumption in the dynamic treatment effects literature. It states that the exit order with respect to u is preserved across policy regimes for not yet treated agents. In fact this assumption qualifies the definition of U by requiring that, if assumption $A.II$ can hold conditional on observed baseline covariates, then the ordering of realizations u at $t = 0$ is defined such that assumption $A.II$ must hold. Returning to our health science example, the rank invariance assumption imposes that the potential sequence of death when falling sick in a world in which the sick have some information about their exposure and the effect of the drug is the same as the sequence of death in the randomized trial world in which the sick do not anticipate receiving the drug nor its effect.

An immediate result of this assumption is that there exists a unique point in time s' in policy regime $Z = 0$ at which the distribution of unobserved heterogeneity for the untreated survivors is the same as at point in time s in policy regime $Z = 1$ as stated in the following Lemma,

Lemma 1. *Let F be a cdf, $F_{T^{1,\infty}}$ be the cdf of $T^{1,\infty}$, and $F_{T^{0,\infty}}$ be the cdf of $T^{0,\infty}$. Under Axiom A.I and Assumption A.II, for any given $S^1 = s$ we define $S^0 = s'$ by,*

$$s' = F_{T^{0,\infty}}^{-1}(F_{T^{1,\infty}}(s))$$

such that,

$$F(u|T^{0,S^0 \geq s'} \geq s') = F(u|T^{1,S^1 \geq s} \geq s)$$

In the above, s' corresponds to the same quantile of the $T^{0,\infty}$ distribution as s does to the $T^{1,\infty}$ distribution. In other words, the point in time $S^0 = s'$ is defined such that $\Pr(T^{0,S^0 \geq s'} \geq s') = Pr(T^{1,S^1 \geq s} \geq s)$ holds. The equivalence of the distribution of U for untreated survivors then follows immediately from the rank invariance assumption $A.II$. Of course, even if one can argue that the rank invariance assumption holds conditional on a sufficiently rich set of baseline covariates at $t = 0$, it is unlikely to hold exactly in practice if there are many unobserved intermediate variables at $t > 0$, such as health shocks, which influence the outcome probability. We address this problem in section 3 and discuss other possible empirical violations of this assumption later in this section.

Axiom $A.I$ and assumption $A.II$ allow us to reformulate equations 2 and 3 into comparable causal effects within a meaningful decomposition framework for optimal policy design. Towards constructing this decomposition, we set an overarching notation in which we define,

$$\mathbb{E}[Y^{1,1}(1, s, \triangle)] = \Pr(T^{1,s} < s + \triangle)$$
$$= \Pr(T^{1,s} < s + \triangle | T^{1,s} \geq s) \Pr(T^{1,s} \geq s) + \Pr(T^{1,s} < s)$$

Then, in the spirit of Gelman and Imbens' (2013) reverse causal questioning, the fundamental question that our decomposition seeks to answer is: *when evaluating the probability of exit at*

$s + \triangle$, *what different effects apply to the population of treated survivors at $S^1 = s$ in policy regime $Z = z$ compared to a world in which they were assigned to policy regime $Z = 0$ and were left untreated?*

To answer this question in terms of forward causal inference, first note that under *A.II* we know for each agent that $T_i^{1,s} < s$ if and only if $T_i^{0,s'} < s'$ which implies that if $s > s'$ and $T_i^{1,s} < s$, then $T_i^{0,s} < s$.[8] As a result, assuming $s > s'$, we define the following decomposition of causal effects for the population of agents subject to policy regime $Z = 1$, intended to be treated (ITT) at $S^1 = s$ and with ex-post evaluation interval $\triangle \geq 0$,

Proposition 1.(*Total Policy Decomposition*) *Let $s + \triangle \geq s > s'$. Under Axiom A.I and Assumption A.II we define the total policy decomposition by,*

$$\mathbb{E}[Y^{0,0}(1, s, \triangle)] = \mathbb{E}[Y^{1,1}(1, s, \triangle)] - \Delta_{ex-ante}^{ITT}(1, s) - \Delta_{ex-post\ base}^{ITT}(1, s, \triangle) - \Delta_{ex-post\ int.}^{ITT}(1, s, \triangle)$$

for which causal decomposition effects are given by,

Ex-ante effect:

$$\Delta_{ex-ante}^{ITT}(1, s) = \Pr(T^{1,S^1 \geq s} < s) - \Pr(T^{0,S^0 \geq s} < s)$$
$$(A.II) = \left[\Pr(T^{1,S^1 \geq s} < s | T^{1,S^1 \geq s} \geq s) - \Pr(T^{0,S^0 \geq s} < s | T^{1,S^1 \geq s} \geq s) \right] \Pr(T^{1,S^1 \geq s} \geq s)$$
$$(A.II) = - \Pr(T^{0,S^0 \geq s} < s | T^{0,S^0 \geq s'} \geq s') \Pr(T^{1,S^1 \geq s} \geq s)$$

Ex-post baseline effect:

$$\Delta_{ex-post\ base}^{ITT}(1, s, \triangle) = \Pr(T^{0,s'} < s' + \triangle) - \Pr(T^{0,S^0 \geq s' + \triangle} < s' + \triangle)$$
$$(A.II) = \left[\Pr(T^{0,s'} < s' + \triangle | T^{1,S^1 \geq s} \geq s) - \Pr(T^{0,S^0 \geq s' + \triangle} < s' + \triangle | T^{1,S^1 \geq s} \geq s) \right] \Pr(T^{1,S^1 \geq s} \geq s)$$
$$(A.II) = \Big[\Pr(T^{0,S^0 = s'} < s' + \triangle | T^{0,S^0 \geq s'} \geq s')$$
$$- \Pr(T^{0,S^0 \geq s' + \triangle} < s' + \triangle | T^{0,S^0 \geq s'} \geq s') \Big] \Pr(T^{1,S^1 \geq s} \geq s)$$

Ex-post interaction effect:

$$\Delta_{ex-post\ int.}^{ITT}(1, s, \triangle) = \Pr(T^{1,s} < s + \triangle) - \Pr(T^{0,s'} < s' + \triangle)$$
$$(A.II) = \left[\Pr(T^{1,s} < s + \triangle | T^{1,S^1 \geq s} \geq s) - \Pr(T^{0,s'} < s' + \triangle | T^{1,S^1 \geq s} \geq s) \right] \Pr(T^{1,S^1 \geq s} \geq s)$$
$$(A.II) = \left[\Pr(T^{1,s} < s + \triangle | T^{1,S^1 \geq s} \geq s) - \Pr(T^{0,s'} < s' + \triangle | T^{0,S^0 \geq s'} \geq s') \right] \Pr(T^{1,S^1 \geq s} \geq s)$$

The *ex-ante effect* is the change in the probability of exit prior to s due to changing the policy regime from $Z = 0$ to $Z = 1$ for those intended to be treated at $S^1 = s$, 'intended' in that the effect also includes the 0 effect for those who exit before period $S^1 = s$ when $Z = 1$. The *ex-post baseline effect* is the change in the probability of exit over interval $\triangle$ had those intended to be treated at $S^1 = s$ been exposed to policy regime $Z = 0$. The *ex-post interaction effect* is the additional change in effect of treatment over ex-post interval $\triangle$ due to being subject to policy regime $Z = 1$ rather than $Z = 0$. The ex-post interaction effect will therefore only be

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[8]All discussion and solutions are symmetric if $s < s'$.

non-zero if unobserved variables U interact non-trivially with the policy regime Z.[9] In section 3 we will provide a more general interpretation of these effects when allowing for unobserved intermediate variables.

A crucial point to note when understanding the above decomposition is that $\triangle$ and the treated population are held constant, and the ex-ante and ex-post effects are all equal to 0 for the agents who exit before period $S^1 = s$ when $Z = 1$, $T^{1,S^1 \geq s} < s$, and before period $S^0 = s'$ when $Z = 0$, $T^{0,S^0 \geq s'} < s'$. This implies that causal variation in all three effects is generated by the same untreated surviving population defined by $F(u|T^{1,S^1 \geq s} \geq s) = F(u|T^{0,S^0 \geq s'} \geq s')$.[10] For this reason, in contrast to equation 3 without assumption $A.II$, the ex-ante effect we propose guarantees that the effect applies to the same untreated surviving population as the ex-post baseline and interaction effects.

Furthermore, since the ex-ante and ex-post effects are all equal to 0 for the agents who exit before period $S^1 = s$ if $Z = 1$, and before period $S^0 = s'$ if $Z = 0$, a policymaker may be more interested in obtaining the causal decomposition on the sub-group of agents whose causal effects change due to the change in policy regime. These are the compliers for our setting in analogy to Imbens and Angrist (1994), Angrist, Imbens and Rubin (1996). This group of interest is the *sub-population of previously untreated survivors at s under policy regime $Z = 1$*. To obtain causal effects on this sub-population which we refer to as 'ATS', we must divide all three causal effects by $\Pr(T^{1,S^1 \geq s} \geq s)$.[11]

_Proposition 2.__(Total Policy Decomposition on Untreated Survivors) Let_
$\mathbb{E}[Y^{1,1}(1, s, \triangle)|T^{1,S^1 \geq s} \geq s] = \Pr(T^{1,s} < s + \triangle|T^{1,S^1 \geq s} \geq s)$ _and_ $s + \triangle \geq s > s'$. _Under Axiom A.I and Assumption A.II we define the total policy decomposition on untreated survivors,_

$$\mathbb{E}[Y^{0,0}(1, s, \triangle)|T^{1,S^1 \geq s} \geq s]$$
$$= \mathbb{E}[Y^{1,1}(1, s, \triangle)|T^{1,S^1 \geq s} \geq s] - \Delta^{ATS}_{ex-ante}(1, s) - \Delta^{ATS}_{ex-post\ base}(1, s, \triangle) - \Delta^{ATS}_{ex-post\ int.}(1, s, \triangle)$$

for which the causal decomposition effects on untreated survivors are given by,

$$\Delta^{ATS}_{ex-ante}(1, s) = -\Pr(T^{0,S^0 \geq s} < s|T^{0,S^0 \geq s'} \geq s')$$

$$\Delta^{ATS}_{ex-post\ base}(1, s, \triangle) = \Pr(T^{0,S^0 = s'} < s' + \triangle|T^{0,S^0 \geq s'} \geq s') - \Pr(T^{0,S^0 \geq s' + \triangle} < s' + \triangle|T^{0,S^0 \geq s'} \geq s')$$

$$\Delta^{ATS}_{ex-post\ int.}(1, s, \triangle) = \Pr(T^{1,s} < s + \triangle|T^{1,S^1 \geq s} \geq s) - \Pr(T^{0,s'} < s' + \triangle|T^{0,S^0 \geq s'} \geq s')$$

These are the effects we focus on throughout the rest of the paper. Figure 1 presents the intervals over which these exit probabilities are evaluated. We can further obtain the average effects over all potential treatment times by integrating over the feasible support of S^1.

---

[9] Throughout the paper we take the view that time itself is non-causal, it is only the medium upon which causality operates.

[10] Since we hold $\triangle$ constant, $\mathbb{E}[Y^{0,0}(1, s, \triangle)] \neq \Pr(T^{0,S \geq s + \triangle} < s + \triangle)$ unless the ex-ante effect is null.

[11] Since our focus is on dynamic selection problems, we do not discuss here settings in which untreated survivors can choose to always take the treatment when offered at s.

$$
\begin{array}{lll}
& \text{Ex-ante effect } s & \text{Ex-post interaction effect} \qquad s + \triangle \\
Z = 1 \quad 0 & & \\
& \Pr(T^{1,s} \geq s) & \Pr(T^{1,s} < s + \triangle) \\
\end{array}
$$

$$
\begin{array}{lll}
& s' & \text{Ex-post baseline effect} \qquad s' + \triangle \\
Z = 0 \quad 0 & & \\
& \Pr(T^{0,s'} \geq s') & \Pr(T^{0,s'} < s' + \triangle) \\
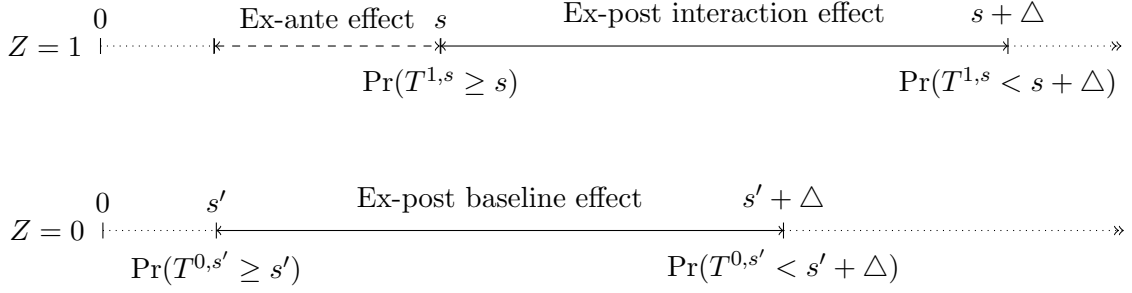\end{array}
$$

Figure 1: Causal effect evaluation intervals on untreated survivors at $S^1 = s$

Returning to our health example, say we are interested in decomposing the probability of dying within the first three years ($\triangle$) of falling sick for those who receive the drug treatment after 12 months (s), having fallen sick after the drug was rolled out ($Z = 1$). The ex-ante ATS effect tells us how the probability of death changes in the first year for these sick people compared to the probability of death in the first year for those same people had they been exposed to the randomized trial in which they had no knowledge about the drug ($Z = 0$). For the same group of untreated surviving sick people, the ex-post baseline ATS effect is the change in the probability of death over a 3 year period due to receiving the drug versus not receiving the drug had they fallen sick and been subject to the unanticipated randomized trial. Finally, the ex-post interaction ATS effect is the additional effect of receiving the drug in the world in which they were informed of the drug and its effect compared to the world in which these people were not previously informed about the existence of a drug.

The rank invariance assumption will likely not hold in all settings and its validity should be judged by the researcher a priori given any particular application. For instance, in our health science example, one must exclude that certain sub types of sick people react in a diametrically opposed way than others depending on whether or not they have prior information on the drug. While some may find such an assumption wanting, the alternatives are scarce for researchers intent on policy relevance. One may instead build a sophisticated behavioural-structural model in which the researcher calibrates key behavioural parameters to produce policy predictions. Unfortunately, these models rely themselves on strong functional form assumptions about agent utilities, expectations and underlying search processes. The other alternative is to run a randomized trial and assume the treatment effect will remain invariant when rolled out, effectively ignoring the crucial problem at hand. The framework proposed in this paper intends to provide a middle ground between these two approaches. The credibility of the rank invariance assumption in application would be strengthened by including control variables which can proxy for behavioural preferences such as risk-aversion or time-inconsistent preferences, among others. Ultimately, which setup, framework and set of assumptions are appropriate should depend on the question at hand.

2.2 Relation to Classical Mediation Analysis

It is worth comparing our approach to the classical framework of mediation analysis (Imai, Keele and Yamamoto, 2010). In mediation analysis, the most common focus is on the decomposition which in our setting can be presented as

$$
\begin{aligned}
\Delta_{mediation\ (natural)} &= \left[\Pr(T^{1,S^1} < t) - \Pr(T^{0,S^1} < t)\right] + \left[\Pr(T^{0,S^1} < t) - \Pr(T^{0,S^0} < t)\right] \\
&= \left[\Pr(T^{1,S^0} < t) - \Pr(T^{0,S^0} < t)\right] + \left[\Pr(T^{1,S^1} < t) - \Pr(T^{1,S^0} < t)\right]
\end{aligned}
$$

which is composed of the natural (average) direct effects,

$$
\Pr(T^{1,S^z} < t) - \Pr(T^{0,S^z} < t)
$$

and the natural (average) indirect effects,

$$
\Pr(T^{z,S^1} < t) - \Pr(T^{z,S^0} < t)
$$

for $z = 0, 1$. The natural direct effect captures the difference in the probability of exit when exogenously varying the policy regime but keeping the treatment distribution fixed at the potential treatment distribution under policy regime $Z = z$. The natural indirect effect corresponds to the difference in the probability of exit when exogenously varying the treatment distributions to their potential values under $Z = 1$ and $Z = 0$ but keeping the policy regime fixed at $Z = z$.

To understand the fundamental problem with this framework, take the case in which we fix $Z = 1$ for the natural direct effects, so we are comparing $\Pr(T^{1,S^1} < t)$ to $\Pr(T^{0,S^1} < t)$. $\Pr(T^{1,S^1} < t)$ is simple to interpret since it is the probability of exit in policy regime $Z = 1$ with the potential durations to treatment under policy regime $Z = 1$. However, the second term $\Pr(T^{0,S^1} < t)$ is the probability of exit under policy regime $Z = 0$ when the distribution of durations to treatment follows that which would occur under $Z = 1$. The problem is that both factually and conceptually, the distribution of treatment among agents at any given time s under $Z = 0$ cannot coincide with the distribution of treatment among agents at s under $Z = 1$. The only exception is if the policy regime assignment z has no effect on the timing of treatment, so if $F_{S^0}(s) = F_{S^1}(s)$. And if that is the case, then the natural indirect effects have no effect, which defeats the purpose of a causal decomposition. In our example, people who fall sick in the unanticipated randomized trial will never all receive the treatment as they would after the rolling out of treatment, unless the rolling out of treatment has no effect on when a sick person receives the drug. Thus, identification of mediation effects for the above decomposition requires extrapolating implications about outcomes which by definition can never be observed. The present authors among others argue that such inferences are contrary to the purpose of identification (Rubin, 2005, page 325).

A second decomposition in the classical mediation analysis literature relies on the idea that unexpected treatment times can be forced upon agents (Robins, 2003). In our setting we can write this decomposition as

$$
\begin{aligned}
\Delta_{mediation\ (controlled)} &= \left[\Pr(T^{1,s} < t) - \Pr(T^{0,s} < t)\right] + \left[\Pr(T^{0,s} < t) - \Pr(T^{0,s'} < t)\right] \\
&= \left[\Pr(T^{1,s'} < t) - \Pr(T^{0,s'} < t)\right] + \left[\Pr(T^{1,s} < t) - \Pr(T^{1,s'} < t)\right]
\end{aligned}
$$

which is composed of the controlled direct effects,

$$\Pr(T^{1,s} < t) - \Pr(T^{0,s} < t)$$

and the controlled indirect effects,

$$\Pr(T^{z,s} < t) - \Pr(T^{z,s'} < t)$$

for $z = 0,1$ and $s \neq s'$. The above decomposition for controlled effects differs from that of natural effects in that the treatment times are manipulated externally by, for instance, a researcher. For example, the controlled direct effect compares the potential outcome in policy regime $Z = 1$ with treatment at time s to the potential outcome in policy regime $Z = 0$ had the person been forced to receive treatment at time s even though this would not have been their likely or expected treatment time under policy regime $Z = 0$. The main problem with this decomposition is that due to dynamic selection, the effects will not create a strict separation between variation in the policy regime and variation in the treatment. For example, the controlled direct effects may be comparing an individual who in $Z = 1$ would exit at $T^{1,s} \geq s$, thereby having received treatment at s, while if subject to $Z = 0$ would exit at $T^{0,s} < s$, having not received treatment. A similar argument can be made for the controlled indirect effects. Since the goal of a causal decomposition is to separately describe changes in the outcome due to an initial randomization from variation due to a later randomization, the above decomposition is of limited value to a policymaker when both the treatment and outcome are duration variables. In contrast, the decomposition we propose is formulated in terms of possible counterfactuals, and appropriately saturates the causal decomposition on the policy regime and treatment effect.

2.3   Non-parametric identification of causal effects

In this section, we discuss nonparametric identification when all unobserved characteristics determining exit are captured by the initial agent type U. The evaluation framework will be presented in continuous time with $t \in \mathbb{R}^+$ and $s \in \mathbb{R}^+$ mainly because the identifying assumptions in our setting are made more credible with data observed at relatively small time intervals. We will however mention discrete time, $t \in \mathbb{N}$ and $s \in \mathbb{N}$, identification properties when relevant and produce reciprocal discrete time causal effects in appendix A. For clarity we also leave aside the issue of exogenous right censoring since it is straightforward to augment our assumptions to account for it.

For each agent i, the econometrician would like to observe the joint distribution (Z_i, S_i, T_i, X_i) where Z_i is the assignment policy, S_i the time to treatment, T_i the time to exit, and X_i is a set of baseline covariates at $t = 0$.[12] However, this joint distribution is usually not fully observed for agents who exit before treatment. For these agents we only know that $T_i < S_i$. In our formulation of assumptions we assume treatment at t has an instantaneous effect on exit, so taking a drug can influence survival immediately. We suppress hereafter all conditioning on

---

[12]We postpone the discussion of time varying covariates for section 3.

13

the $t = 0$ set of covariates X_i since it is implicit in non-parametric identification, and we also suppress subscript i.

As a first step to identification, a consistency assumption is sometimes invoked in order to link potential outcomes to observed outcomes (Robins, 1997; Murphy, 2003),

Assumption A.III: Consistency

For $z = 0, 1$, $s \in \mathbb{R}^+$,

$$S^z = S \qquad if \ Z = z$$
$$T^{z,s} = T \qquad if \ S = s, Z = z$$

This assumption states that an agent's potential treatment duration under policy regime z equals his observed treatment duration when he is actually subject to regime z. Similarly, an agent's potential outcome duration will equal his observed outcome duration if the agent is treated at $S^z = s$ as prescribed by policy regime z. The above consistency assumption is related to Rubin's Stable Unit Treatment Value Assumption (SUTVA) in the sense that the potential outcomes for an agent do not depend on the observed or counterfactual outcomes of any other agent (Rubin, 1980). SUTVA is often invoked to disallow the type of ex-ante and ex-post interaction effects we explicitly model in this paper. Here we invoke it to prevent any other type of effects, such as network effects, which would prevent extrapolating the effects measured in the sample to a wider population.

Next, identification requires that the policy regime is randomized and that treatments are randomized while accounting for dynamic selection. For this we invoke the following dynamic unconfoundedness assumptions,

Assumption A.IV: Dynamic Unconfoundedness

For $z = 0, 1$, $s \in \mathbb{R}^+$, $t \in \mathbb{R}^+$, $s \geq t$, denote by $\{T^{z,s}\}$, $\{S^z\}$ the sets of all permutations of potential variables, then,

$$(\{T^{z,s}\}, \{S^z\}) \ \perp\!\!\!\perp \ Z$$
$$\{T^{z,s}\} \ \perp\!\!\!\perp \ \mathbb{1}(S = t) \mid S \geq t, T \geq t, Z = z$$

The first assumption states that the policy regime is randomized at $t = 0$ in the sense that it is independent of potential outcomes and potential treatment times. The second assumption is a dynamic version of an unconfoundedness assumption which says that treatment is randomized on the untreated survivors. More precisely, it says that future potential outcomes are independent of treatment assignment at time t among the untreated survivors within a policy regime z. It excludes that a subset of untreated survivors can influence whether they receive treatment at t based on U. Recall that X is implicit in the conditioning set so we can allow selection on these covariates into treatment for the untreated survivors or into the policy regime. In addition, as implied by assumption $A.II$, it should be noted that assumption $A.IV$ does not exclude that agents can manipulate their exit time based on some knowledge of the treatment distribution under policy z.

In an experimental setup, assumption $A.IV$ can be made credible by externally randomizing treatment either sequentially on survivors, or randomize at $t = 0$ all future treatment times and

withholding this information from agents.[13] For example, a researcher at baseline can randomize individuals to two groups, one which is told they have a low chance to receive treatment (or are told nothing), another which is told at baseline that they are exposed to receiving a future treatment. Individuals from each group are then subsequently treated according to their prescribed group. Assumption $A.IV$ was first proposed by Robins (1997) and introduced into the economics literature by Lechner (2009). If there is censoring in the sample, one can add a similar dynamic unconfoundedness condition under which right censored observations are dynamically missing (completely) at random.

In addition, we impose that policy regimes, treatments and the decomposition can be evaluated (Robins, 1997; Lok, Gill, Van der Vaart and Robins, 2004),

Assumption A.V: Overlapping Support

Let $\theta_t^T(S > t, z) = \lim_{dt\downarrow 0} \Pr(T \in [t, t+dt]|T \geq t, S > t+dt, Z = z)$ and
$\theta_t^S(z) = \lim_{dt\downarrow 0} \Pr(S \in [t, t+dt]|S \geq t, T \geq t, Z = z)$ *then for $z = 0, 1$, $t \in \mathbb{R}^+$,*

$$(i) \quad 0 < \Pr(Z = z) < 1$$
$$(ii) \quad 0 \leq \theta_t^S(z) < 1 \quad \text{if} \quad \Pr(S \geq t, T \geq t|Z = z) > 0$$
$$(iii) \quad \theta_t^S(z) = 0 \quad \text{iff} \quad \theta_{t-dt}^T(S > t - dt, z) = 0$$

where $\theta_t^T(S > t, z)$ and $\theta_t^S(z)$ are the observed non-treated exit hazard and treatment hazard. This overlapping support assumption guarantees that we observe agents under both policy regimes $(A.V(i))$, and that there exists no time t at which the treatment is allocated to all untreated survivors $(A.V(ii))$. This assumption is more credible in practice if the treatment allocation mechanism prescribed by the policy regime is stochastic. In our health science example, falling sick in the unanticipated randomized trial world or the post-rolling out world may influence the hazard of receiving the drug but will not treat all sick people at a specific duration of their sickness. If this assumption is not credible for some combination of covariates then it is always possible to trim distributions to a population which has overlapping covariates (Smith and Todd, 2005).

$A.V(iii)$ is a new assumption which imposes that no agent is treated at time t if no agent exited immediately before t, and vice-versa. This assumption is a condition on the dynamic rank invariance assumption $A.II$. It guarantees the existence and uniqueness of a period s' in policy regime $Z = 0$ at which the distribution of U is the same as that at period s in policy regime $Z = 1$. It is a necessary condition to identify average causal decomposition effects over a treatment time window.[14]

In terms of testing, the rank invariance assumption $A.II$ cannot be tested directly since it is an assumption on unobserved variables. However, it can be tested indirectly through $A.V(iii)$ which is a necessary condition for the rank invariance assumption to hold. $A.V(iii)$

---

[13]Such a setup is for example presented in Kastoryano and van der Klaauw (2015) in an active labour market setting in which and external firm is contracted by the unemployment benefits agency to randomize unemployed individuals to enter a program based on a select number of observed baseline variables.

[14]Some causal comparisons at particular treatment quantile times $S^1 = s$ could still be identified if we only assume $\theta_{t-dt}^T(S > t - dt, z) = 0$ if $\theta_t^S(z) = 0$ but not vice versa. This would mean we could compare treatment at $S^1 = s$ to several treatment times in an interval $[s', s'']$ in $Z = 0$.

can be shown to be implausible by producing a test comparing the treatment distribution and untreated survival distribution within policy regimes. It can also be tested in conjunction with $A.II$ by comparing those distributions across policy regimes. We do not develop these tests in the current paper. The consistency and dynamic unconfoundedness assumptions do not restrict the observed data and are therefore untestable (Gill and Robins, 2001).

The reciprocal to Lemma 1 in terms of observed data follows directly from the assumptions,

Lemma 2. _Let F be a cdf. Under Axiom A.I and Assumptions $A.II - A.V$ for any given s in $Z = 1$ we define s' in $Z = 0$ such that,_

$$\Pr(T \geq s'|S \geq s', Z = 0) = \Pr(T \geq s|S \geq s, Z = 1)$$

which implies,

$$F(u|S \geq s', T \geq s', Z = 0) = F(u|S \geq s, T \geq s, Z = 1)$$

$S^0 = s'$ corresponding to $S^1 = s$ is identified by the data which implies that at $S^0 = s'$ and $S^1 = s$ the untreated surviving populations have the same distribution of unobservables. In addition, the treated at $S^0 = s'$ and $S^1 = s$ have the same distribution of unobservables due to the dynamic randomization of treatment $(A.IV)$. As a result, we can separate the change in treatment effect between policies from the change in the composition of treated agents due to dynamic selection.

Under $A.II - A.V$ and if $s + \triangle \geq s > s'$ we can identify the ex-ante and ex-post effects on the *sub-population of untreated survivors at s under policy regime $Z = 1$* from

Proposition 3. _Let $\theta_t^T(S > t, z) = \lim_{dt\downarrow 0} \Pr(T \in [t, t+dt)|T \geq t, S > t+dt, Z = z)$, $\theta_t^T(s, z) = \lim_{dt\downarrow 0} \Pr(T \in [t, t+dt)|T \geq t, S = s, Z = z)$, and $s + \triangle \geq s > s'$. Under Axiom A.I and Assumptions $A.II - A.V$ we can identify the causal decomposition effects,_

$$\Delta_{ex-ante}^{ATS}(1, s) = -\Pr(T^{0, S^0 \geq s} < s|T^{0, S^0 \geq s'} \geq s') = \exp(-\int_{s'}^{s} \theta_t^T(S > t, 0)dt) - 1$$

$$\Delta_{ex-post\ base}^{ATS}(1, s, \triangle) = \Pr(T^{0, S^0 = s'} < s' + \triangle|T^{0, S^0 \geq s'} \geq s') - \Pr(T^{0, S^0 \geq s'+\triangle} < s' + \triangle|T^{0, S^0 \geq s'} \geq s')$$

$$= \exp(-\int_{s'}^{s'+\triangle} \theta_t^T(S > t, 0)dt) - \exp(-\int_{s'}^{s'+\triangle} \theta_t^T(s', 0)dt)$$

$$\Delta_{ex-post\ int.}^{ATS}(1, s, \triangle) = \Pr(T^{1, s} < s + \triangle|T^{1, S^1 \geq s} \geq s) - \Pr(T^{0, s'} < s' + \triangle|T^{0, S^0 \geq s'} \geq s')$$

$$= \exp(-\int_{s'}^{s'+\triangle} \theta_t^T(s', 0)dt) - \exp(-\int_{s}^{s+\triangle} \theta_t^T(s, 1)dt)$$

In order to obtain ex-ante and ex-post causal effects on the full population (ITT), then the researcher must identify and multiply the above terms by the probability of being an untreated survivor,

Corollary 1. _Let_ $\theta_t^T(S > t, z) = \lim_{dt \downarrow 0} \Pr(T \in [t, t + dt] | T \geq t, S > t + dt, Z = z)$.
Under Axiom A.I and Assumptions A.II − A.V we can identify $\Delta_{ex-ante}^{ITT}(1, s)$,
$\Delta_{ex-post\ base}^{ITT}(1, s, \triangle)$ _and_ $\Delta_{ex-post\ int.}^{ITT}(1, s, \triangle)$ _by multiplying the causal decomposition effects in Proposition_ 3 _by,_

$$\Pr(T^{1, S^1 \geq s} \geq s) = \exp(-\int_0^s \theta_t^T(S > t, 1)dt) \ \text{ for } \ s > 1$$

Finally, if the researcher wants to integrate effects over a window of treatment times, she also needs to identify the probability of treatment at period s under regime z,

Corollary 2. _Let_ $\theta_t^S(z) = \lim_{dt \downarrow 0} \Pr(S \in [t, t + dt] | S \geq t, T \geq t, Z = z)$. _Under Axiom A.I and Assumptions A.II − A.V we can identify,_

$$\Pr(S^z = s) = \theta_s^S(z) \cdot \exp(-\int_0^s \theta_t^S(z)dt)$$

We present proofs to Proposition 3 and Corollaries 1-2 in appendix A along with their reciprocal discrete time formulas. We also provide proofs of identification for equation 2 and equation 3.

The above equations are adapted versions of Robins' (1997) and Gill and Robins' (2001) "g-computation formula" which also admits non-binary and non-permanent treatments as well as non-duration outcomes. As in the general formula, our version allows for causal dependence of treatment histories on outcomes but also of outcome histories on the treatment. In our example, the latter may be relevant if doctors take into account the time a person has been sick when deciding whether to offer them the drug treatment.

One objection to the proposed framework is that the rank invariance assumption in its current form is unlikely to hold exactly in practice. As defined in $A.II$, a given agent characterized by u_j will always leave before agent u_k when all other time 0 variables are held equal. This assumption ignores the possibility that agent u_j with particularly unfavourable intermediate variables (shocks) at $t > 0$ exits after agent $u_k = u_j + \epsilon$ with particularly favorable intermediate variables for arbitrarily small ϵ. One could argue that $A.II$ holds approximately when controlling for a rich set of baseline covariates such that the effect of unobserved intermediate variables on exit do not interact strongly with the policy regime. However, in many cases this argument will remain unsatisfactory. In our health science example, people may face all sorts of health shocks after contracting HIV which lead to violations of the rank invariance assumption. In the next section we therefore push identification further by considering what conditions on unobserved intermediate variables are necessary to preserve identification.

3   Unobserved intermediate shocks

Let us first define W_t as a vector of unobserved intermediate random variables at period t which are relevant to future exit decisions. We also define the history of unobserved intermediate variables up until time t by $\overline{W}_t$. The history of realizations for agents who have not yet exited is given by $\overline{W}_t = \overline{w}_t$, $\overline{w}_t \in \mathcal{W}$. In our health science example, W_t could include variables concerning whether the agent contracted a bacterial infection, underwent large amounts of stress

or experienced malnutrition, all of which may interact with the underlying HIV to influence the agent's probability of death. In most real world applications, it is unlikely each agent knows all unobserved variables in the past relevant to their probability of exit and can perfectly predict the joint distribution of variables in the future. Instead, we assume agents adapt their exit decisions based on a heuristic. Each agent forms a possibly inaccurate belief of the value of unobserved variables for the following period given by a function of his history, $\psi_{t+dt}(\overline{W}_t = \overline{w}_t, \mathbb{1}(S \leq t), ..., \mathbb{1}(S \leq dt), T > t, Z = z, U = u)$.

We further assume the agent is Bayesian in that he only updates his predictions of the future if the realization of W_t does not conform to his prior belief $\psi_t(\cdot)$. From this we can define the intermediate shock V_t for each agent as the deviation,

$$V_t = W_t - \lim_{dt \downarrow 0} \psi_t(\overline{W}_{t-dt} = \overline{w}_{t-dt}, \mathbb{1}(S \leq t - dt), ..., \mathbb{1}(S \leq dt), T > t - dt, Z = z, U = u)$$

We also denote by v_t the realization of random variable V_t and $\overline{V}_t$ as the history of unobserved shocks.

3.1 Symmetry of shock effects

When allowing for unobserved intermediate variables, we need to revisit assumptions $A.III - A.V$. Although assumptions $A.III - A.V$ remain unchanged, their interpretation should be augmented. The dynamic unconfoudnedness assumption $A.IV$ when allowing unobserved intermediate variables assumes that the policy regime Z is randomized on potential unobserved intermediate variables. It also assumes treatment at time $S = t$ is independent of the entire path of future potential variables $W_{t^*}^{z,s}$, $t^* \geq t$, and past realized variables W_{t^*}, $t^* < t$, conditional on being an untreated survivor at time t under policy regime z.[15] For example, there cannot be a water contamination unobserved by the researcher right after t in which only the treated at $S = t$ are affected. However, independence does not exclude that the distribution of potential unobserved intermediate variables $W_{t^*}^{z,s}$ is different depending upon the policy regime z and treatment time s.[16] There also cannot be any variables in W_{t^*}, $t^* < t$ which allow a subset of agents to anticipate exactly when they will receive the drug and allow an unidentifiable subset of these agents to resist dying in response to this knowledge. More precisely, anticipation cannot imply that $F(u|T^{z,S^z \geq s} \geq s, S^z = s) \neq F(u|T^{z,S^z \geq s} \geq s, S^z > s)$, so the surviving treated at s cannot be different in terms of unobservables U than the surviving non-treated at s. Last, the support assumptions in $A.V$ cannot be violated due to past unobserved intermediate variables.

---

[15]One could explicitly write the dynamic unconfoundedness assumption on unobserved intermediate variables as: For $z = 0, 1$, $z' = 0, 1$, $s \in \mathbb{R}^+$, $t \in \mathbb{R}^+$, $t\dagger \in \mathbb{R}^+$, $t* \in \mathbb{R}^+$, $s \geq t$, $t* \geq t$ denote by $\{W_t^{z,s}\}$, the set of all permutations of potential unobserved intermediate variables at t, then, $\{W_{t\dagger}^{z,s}\} \perp\!\!\!\perp Z$ and $\{\overline{W}_{t*}^{z,s}\} \perp\!\!\!\perp \mathbb{1}(S = t) \mid S \geq t, T \geq t, Z = z$.

[16]So we cannot allow an experimental setup in which, for that particular implementation of the experiment, only those exposed to $Z = 1$ experience a water contamination. However we can allow the thought experiment in which, for every possible world, someone who is exposed to policy regime $Z = 1$ will always experience a water contamination, but will not if exposed to $Z = 0$. In this thought experiment, the water contamination (unobserved) is defined as an entangled effect of the policy regime. Similar arguments apply to unobserved intermediate variables entangled with treatment at some time $S = t$.

However, even this broader understanding of assumptions $A.III - A.V$ will not be enough to credibly justify the rank invariance assumption $A.II$. The main problem when allowing for unobserved intermediate variables is that the unobserved heterogeneity components $(\overline{w}_t, u)$ for untreated survivors at any time $t > 0$ can interact arbitrarily with the policy regime z. For instance, for two sick people u_j and $u_k = u_j + \epsilon$, it could be that u_j exits before u_k if they both experience the same bacterial infection in the unanticipated randomized trial world, $Z = 0$, but u_k exits before u_j if they are in the world in which they know they are exposed to receiving the new drug, $Z = 1$. It may also be that if they both experience the same type of malnutrition, u_k exits before u_j even if both sick people are exposed to the randomized trial world, $Z = 0$. In both these cases, the dynamic rank invariance assumption will be violated for some realizations of $\overline{w}_t$. As a result, without further assumptions on unobserved intermediate variables, we cannot justify the existence of two corresponding points $S^1 = s$ and $S^0 = s'$ at which the distribution of u for survivors is the same. This in turn implies that we cannot identify the causal decomposition effects in Propositions 3.

To preserve non-parametric identification, we propose the following condition under which we can integrate out the effect of unobserved intermediate variables.

Assumption A.VI: Symmetric shock effects

For all $u \in \mathcal{U}$, $\overline{w}_t \in \mathcal{W}$, $t \in \mathbb{R}^+$, $z = 0, 1$, it holds that,

$$(i) \quad \Pr(T \geq t | S > t, Z = z, U = u) = \mathbb{E}_{\overline{W}_t}[\Pr(T \geq t | S > t, Z = z, \overline{W}_t = \overline{w}_t, U = u)]$$
$$= \Pr(T \geq t | S > t, Z = z, \overline{V}_t = \overline{0}_t, U = u)$$

$$(ii) \quad \Pr(S \geq t | Z = z, U = u) = \mathbb{E}_{\overline{W}_t}[\Pr(S \geq t | Z = z, \overline{W}_t = \overline{w}_t, U = u)]$$
$$= \Pr(S \geq t | Z = z, \overline{V}_t = \overline{0}_t, U = u)$$

The first part of this assumption imposes that for each agent defined by u, the expected value of the conditional probability of surviving taken over all unobserved intermediate shock paths $\overline{W}_t = \overline{w}_t$ up until time t, is equivalent to the conditional probability of survival had the agent always received his expected unobserved intermediate variables. This is equivalent to saying that positive and negative shocks from the point of view of the agent have a symmetric effect on the probability of survival. The second part of the assumption claims the same holds for the conditional probability of survival to treatment.

With this assumption in hand, we can reinterpret the rank invariance assumption $A.II$. In the previous setting, the definition of U required that, if assumption $A.II$ could hold conditional on observed baseline covariates, then the ordering of realizations u at $t = 0$ was defined such that assumption $A.II$ must hold. In contrast, in the setting with unobserved intermediate variables, the definition of U encloses the future expected shocks path $\overline{V}_\infty = \overline{0}_\infty$ such that, if assumption $A.II$ can hold conditional on observed baseline covariates and future expected shocks path $\overline{V}_\infty = \overline{0}_\infty$, then the ordering of realizations u at $t = 0$ is defined such that assumption $A.II$ must hold. It directly follows from assumptions $A.II - A.VI$ that all causal decomposition effects are non-parametrically identified as stated in the following,

Proposition 4. *Under Axiom A.I and Assumptions $A.II - A.VI$, Lemma 2 and Proposition 3 hold when allowing for unobserved intermediate variables.*

The succinct proof to Proposition 4 is presented in appendix B. In fact, $A.VI(ii)$ is not necessary to identify causal decomposition effects at any particular time s. It is however required to identify the time to treatment density which is necessary if the researcher wants to obtain expected causal effects over a window of treatment times.

It is worth considering in this setting why the ex-post interaction effect may be non-null. In the setting without unobserved intermediate variables a non-null effect could arise if initial unobserved variable U interact with the policy regime Z to change the effect of the treatment. In the current setting, the ex-post interaction effect can also be different due to the fact that the treated in $Z = 0$ vs. $Z = 1$ have different distributions of their histories of unobserved intermediate variables simply because they are evaluated at different times s and s'. Selection on past $\overline{W}_t$ is in fact part of the effect of the policy regime and treatment. However, this selection does not break down identification of the causal effects under study since the decomposition of causal mechanisms is solely defined on $F(u|T^{1,S^1 \geq s} \geq s)$, the distribution of unobservables U for the untreated survivors at $S^1 = s$.

A last point concerns *observed* intermediate variables X_t for $t > 0$. In general, unless these observed variables are independent of the policy regime and the treatment, and have effects which are independent of the policy regime and the treatment, introducing them will cause identification to break down. Furthermore, if these independence assumptions hold, then identification will still hold when ignoring the observed intermediate variables since they follow the same assumptions as those on unobserved intermediate variables (VanderWeele, Vansteelandt and Robins, 2014; VanderWeele and Vansteelandt, 2013).

3.2   Underlying mixed proportional hazard structure

While Proposition 4 allows for non-parametric identification, the mechanics of assumption $A.VI$ remain somewhat opaque. It is therefore constructive to explore what underlying modelling structure may be permissible under this assumption. In the following we will show that $A.VI$ will hold in a mixed proportional hazard model which assumes positive and negative shocks produce symmetric effects on the hazard rate. To do so, we first define $\eta_0^T(u) = \eta_t^T(\overline{V}_t = \overline{0}_t, u)$ and $\eta_0^S(u) = \eta_t^S(\overline{V}_t = \overline{0}_t, u)$ as the unobserved heterogeneity effect on the hazards for an agent of type u who always received his expected intermediate variable, so when $\overline{W}_t = \psi_t(\cdot)$ for all t. Notice that $\eta_0^T(u)$ and $\eta_0^S(u)$ are time independent given our assumption on belief updating. With this notation, we impose the following proportional hazard restriction on the survivor function integrated over the effect of all intermediate shocks,

Assumption A.VII: Mixed proportional hazard

For all $u \in \mathcal{U}$, $\overline{w}_t \in \mathcal{W}$, $t \in \mathbb{R}^+$, $z = 0, 1$. There exist $\lambda_t^T(z) : \mathcal{U} \times \{0,1\} \to \mathbb{R}^+$, $\lambda_t^S(z) : \mathcal{U} \times \{0,1\} \to \mathbb{R}_*^+$ continuous such that $\lim_{T \to +\infty} \int_0^T \lambda_t^T(z) dt = +\infty$. Also, $\eta_0^T : \mathcal{U} \times \mathcal{W} \to \mathbb{R}^+$ and $\eta_0^S : \mathcal{U} \times \mathcal{W} \to \mathbb{R}^+$ are integrable. Then the observed non-treated exit hazard $\theta_t^T(z, S > t, \overline{v}_t, u)$ and observed treatment hazard $\theta_t^S(z, \overline{v}_t, u)$ are such that,

$$(i) \quad \Pr(T \geq t | S > t, Z = z, \overline{V}_t = \overline{0}_t, U = u) = \exp(-\int_0^t \theta_\tau^T(z, S > t, \overline{0}_\tau, u) d\tau)$$

$$= \exp(-\int_0^t \lambda_\tau^T(z) \eta_0^T(u) d\tau)$$

$$(ii) \quad \Pr(S \geq t | Z = z, \overline{V}_t = \overline{0}_t, U = u) = \exp(-\int_0^t \theta_\tau^S(z, \overline{0}_\tau, u) d\tau) = \exp(-\int_0^t \lambda_\tau^S(z) \eta_0^S(u) d\tau)$$

where $\theta_t^T(z, S > t, \overline{v}_t, u) = \lim_{dt \downarrow 0} \Pr(T \in [t, t + dt) | T \geq t, S > t + dt, Z = z, \overline{V}_t = \overline{v}_t, U = u)$ and $\theta_t^S(z, \overline{v}_t, u) = \lim_{dt \downarrow 0} \Pr(S \in [t, t + dt) | S \geq t, T \geq t, Z = z, \overline{V}_t = \overline{v}_t, U = u)$. $A.VII$ assumes that the expected exit and treatment hazards for non-treated agents at t can be separated into two multiplicative components. The first terms are policy regime specific baseline duration dependent hazards $\lambda_t^T(z)$ and $\lambda_t^S(z)$, and the second terms capture period unobserved heterogeneity $\eta_0^T(u)$ and $\eta_0^S(u)$.[17]

Under this underlying semi parametric modelling assumption for the not yet treated survivors we can identify all causal decomposition effects in Proposition 3,

Proposition 5. *Under assumptions $A.II - A.VI$, and assuming the underlying model follows the mixed proportional hazard structure of assumption $A.VII$, Lemma 2 and Proposition 3 hold when allowing for unobserved intermediate variables.*

The proof is presented in appendix B. All of the causal decomposition effects are identified without needing to identifying the specific components $\lambda_t^T(z)$, $\lambda_t^S(z)$, $\eta_0^T(u)$ and $\eta_0^S(u)$. Online appendix D relates at length our framework to dynamic discrete choice models in economics and provides more intuition on the interpretation of the separate components in the mixed proportional hazard model.

3.3 Estimation approach

In this section we propose a simple semi-parametric proportional hazard model for estimation.[18] We write the joint exit and treatment hazards in the proportional hazard specification,

$$\theta_t^T(x, z, s, u^T) = \lambda_t^T(z, \mathbb{1}(s \leq t)) \exp(x'\beta^T + u^T)$$
$$\theta_t^S(x, z, u^S) = \lambda_t^S(z) \exp(x'\beta^S + u^S)$$

We propose the following procedure to estimate ex-ante and ex-post effects.[19] We first parameterize the duration dependence functions $\lambda_t^T(z, \mathbb{1}(s \leq t))$ and $\lambda_t^S(z)$ as separate piecewise constant baseline hazards depending on Z and whether treatment occurred $\mathbb{1}(s \leq t)$. We then estimate the parameters of the joint density of the above by Maximum Likelihood.

Then, for each observation $i = 1, \ldots, n$ characterized by covariates x_i in the dataset, we generate for each $t = 1, \ldots, T^{max}$ under $Z = 1$ the non-treated survivor function $exp(-\int_0^t \hat{\theta}_\tau^T(x_i, 1, S >$

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[17]To be clear, these terms explicit on baseline covariates $X = x$ are $A.VII(i)$: $\exp(-\int_0^t \lambda_\tau^T(x, z) \eta_0^T(x, u) d\tau)$ and $A.VII(ii)$: $\exp(-\int_0^t \lambda_\tau^S(x, z) \eta_0^S(x, u) d\tau)$.

[18]See Kastoryano and van der Klaauw (2015) for non-parametric discrete time duration estimators which may be adapted to our setting and for a discussion of tradeoffs between discrete and continuous time estimation approaches.

[19]R programming code is available at www.skastoryano.com.

$t, u_i^T)d\tau$) and for each t under $Z = 0$ the non-treated survivor function $exp(-\int_0^t \hat{\theta}_\tau^T(x_i, 0, S > t, u_i^T)d\tau)$. Then, for each observation i and for each value of s under $Z = 1$ we choose as s_i' the time s_i^* under $Z = 0$ which minimizes

$$\left|\left| exp(-\int_0^s \hat{\theta}_\tau^T(x_i, 1, S > s, u_i^T)d\tau) - exp(-\int_0^{s_i^*} \hat{\theta}_\tau^T(x_i, 0, S > s_i^*, u_i^T)d\tau) \right|\right|$$

The ex-ante and ex-post effects on the treated at $S^1 = s$ can then be computed as,

Ex-ante effect estimator:

$$\hat{\Delta}_{ex-ante}^*(1, s) =$$

$$\sum_{\{i\}} \hat{w}_i^*(s) \left(exp(-\int_0^s \hat{\theta}_\tau^T(x_i, 0, S > s, u_i^T)d\tau) - exp(-\int_0^s \hat{\theta}_\tau^T(x_i, 1, S > s, u_i^T)d\tau) \right)$$

Ex-post baseline effect estimator:

$$\hat{\Delta}_{ex-post\ base}^*(1, s, \triangle) =$$

$$\sum_{\{i\}} \hat{w}_i^*(s) \left(exp(-\int_0^{s_i'+\triangle} \hat{\theta}_\tau^T(x_i, 0, S > s_i' + \triangle, u_i^T)d\tau) - exp(-\int_0^{s_i'+\triangle} \hat{\theta}_\tau^T(x_i, 0, s_i', u_i^T)d\tau) \right)$$

Ex-post interaction effect estimator:

$$\hat{\Delta}_{ex-post\ int.}^*(1, s, \triangle) =$$

$$\sum_{\{i\}} \hat{w}_i^*(s) \left(exp(-\int_0^{s_i'+\triangle} \hat{\theta}_\tau^T(x_i, 0, s_i', u_i^T)d\tau) - exp(-\int_0^{s+\triangle} \hat{\theta}_\tau^T(x_i, 1, s, u_i^T)d\tau) \right)$$

where for the ATS effect on the untreated survivors we take the weight $\hat{w}_i^*(s)$ as,

$$\hat{w}_i^{ATS}(s) = \frac{\hat{\theta}_s^S(x_i, 1, u_i^S) exp(-\int_0^s \hat{\theta}_\tau^S(x_i, 1, u_i^S)d\tau)}{\sum_{\{i\}} \hat{\theta}_s^S(x_i, 1, u_i^S) exp(-\int_0^s \hat{\theta}_\tau^S(x_i, 1, u_i^S)d\tau) exp(-\int_0^s \hat{\theta}_\tau^T(x_i, 1, S > s, u_i^T)d\tau)}$$

Notice that under usual regularity conditions the weight for our population of interest, namely the untreated survivors at $S = s$ under regime $Z = 1$, will follow

$$\sum_{\{i\}} \hat{\theta}_s^S(x_i, 1, u_i^S) exp(-\int_0^s \hat{\theta}_\tau^S(x_i, 1, u_i^S)d\tau) exp(-\int_0^s \hat{\theta}_\tau^T(x_i, 1, S > s, u_i^T)d\tau)$$

$$\overset{p}{\to} \Pr(S = s, T \geq s | Z = 1)$$

as $n \to \infty$. We can also use the weights for the population intended to be treated (ITT) at $S = s$ under regime $Z = 1$ regardless of whether the agent exited before period s given by,

$$\hat{w}_i^{ITT}(s) = \frac{\hat{\theta}_s^S(x_i, 1, u_i^S) exp(-\int_0^s \hat{\theta}_\tau^S(x_i, 1, u_i^S)d\tau)}{\sum_{\{i\}} \hat{\theta}_s^S(x_i, 1, u_i^S) exp(-\int_0^s \hat{\theta}_\tau^S(x_i, 1, u_i^S)d\tau)}$$

We then have $\sum_{\{i\}} \hat{\theta}_s^S(x_i, 1, u_i^S) exp(-\int_0^s \hat{\theta}_\tau^S(x_i, 1, u_i^S)d\tau) \overset{p}{\to} \Pr(S = s | Z = 1)$ as $n \to \infty$.

To obtain the expected value of the effects over an interval of treatment times $[1, \overline{s}]$, one should simply sum the effects with the denominators in the weights summed over $s = 1, \ldots, \overline{s}$. Finally, we use the delta method to compute standard errors around the causal effects of interest. In online appendix D we provide some simulation results assessing the robustness of our estimation method on simulated data generated from a dynamic discrete choice model. All in all our continuous-time hazard estimator seems to perform well despite its low level of complexity when the underlying model is not excessively non-linear.

4 Empirical application decomposing effects of Job Corps

In this section we illustrate our optimal policy design methodology by decomposing the effects of the Job Corps program on crime operating through employment. The Job Corps program, at a cost of \$1.7 billion yearly, comprises 60% of annual funding allocation from the US Department of Labor. Over 131 nationwide centers offer intensive on-sight vocational training and academic education, as well as a range of other health, counselling and social skills, and job placement services to youths aged 16-24 originating from disadvantaged socio-economic communities. Along with its size and intensity, a unique feature of the Job Corps program is that 87% of its participants reside at the centers.

Our application employs data from the National Job Corps Study, an experimental evaluation of the US Job Corps program conducted between 1994-2000. Despite its age Job Corps has not changed substantially since the late 1990s.[20] Schochet, Burghardt and McConnell (2001, 2008) present a thorough description of the national Job Corps Study experimental design. In brief, the National Job Corps Study randomized youths eligible for Job Corps across the US in the 14 months prior to February 1996 to either a program group $(9, 409)$ or a control group $(5, 977)$. Almost three quarters of youths assigned to the program group enrolled in centers within one to four weeks of their center assignment while control group members were not allowed to join Job Corps for the 36 months after random assignment. The duration of stay in Job Corps for program participants was about 8 months but varied considerably and the hours of instruction amounted on average to nearly $1, 200$ hours. The program and control group responded to baseline interview questions as well as 12, 30, and 48 months follow up surveys after random assignment. The survey sample which completed the 48-month interview includes $11, 313$ youths ($6, 828$ program participants and $4, 485$ controls). Of particular interest in our evaluation are the survey results on the duration until and number of hours spent by week in employment and the post-randomization timeline of criminal activity.

Schochet et. al. (2001; 2008) offer results on some of the effects of the randomization to Job Corps. Some previous studies also apply classical mediation analysis to the Job Corps study dataset. These assume selection only on observed variables to decompose earnings and employment effects mediated by work experience (Flores and Flores-Lagunes, 2009), health effects mediated by employment (Huber, 2014) or dose response effects of Job Corps on arrests mediated through employment (Hsu et. al. 2018). Flores and Flores-Lagunes (2010) go further

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[20]In fact, recent criticism by federal officials point to the long overdo need for reform of the Job Corps program given its cost of $15, 000$ to $45, 000$ per student to taxpayers (US Department of Labor, 2018).

by bounding the so called indirect effect of Job Corps on earnings and employment operating through the obtention of a GED, High School or vocational training degree, while also proposing a partial identification approach direct effects when the mediator is endogenous.[21]

4.1 Data

Our outcome duration T of interest is the post-randomization time until first arrest. As opposed to Schochet, et. al. (2001; 2008) which consider quarterly data, we construct duration variables from the raw survey results to build monthly data. The duration until arrest outcome is constructed as the first month an individual reported to have been arrested.[22]

The policy regime randomization Z is the Job Corps assignment to program or control group. There is some non-compliance in the control group, where 1.4% entered Job Corps before the 3 year restriction, so the randomization Z is in fact an intention to treat. The duration to treatment S is the post-randomization time until employment. We consider as employed the first month a youth works 25 hours or more in a given week during that month[23] and is observed to keep working for at least the following 3 months.

We drop observations for which time to employment or arrests is entirely missing. In the estimation we control for a rich set of baseline variables.[24] We further assume that censoring of duration variables is missing at random conditional on the included covariates. Finally, we implement a random forest algorithm to impute values to the relatively limited amount of missing values in the covariate matrix.

In terms of our assumptions, the Job Corps randomization serves to justify the unconfoundedness assumption on the independence of Z in $A.IV$, while we rely on selection on observed variables to justify $A.V$. This means we assume here that no other baseline variables beyond the ones we control for jointly determine the timing of employment and of being arrested. In addition, we assume that all unobserved intermediate variables occurring after the randomization to the Job Corps program or control group can be integrated out as under assumption $A.VI$. Last, for the causal effects in the decomposition to be compared in a meaningful way, we need to assume the dynamic rank invariance assumption $A.II$ holds. In this context we therefore must assume that for those who did not yet obtain a job, the sequence of being arrested would be the same had the youths been subject to the Job Corps program or control group. This excludes for instance that some youths' decision to commit a crime at a certain time is influenced by whether or not they participate in the Job Corps, while others' decision to commit a crime is unaffected by their Job Corps assignment.

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[21]Online Appendix C offers some additional literature and discussion on the relation between programs for at risk youth, employment and crime.

[22]Despite being individual survey reports, a study by Needels and Burghardt (2000) covering the 30-month follow up in North Carolina and Texas using official crime data are consistent with the Job Corps arrest survey data.

[23]We set the minimum threshold to approximately the 10% quantile of the hours per week distribution for those with observed hours above 0. This allows a more realistic representation of employment.

[24]This set includes binary indicators for *aged 16-17*, *aged 18-20*, *High School degree*, *gender*, *has children*, *ever arrested*, *race black*, *race hispanic*, *race other non-white*, *dense metro area*, *very dense metro area*, *currently employed*, *worked previous year*, *received any benefits previous year*.

4.2 Empirical results

Table 1 and Figures 2-4 present the decomposition of effects on the probability of arrest for youths who find their first job at different times after the initial Job Corps randomization. Following our simulation results, we specify the duration dependence terms to have five segments with cutoffs at $s = 5, 10, 23, 36, 48$ months. The first column of Table 1 presents results for the sub-population of youths who were randomized to the Job Corps program, did not get arrested before finding a job, and became employed 12 months after randomization. For these youths, the total policy design effect of going from no Job Corps program ($Z = 0$) and not finding employment compared to participating in the Job Corps program ($Z = 1$) and finding a job at 12 months ($s = 12$ months) is a decrease of 21.1 percentage points in the probability of being arrested within 36 months.

Table 1: Effect decomposition for Job Corps - Employment - Arrest

	$s + \triangle$ fixed at 36 months			$\triangle$ fixed at 6 months
	ATS $s = 12$	ATS $s \in [1, 30]$	ITT $s \in [1, 30]$	ATS $s \in [1, 30]$
$\hat{\triangle}_{total}(1, s, \triangle)$	-0.211**	-0.191**	-0.174**	-0.294**
	(0.024)	(0.023)	(0.021)	(0.020)
$\hat{\triangle}_{ex-ante}(1, s)$	-0.047**	-0.044**	-0.040**	-0.044**
	(0.007)	(0.006)	(0.005)	(0.006)
$\hat{\triangle}_{ex-post\ base}(1, s, \triangle)$	-0.019	-0.022*	-0.020*	-0.013
	(0.015)	(0.009)	(0.008)	(0.007)
$\hat{\triangle}_{ex-post\ int.}(1, s, \triangle)$	-0.145**	-0.125**	-0.114**	-0.238**
	(0.020)	(0.015)	(0.013)	(0.014)
N	12707	12707	12707	12707
N (Z=1)	7888	7888	7888	7888

** indicates significance at the 1% level, * significance at the 5% level.

The policy design effect can be decomposed into its subcomponents. For youths who are part of the Job Corps program ($Z = 1$), two effects occur. First, they experience a reduction of 4.7 percentage points due to the ex-ante effect, which shows that prior to finding employment youths in the Job Corps program are less likely to be arrested. This could be explained by the high intensity investment of Job Corps on formation and educational attainment which produces locking-in effects. However, we also notice that those who are part of the Job Corps program and who find a job at 12 months have a large additional negative effect of 14.5 percentage points on arrests relative to finding employment without the Job Corps program. For the youths who were not assigned to the Job Corps program, the ex-post baseline effect is -1.9 percentage points, but is not significant. This suggests that, for the relevant previously untreated surviving youths, obtaining a job does not reduce the probability of arrest relative to staying unemployed for those who were not assigned to the Job Corps program. When looking at the employment-crime relationship, the main impact on crime reduction therefor seems to be driven by finding employment having participated in the Job Corps program.

Figures 2-4 allow us to further inspect the evolution of the effects over employment times. We find the ex-ante effect on arrests decreases gradually over time, indicating persistent lock-in

Effect Decomposition of Job Corps on Arrests by Employment time ($s + \triangle$ fixed at 36 months)
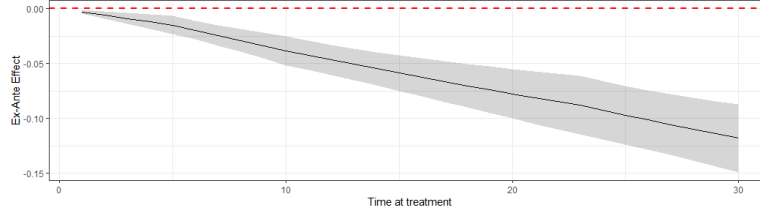

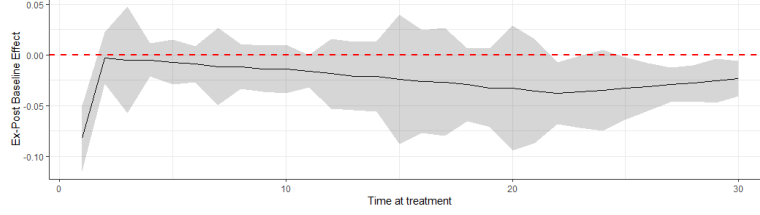Figure 2: Ex-Ante Effects

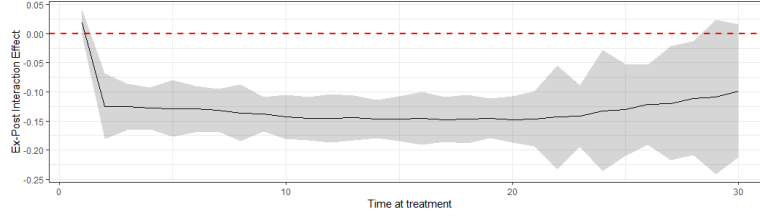Figure 3: Ex-Post Baseline Effects

Figure 4: Ex-Post Interaction Effects

effects of the Job Corps program. In terms of ex-post effects, finding a job in the first month has a strong initial negative ex-post baseline effect on arrests, but returns to zero for the surviving treated in the second month and remains close to zero until 24 months after which the effect becomes significant around -3.0 percentage points. The ex-post interaction effect indicates that obtaining a job in the first month has the same effect whether or not a youth participated in the Job Corps program. Thereafter, the probability of arrest within 36 months is significant and constantly lower for youths who participated in the Job Corps program. We also notice an upward curve towards zero after 22 months as the ex-post interaction effect evaluation period becomes smaller ($\triangle$ reduces since we hold $s + \triangle$ fixed at 36 months).

The second column of Table 1 presents averages of the effects over the window of employment times $[1, 30]$. We see that the negative total policy design effect on arrests is mainly driven by ex-post interaction effects. However, we also see that the large number of people who find a job in the first month implies that the average ex-post baseline effect is negative and significant. The third column presents the average effects on the subpopulation of agents intended to be treated (ITT). This effect includes the zero effect on youths who are arrested prior to finding a job at s having participated in the Job Corps program which is why the effects are deflated towards zero. The fourth column presents average effects over employment times $[1, 30]$ but fixing the ex-post evaluation period $\triangle$ to 6 months. From these results we can see that the negative ex-post interaction effect on arrests observed in column two is mainly due to stronger

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short run ex-post effects on arrests of finding employment having participated in the Job Corps program.

Altogether the results suggest that employment plays a large part in explaining the reduction in crime provoked by the Job Corps program. The main reason for a decrease in arrests among the Job Corps program group is not the locking in effect of the program. It seems the interaction between obtaining a job while having participated in the Job Corps program is the driving force. Although we do not delve deeply into the reasons for this difference, one possibility could be that the job matches for Job Corps participants are more stable or produce stronger commitment (Apel and Horney, 2017) than those of controls thereby leading to lower arrest rates. Delving into causal mechanisms is particularly relevant given that the US Department of Labor is considering an overhaul of the Job Corps program (US Department of Labor, 2018).

5   Conclusion

The framework and causal effects developed in this paper allow for the first policy relevant decomposition of causal mechanisms with minimal functional form assumptions about agent utilities, expectations and underlying search processes for researchers interested in optimal policy design when the treatment and outcome are duration variables. The framework can be extended to include sequences of more than two randomizations, or to include non-binary treatments and multiple policy regimes. Future research may also include developing tests for the necessary condition on our rank invariance assumption $(A.V(iii))$, allowing for non-compliance to the policy regime or treatment assignments, producing asymptotic results for our proposed mixed proportional hazard estimators, or developing fully non-parametric estimators.

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A   Identification of Causal Effects

In our derivation of identification results we maintain the overlap assumptions in $A.V$ throughout and apply $A.III$ whenever converting potential variables to observed variables. All identification results remain the same when there is censoring if we assume censoring always occurs before treatment and exit at time t and censored observations are dynamically missing at random. Identification of the ex-ante and ex-post effects requires in addition the rank invariance assumption $A.II$. We derive effects in both continuous and discrete time. In continuous time $t \in \mathbb{R}^+$ and $s \in \mathbb{R}^+$. In discrete time $t \in \mathbb{N}$ and $s \in \mathbb{N}$, we take a period $\tau = t$ such that $dt = 1$ with the adjusted discrete time reciprocals to $A.II - A.V$.

Proof of Lemma 2: The results follow directly from Axiom I, Assumption $II - A.V$ and the definition of a cdf,

$$F(u|S \geq s', T \geq s', Z = 0) = F(u|T^{0,S^0 \geq s'} \geq s')$$
$$= F(u|T^{1,S^1 \geq s} \geq s) = F(u|S \geq s, T \geq s, Z = 1)$$

Where the first and third equality are guaranteed by Assumption $A.IV$ and the second equality is due to Assumption $A.II$.

Proof of Proposition 3: Assuming $s + \triangle \geq s > s'$ we can first derive causal effects on the untreated survivors at period s,

Ex-ante effect on survivors:

$$\Delta_{ex-ante}^{ATS}(s,1) = -\Pr(T^{0,S^0 \geq s} < s | T^{0,S^0 \geq s'} \geq s')$$

$$= \Pr(T^{0,S^0 \geq s} \geq s | T^{0,S^0 \geq s'} \geq s') - 1$$

$$(A.IV) = \Pr(T^{0,S^0 \geq s} \geq s | S \geq s', T \geq s', Z = 0) - 1$$

$$(A.IV) = \exp(-\int_{s'}^{s} \theta_t^T(S > t, 0)dt) - 1$$

$$\text{(disc. time)} \ (A.IV) = \prod_{\tau=s'}^{s-1} \Pr(T > \tau | S > \tau, T \geq \tau, Z = 0) - 1$$

Ex-post baseline effect on survivors:

$$\Delta_{ex-post \ base}^{ATS}(1,s,\triangle) = \Pr(T^{0,S^0=s'} < s' + \triangle | T^{0,S^0 \geq s'} \geq s') - \Pr(T^{0,S^0 \geq s'+\triangle} < s' + \triangle | T^{0,S^0 \geq s'} \geq s')$$

$$= \Pr(T^{0,S^0 \geq s'+\triangle} \geq s' + \triangle | T^{0,S^0 \geq s'} \geq s') - \Pr(T^{0,S^0=s'} \geq s' + \triangle | T^{0,S^0 \geq s'} \geq s')$$

$$(A.IV) = \Pr(T^{0,S^0 \geq s'+\triangle} \geq s' + \triangle | S \geq s', T \geq s', Z = 0)$$
$$- \Pr(T^{0,S^0=s'} \geq s' + \triangle | S \geq s', T \geq s', Z = 0)$$

$$(A.IV) = \exp(-\int_{s'}^{s'+\triangle} \theta_t^T(S > t, 0)dt) - \exp(-\int_{s'}^{s'+\triangle} \theta_t^T(s', 0)dt)$$

$$\text{(disc. time)} \ (A.IV) = \prod_{\tau=s'}^{s'+\triangle-1} \Pr(T > \tau | S > \tau, T \geq \tau, Z = 0) - \prod_{\tau=s'}^{s'+\triangle-1} \Pr(T > \tau | S = s', T \geq \tau, Z = 0)$$

Ex-post interaction effect on survivors:

$$\Delta_{ex-post \ int.}^{ATS}(1,s,\triangle) = \Pr(T^{1,S^1=s} < s + \triangle | T^{1,S^1 \geq s} \geq s) - \Pr(T^{0,S^0=s'} < s' + \triangle | T^{0,S^0 \geq s'} \geq s')$$

$$= \Pr(T^{0,S^0=s'} \geq s' + \triangle | T^{0,S^0 \geq s'} \geq s') - \Pr(T^{1,S^1=s} \geq s + \triangle | T^{1,S^1 \geq s} \geq s)$$

$$(A.IV) = \Pr(T^{0,S^0=s'} \geq s' + \triangle | S \geq s', T \geq s', Z = 0)$$
$$- \Pr(T^{1,S^1=s} \geq s + \triangle | S \geq s, T \geq s, Z = 1)$$

$$(A.IV) = \exp(-\int_{s'}^{s'+\triangle} \theta_t^T(s', 0)dt) - \exp(-\int_{s}^{t+\triangle} \theta_t^T(s, 1)dt)$$

$$\text{(disc. time)} \ (A.IV) = \prod_{\tau=s'}^{s'+\triangle-1} \Pr(T > \tau | S = s', T \geq \tau, Z = 0) - \prod_{\tau=s}^{s+\triangle-1} \Pr(T > \tau | S = s, T \geq \tau, Z = 1)$$

Proof of Corollary 1: Multiplying the above terms by $\Pr(T^{1,S^1 \geq s} \geq s)$ we obtain the causal effects on those intended to be treated (ITT) at $S^1 = s$. Identification of this probability of survival until s for the untreated under $Z = 1$ follows straightforwardly,

$$\Pr(T^{1,S^1 \geq s} \geq s) = \exp(-\int_0^s \theta_t^T(S > t, 1)dt) \quad , \ s > 1$$

$$\text{(disc. time)} \ = \prod_{\tau=1}^{s-1} \Pr(T > \tau | S > \tau, T \geq \tau, Z = 1) \quad , \ s > 1$$

Proof of Corollary 2: If we want to integrate effects over multiple treatment periods, we also need to identify the probability of treatment at period s under regime z given by,

$$\Pr(S^z = s) = \Pr(S^z = s | S^z \geq s) \Pr(S^z \geq s)$$

$$(A.IV) = \Pr(S^z = s | S^z \geq s, Z = z) \Pr(S^z \geq s | Z = z)$$

$$(A.IV) = \theta_s^S(z) \cdot \exp(-\int_0^s \theta_t^S(z) dt)$$

$$(\text{disc. time}) \quad (A.IV) = \Pr(S = s | S \geq s, T \geq s, Z = z) \cdot \prod_{\tau=1}^{s-1} \Pr(S > \tau | S \geq \tau, T \geq \tau, Z = z)$$

$$\text{where the } s = 1 \text{ term is given by, } \Pr(S = 1 | Z = z)$$

In addition, we can identify several effects without the rank invariance assumption $A.II$ or the condition $A.V(iii)$. A first parameter we can identify is the change in the exit probability for the untreated at s due to a change in the policy regime given in equation 2,

$$\Pr(T^{1,S^1 \geq s} < s) - \Pr(T^{0,S^0 \geq s} < s) = \Pr(T^{0,S^0 \geq s} \geq s) - \Pr(T^{1,S^1 \geq s} \geq s)$$

$$(A.IV) = \Pr(T^{0,S^0 \geq s} \geq s | Z = 0) - \Pr(T^{1,S^1 \geq s} \geq s | Z = 1)$$

$$(A.IV) = \exp(-\int_0^s \theta_t^T(S > t, 0) dt) - \exp(-\int_0^s \theta_t^T(S > t, 1) dt) \ , \quad s > 1$$

$$(\text{disc. time}) \quad (A.IV) = \prod_{\tau=1}^{s-1} \Pr(T > \tau | S > \tau, T \geq \tau, Z = 0) - \prod_{\tau=1}^{s-1} \Pr(T > \tau | S > \tau, T \geq \tau, Z = 1), s > 1$$

It is also straightforward to identify the effect of treatment at s on survivors, $[\Pr(T^{z,s} \leq t) - \Pr(T^{z,S^z \geq t} \leq t)] / \Pr(T^{z,S^z \geq s} \geq s)$.

We can also identify the ex-post effect of the treatment within the same treatment regime given in equation 3. We first derive the ex-post effect on the untreated survivors at $S^z = s$ from,

$$\Pr(T^{z,S^z = s} < t | T^{z,S^z \geq s} \geq s) - \Pr(T^{z,S^z \geq t} < t | T^{z,S^z \geq s} \geq s)$$

$$= \Pr(T^{z,S^z \geq t} \geq t | T^{z,S^z \geq s} \geq s) - \Pr(T^{z,S^z = s} \geq t | T^{z,S^z \geq s} \geq s)$$

$$(A.IV) = \Pr(T^{z,S^z \geq t} \geq t | S \geq s, T \geq s, Z = z) - \Pr(T^{z,S^z = s} \geq t | S \geq s, T \geq s, Z = z)$$

$$(A.IV) = \exp(-\int_s^t \theta_t^T(S > t, z) dt) - \exp(-\int_s^t \theta_t^T(s, z) dt)$$

$$(\text{disc. time}) \quad (A.IV) = \prod_{\tau=s}^{t-1} \Pr(T > \tau | S > \tau, T \geq \tau, Z = z) - \prod_{\tau=s}^{t-1} \Pr(T > \tau | S = s, T \geq \tau, Z = z)$$

Multiplying the above term by $\Pr(T^{z,S^z \geq s} \geq s)$ we obtain the ex-post effect of the treatment for those intended to be treated at $S^z = s$ within treatment regime $Z = z$.

B   Identification with unobserved intermediate variables

Non-Parametric Identification with Unobserved Intermediate Variables:

Throughout the derivation of proofs we maintain $A.V$ and apply $A.III$ whenever converting potential variables to observed variables. In all of our notation, we keep the conditioning on observed covariates $X = x$ implicit. First we prove that there are two unique corresponding points, s in $Z = 1$ and s' in $Z = 0$, at which the distribution of unobservables for the untreated survivors are the same.

Proof of Proposition 4: We want to show that,

$$\mathbb{E}_{\overline{W}_{s'}}[F(u|T^{0,s'} \geq s', \overline{W}_{s'} = \overline{w}_{s'})] = F(u|T^{0,s'} \geq s')$$

$$\mathbb{E}_{\overline{W}_{s}}[F(u|T^{1,s} \geq s, \overline{W}_{s} = \overline{w}_{s})] = F(u|T^{1,s} \geq s)$$

such that the following holds

$$F(u|T^{0,s'} \geq s') = F(u|T^{1,s} \geq s)$$

For the above to hold it suffices to show that, under assumptions $A.IV$,

$$\frac{\Pr(T \geq s'|S > s', Z = 0, U = u)}{\Pr(T \geq s'|S > s', Z = 0)} = \frac{\Pr(T \geq s|S > s, Z = 1, U = u)}{\Pr(T \geq s|S > s, Z = 1)}$$

which follows immediately from assumptions $A.II$ and $A.VI(i)$ when allowing for unobserved intermediate variables since $A.VI$ implies,

$$\frac{\Pr(T \geq s'|S > s', Z = 0, U = u)}{\Pr(T \geq s'|S > s', Z = 0)} = \frac{\mathbb{E}_{\overline{W}_{s'}}[\Pr(T \geq s'|S > s', Z = 0, \overline{W}_{s'} = \overline{w}_{s'}, U = u)]}{\mathbb{E}_{\overline{W}_{s'}}[\Pr(T \geq s'|S > s', Z = 0, \overline{W}_{s'} = \overline{w}_{s'})]}$$

$$= \frac{\Pr(T \geq s'|S > s', Z = 0, \overline{V}_{s'} = \overline{0}_{s'}, U = u)}{\Pr(T \geq s'|S > s', Z = 0, \overline{V}_{s'} = \overline{0}_{s'})}$$

$$\frac{\Pr(T \geq s|S > s, Z = 1, U = u)}{\Pr(T \geq s|S > s, Z = 1)} = \frac{\mathbb{E}_{\overline{W}_{s}}[\Pr(T \geq s|S > s, Z = 1, \overline{W}_{s} = \overline{w}_{s}, U = u)]}{\mathbb{E}_{\overline{W}_{s}}[\Pr(T \geq s|S > s, Z = 1, \overline{W}_{s} = \overline{w}_{s})]}$$

$$= \frac{\Pr(T \geq s|S > s, Z = 1, \overline{V}_{s} = \overline{0}_{s}, U = u)}{\Pr(T \geq s|S > s, Z = 1, \overline{V}_{s} = \overline{0}_{s})}$$

Identification of the causal decomposition in Proposition 3 and all its causal effects follows straightforwardly from integrating effects over unobserved intermediate variables using $A.VI$.

Identification in Mixed Proportional Hazard Model:

Proof of Proposition 5: As in the proof of Proposition 4, it suffices to show that,

$$\frac{\Pr(T \geq s'|S > s', Z = 0, U = u)}{\Pr(T \geq s'|S > s', Z = 0)} = \frac{\Pr(T \geq s|S > s, Z = 1, U = u)}{\Pr(T \geq s|S > s, Z = 1)}$$

Using $A.VI$ and $A.VII(i)$ we can rewrite this as,

$$\frac{\exp(-\eta_0^T(u) \int_0^{s'} \lambda_\tau^T(0)d\tau)}{\mathbb{E}_{\{U\}}[\exp(-\eta_0^T(U) \int_0^{s'} \lambda_\tau^T(0)d\tau)]} = \frac{\exp(-\eta_0^T(u) \int_0^{s} \lambda_\tau^T(1)d\tau)}{\mathbb{E}_{\{U\}}[\exp(-\eta_0^T(U) \int_0^{s} \lambda_\tau^T(1)d\tau)]}$$

We see that the necessary condition for this equality to hold is that there exists a unique s' satisfying $\int_0^{s'} \lambda_\tau^T(0)d\tau = \int_0^{s} \lambda_\tau^T(1)d\tau$. This condition holds since $\int_0^{s^*} \lambda_\tau^T(z)d\tau$ is strictly increasing over $\mathbb{R}^+$ and tends to $+\infty$ as $s^* \to \infty$ which implies that the function $s^* \to \int_0^{s^*} \lambda_\tau^T(z)d\tau$ is invertible. We can show in a similar manner that $\exp(-\int_0^{s'} \lambda_\tau^S(0)\eta_0^S(u)d\tau) = \exp(-\int_0^{s} \lambda_\tau^S(1)\eta_0^S(u)d\tau)$ under $A.VII(ii)$.

It is also straightforward to see that at points s' and s it holds for all $u \in \mathcal{U}$ that

$$Pr(T^{0,S^0=s',u} \geq s') = \exp(-\eta_0^T(u) \int_0^{s'} \lambda_\tau^T(0)d\tau) = \exp(-\eta_0^T(u) \int_0^{s} \lambda_\tau^T(1)d\tau) = \Pr(T^{1,S^1=s,u} \geq s)$$

If we relax the assumption that $\int_0^{s^*} \lambda_\tau^T(z)d\tau \to +\infty$ as $s^* \to +\infty$, i.e. if for at least one of $z = 0,1$ $\int_0^{s^*} \lambda_\tau^T(z)d\tau \to A_z < +\infty$, then we assume some individuals will not exit in finite time. In this case we can still find a unique s' corresponding to s for all individuals who do exit in finite time. The set of times s for which there exists a unique s' is $s \in [0, s^*)$ such that for $z = 0,1$ and $z' = 0,1$, $\int_0^{s^*} \lambda_\tau^T(1)d\tau = A_z \leq A_{z'}$ under the additional assumption $\int_0^t \lambda_\tau^T(1)d\tau \geq \int_0^t \lambda_\tau^T(0)d\tau$ (or vice versa if $s' > s$). Identification of the causal decomposition in Proposition 3 and all its causal effects follows again straightforwardly from integrating effects over unobserved intermediate variables using $A.VI$.

ONLINE APPENDIX

C   Job Corps Study empirical application

C.1   Background and literature

Schochet et. al. (2001; 2008) offer results on some of the effects of the randomization to Job Corps that are of interest for our application. They show that Job Corps program participants see a strong initial effect of increased participation in education or vocational training compared to controls (75% vs. 30%) but the difference reduces to zero after 9 quarters.[25]   Program participants are also more likely to obtain a GED or vocational degree. In terms of employment and earnings, program participants have lower earnings and are less likely to hold a job within the first 6 quarters after the randomization but surpass the control group in both outcomes after 10 quarters.

For crime, program participants are less likely to be arrested during the first year post randomization but display no significant differences in crime outcomes thereafter. These results taken together would suggest some lock in effects of the Job Corps program on employment and arrest outcomes operating mainly through the intensity of the program and education. However, without any formal model, it is impossible to quantify the decomposition of causal effects on employment and arrests into different subcomponents.

Some previous studies apply classical mediation analysis to the Job Corps study dataset. These assume selection only on observed variables to decompose earnings and employment effects mediated by work experience (Flores and Flores-Lagunes, 2009), health effects mediated by employment (Huber, 2014) or dose response effects of Job Corps on arrests mediated through employment (Hsu et. al. 2018). Flores and Flores-Lagunes (2010) go further by bounding the so called indirect effect of Job Corps on earnings and employment operating through the obtention of a GED, High School or vocational training degree, while also proposing a partial identification approach for so called direct effects when the mediator is endogenous. While relevant, all of these mediation analyses provide decompositions on binary or continuous non-duration variables, even though several of the variables of interest are constructed from duration variables. Our methods allow for a new type of decomposition on duration variables themselves.

To illustrate our decomposition we focus on the reduction in crimes due to the Job Corps program, in particular in its relation to employment. The limited research on this issue is surprising given the attention Job Corps has received in relation to employment and crime (Thrush, 2018). The theoretical literature generally supports that employment provokes a reduced propensity to crime through several channels. The Beckerian model provides that work experience, as an important source of human capital, will decrease the returns to criminal activity (Becker, 1968, Lochner, 2004). In addition, employment can be seen as a source of social-capital with reinforcement mechanisms which are conducive to remaining a law-abiding citizen (Hirschi, 1986). In terms of the empirical literature, studies on the relationship between employment and crime have largely focused on local unemployment rates rather than individual

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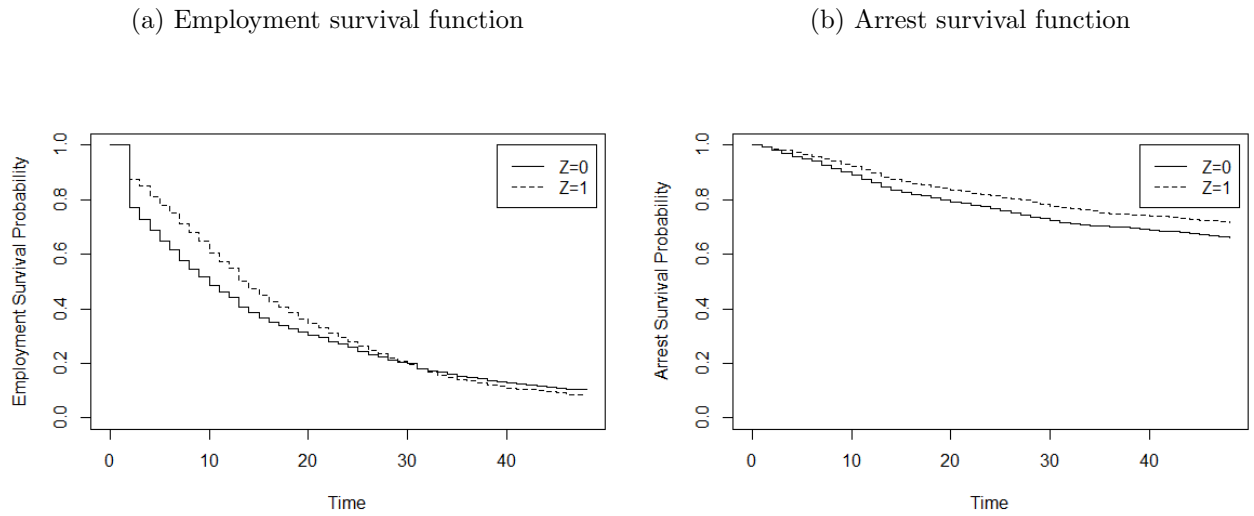[25]Hours of education by quarter produce similar trends.

employment (Fougère, Kramarz, Pouget, 2009) and also tend to focus on recidivism (Schnepel, 2018; Bhuller et. al., 2019), producing mixed results.

Fewer studies have focused on the relationship between employment and crime specifically for younger individuals which is of key importance given the well known prison-crime cycle of career criminals (Aizer and Doyle, 2015). The studies by Gelber, et. al. (2016) and Davis and Heller (forthcoming) stand out showing summer employment programs for youths lead to sustained reductions in crime, in particular violent-crime. Other studies (Anderson, 2014) underline the crime reduction due to lock-in effects of keeping young people in school. So while some studies look at effects of programs for the youth on crime and others on crime reducing effects of employment, no study has been able to quantify of how much one factor reduces crime in contrast to another. Our decomposition of causal effects therefore provides a quantifiable understanding of the relationship between programs for disadvantaged youths, employment, and crime.

C.2 Kaplan Meier estimates

Figure 5 presents Kaplan-Meier estimates of the raw duration data depending on whether a youth was randomized to the Job Corps program ($Z = 1$) or control ($Z = 0$) group. We notice for employment that many youth in both the Job Corps program and control group have a job already or find a job in the first month but more so in the control group. The reemployment probability remains similar between both groups until approximately a year at which point reemployment picks up for the Job Corps program group relative to the control group. For arrests we notice that the control group face a higher propensity to arrest in the first 15 months, but their period per period probability of arrest follows that of the Job Corps program group thereafter.

Figure 5: Kaplan-Meier estimates

(a) Employment survival function   (b) Arrest survival function

D   Evaluating estimation approach on simulated data

To relate our dynamic treatment effect model to the literature on dynamic discrete choice models we present in this appendix a standard job search model (e.g. Mortensen, 1986). We then extend it to include a treatment and intermediate shocks. We explain that if agents are not rational in the sense that they do not have perfect foresight, then interaction between the policy regime and the intermediate shocks can result in the ex-post treatment effect being different depending on the policy regime. We adapt the discussion of the search model to our crime application based on the more elaborate dynamic crime model in Chalfin and McCrary (2017). For expositional purposes, the model is solved under simplistic assumptions. We then explain how the dynamic discrete choice model relates to the parameters in our mixed proportional hazard model in Section 3.2. Thereafter we describe the data generating process and present simulation results for our estimator of Section 3.3.

D.1   Standard dynamic discrete choice model

Consider an agent who enters an initial state at time $t = 0$ and assume that time is discrete. In each subsequent period the agent faces the choice of staying within this state or leaving. Whenever he is in the initial state, the agent derives utility w_0 which can be interpreted as the utility from not being incarcerated. In our application, $t = 0$ is the moment a youth is randomized to the Job Corps program or control group and each period the youth can choose whether or not to commit a crime.[26]

Next, with probability λ the agent receives an offer to leave the state. An offer can be interpreted as a 'criminal opportunity'. The agent also faces a cost c corresponding to the necessary effort of looking for a criminal opportunity. Once the agent receives an offer, he has to decide immediately whether or not to accept it. An offer is characterized by its instantaneous utility w drawn from the distribution $G(w)$. Upon accepting an offer, the agent derives the same instantaneous utility w for each subsequent period. So once criminals commit a crime, they derive the gains but also face the possibility of being apprehended for that crime throughout the rest of their life.

We assume the agent optimization behaviour follows a dynamic discrete choice model which nests search models and optimal stopping models.[27] He forms expectations over future instantaneous utilities. Denoting by ρ the discount rate, the present discounted value of not committing a crime at the start of period t, $V_{0,t}$, can be described by the Bellman equation,

$$V_{0,t} = w_0 - c + \rho\lambda\mathbb{E}[\max\{V_1(w), V_{0,t+1}\}] + \rho(1 - \lambda)V_{0,t+1}$$

$V_1(w)$ is the discounted value that the agent would acquire by choosing to commit a crime with instantaneous utility w. The agent follows a stationary reservation utility strategy where w^* is the minimum offer of w required to induce the agent to exit in the following period. Under a

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[26]Here we define 'crime' as the act of committing a new crime although empirically this outcome is only observed for arrested criminals.

[27]For more details, see Rogerson, Shimer, and Wright (2005).

reservation utility strategy, we can reformulate the Bellman equation as

$$V_{0,t} = w_0 - c + \rho\lambda \int_{w^*}^{\infty} (V_1(w) - V_{0,t+1})\, dG(w) + \rho V_{0,t+1}$$

We can augment this model to include a treatment prescribed by a policy regime. Let us assume that the agent knows that he has been assigned to a certain policy regime z which allocates future treatment. For simplicity we consider in this section a stochastic assignment policy where the agent faces the same probability π to receive treatment at each period. So each policy regime z is fully characterized by its value of π. This type of randomization can correspond to a rule imposed by the policymaker. It could also correspond to situations where agents receive different signals of how likely they are to receive treatment, and interpret this signal as a constant hazard π to treatment each period.

In our application, teenagers and young adults from disadvantaged socio-economic backgrounds are randomized to the Job Corps program or control group. The assignment, through added job search assistance or educational formation, can directly influence the probability of finding each period a job, which in our application is the treatment. We can also consider the randomization to the Job Corps program or control group to be a stochastic treatment assignment mechanism since it influences the chances of finding a job but does not determine the exact moment a youth will find a job. In turn, working in a new job could reduce the arrival of criminal opportunities (λ), increase its search costs (c), or change the distribution of the utility from crime ($G(w)$).

To simplify the exposition, assume treatment only affects the distribution of the utility from crime and changes it from $G(w)$ to $G^{tr}(w)$ for an agent upon receiving the treatment. Allowing agents to form expectations over treatment outcomes, the no-crime value functions before finding a job, $V_{0,t}$, and after finding a job, $V_{0,t}^{tr}$, are given by,

$$V_{0,t} = w_0 - c + \rho\lambda\mathbb{E}[\max\{V_1(w), (1-\pi)V_{0,t+1} + \pi V_{0,t+1}^{tr}\}] + \rho(1-\lambda)[(1-\pi)V_{0,t+1} + \pi V_{0,t+1}^{tr}]$$
$$V_{0,t}^{tr} = w_0 - c + \rho\lambda\mathbb{E}[\max\{V_1^{tr}(w), V_{0,t+1}^{tr}\} + \rho(1-\lambda)V_{0,t+1}^{tr}$$

We can solve this model for the reservation utilities after and before treatment,[28]

$$w^*(\pi) = \frac{(1-\rho)(1-\pi)}{1-\rho+\rho\pi}(w_0 - c) + \frac{\rho\lambda(1-\pi)}{(1-\rho)(1-\rho+\rho\pi)}\int_{w^*}^{\infty}(1-G(w))\,dw + \frac{\pi}{1-\rho+\rho\pi}w^{tr*}$$
$$w^{tr*} = w_0 - c + \frac{\rho\lambda}{1-\rho}\int_{w^{tr*}}^{+\infty}\left(1 - G^{tr}(w)\right)dw$$

Under the assumption that $V_0^{tr} > V_0$ one can demonstrate that the pre-treatment reservation utility $w^*(\pi)$ is increasing in pi. This means that if agents foresee positive effects of receiving treatment and are in a policy regime with higher chances to receive treatment, then they will remain longer in the initial state if they are not yet treated.

The previous discussion suggests several policy relevant comparisons for duration outcomes.[29] The first causal effect of interest is the *ex-ante effect* on the hazard rate before

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[28]Full derivation of solutions are presented in subsection D.4

[29]In addition to duration outcomes, the policymaker could be interested in causal effects on the average offer w at exit. This is relevant if the unemployment rate and finding a job also influence the type of crime committed.

treatment of changing the policy regime parameter π to π',

$$\Pr(w \geq w^*(\pi)) - \Pr(w \geq w^*(\pi')) = \int_{w^*(\pi)}^{+\infty} dG(w) - \int_{w^*(\pi')}^{+\infty} dG(w)$$

Notice that the hazard rates $\int_{w^*(\cdot)}^{+\infty} dG(w)$ exhibit no time dependence. This is because prior to entering treatment only $G(w)$ and π (or π') influence the reservation utility, and both are constant over time. The second and most often considered causal parameter is the *ex-post baseline effect* of the treatment compared to not being treated within a given policy regime,

$$\Pr(w^{tr} \geq w^{tr*}|\pi) - \Pr(w \geq w^*(\pi)) = \int_{w^{tr*}}^{+\infty} dG^{tr}(w^{tr}) - \int_{w^*(\pi)}^{+\infty} dG(w)$$

However, this parameter is not fully informative on the ex-post treatment effect since it only describes the effect of treatment compared to a state where agents anticipate based on π. A full description of the ex-post causal effect requires knowing in addition how the treatment effect changes when shifting from π to π'. This *ex-post interaction effect* is:

$$\Pr(w^{tr} \geq w^{tr*}|\pi) - \Pr(w^{tr} \geq w^{tr*}|\pi')$$

The effect will be 0 here since the hazard rate after the individual is treated, $\int_{w^{tr*}}^{+\infty} dG^{tr}(w^{tr})$, does not depend on the policy regime. However, this special case holds only because of our restrictive modelling assumptions which imply that the effect of the policy regime ends upon receiving the treatment. Abbring and Van den Berg (2005) refer to this as the "ex-post exclusion restriction".

The ex-post exclusion restriction hinges on the strong assumption that agents are rational in the sense that they have perfect foresight so there is no unexpected information or shock at $t > 0$ which can affect their beliefs of the future and therefore their reservation utilities. This assumption is restrictive in many microeconomic applications where agents update their behaviour over time. But when we relax it, the reservation utility after treatment becomes non stationary. At period t, the reservation utility can depend arbitrarily on the interaction between the policy regime π and the history of past intermediate shocks $\overline{v}_{t-1} = (v_0, \ldots, v_{t-1})$ such that the ex-post interaction effect becomes,

$$\int_{w^{tr*}(\pi, \overline{v}_{t-1})}^{+\infty} dG_{(\pi, \overline{v}_{t-1})}(w^{tr}(\pi, \overline{v}_{t-1})) - \int_{w^{tr*}(\pi', \overline{v}_{t-1})}^{+\infty} dG_{(\pi', \overline{v}_{t-1})}(w^{tr}(\pi', \overline{v}_{t-1}))$$

The ex-post interaction effect may therefore be non-zero even if an agent would have experienced the same (potential) history of intermediate shocks in both potential policy regimes.

It is worth considering the interpretation of parameters in our model of subsection 3.2 3.2 under different treatment assignment mechanisms prescribed by the policy regime. If the policy regime enforces a constant treatment hazard each period and there is full compliance, then $\theta_t^S(z, \overline{v}_t, u) = \pi^S(z)$. If in addition agents know the policy regime, do not vary their search strategy over time, and the expected value of future variables over intermediate shocks is constant over time then $\lambda_t^T(z)$ will be constant. This constant is then interpreted as the effect on the exit hazard of a constant treatment hazard policy regime. This is the setting considered in our dynamic discrete choice search model above.

However, in many cases the treatment hazard varies over time. In our crime example, the hazard of finding a job (treatment) depends on the randomization to Job Corps (policy regime) which may have varying effects over time. In addition, the intermediate unobserved variables may be different depending on z and change in unexpected ways after treatment occurs. In these dynamic treatment assignment settings, we can interpret $\lambda_t^T(z)$ as the pre-treatment effect on the exit hazard of the average (or perceived average) treatment hazard under policy regime z. This effect itself depends on the average path of intermediate shocks specific to z. In our example, at any time prior to finding a job $\lambda_t^T(z)$ is the average effect on arrests of the Job Corps effect path, where the path may be different for youths assigned to the Job Corps program and control group. In many cases however, the interpretation of model parameters is secondary to the causal effects of interest.

D.2   Data Generating Process

The data generating process for the simulation data follows the dynamic discrete choice model presented in section above with the following specifications,

$$U_{it}^{no-exit} = w_{0it} - c_{it}$$
$$U_{it}^{exit} = w_{it}$$
$$c_{it} = \beta_a^c \cdot a_i + \beta_e^c \cdot e_i$$
$$w_{it} = \beta_a^w \cdot a_i + \beta_s^w \cdot I(s < t) + \xi_{it} \quad \xi_{it} \sim \mathcal{N}(0, \sigma_\xi^2)$$
$$w_{0it} = 0.75 \cdot \beta_a^w \cdot a_i$$

where $U_{it}^{no-exit}$ and U_{it}^{exit} are the instantaneous utilities when the agent chooses to remain in the initial state or exit. λ_{it} follows a Poisson distribution with mean $\beta_a^\lambda \cdot a_i + \beta_e^\lambda \cdot e_i$. The treatment outcome for the group under policy $Z = 1$ is drawn each period from a binary distribution with probability $\pi_{Z=1} = Pr(S = s | S \geq s, Z = 1) = 0.03$. The policy regime assignment for the $Z = 0$ group is $\pi_{Z=0} = Pr(S = s | S \geq s, Z = 0) = 0$. We impose that the treatment takes place before the exit decision in period t.

Using this model we generate the treatment durations, exit durations, and accepted offers for a population of 5000 agents over 5000 periods. Within this population, a_i and e_i assume discrete values in the intervals $[1, 6]$ and $[1, 3]$ respectively. The initial randomization assigns half of the population to each policy regime $Z = 0$ or $Z = 1$. As in the discussion above, we choose to focus on a situation where the treatment affects only the offer distribution $G(w; a)$. The treatment effect is negative and is calibrated to equal one standard deviation of the offer distribution, $-\sigma_w$, with $\beta_s^w = 0$. The full choice of parameters for each policy setting is presented below. To estimate the stationary solution for the accepted offer we simulate expectations of w_{it} over 1000 draws and iterate over the value function until convergence.

D.3   Simulation Results and Discussion

To apply the continuous time methods under study in this paper, it is preferable to have a large dataset where the unit of time represents a relatively short period. In practice, if the unit of time

Table 2: Parameter choices in simulations

μ_w	13.762,	μ_c	0.893,	μ_λ	0.092
σ_w	5.497,	σ_c	0.257,	σ_λ	0.031
β_a^w	4,	β_a^c	0.2,	β_a^λ	0.5/21
β_s^w	-5.497,	β_e^c	0.1,	β_e^λ	0.1/21
ρ	0.995,	σ_ξ	3,	$\pi_{Z=1}$	0.03

is too large it may be challenging to account for dynamic selection and for the simultaneity of treatment and exit outcomes within a period. In the estimation, we treat effort e_i as an unobserved characteristic for the researcher. The researcher observes individual treatment and exit duration outcomes, ability, w_{0it}, an indicator Z for the policy regime assignment, and an indicator if the observation is right censored. After generating the dynamic discrete choice data we censor all observations greater than $t = 250$ ($\sim 7.5\%$) and apply random right censoring to $\sim 7\%$ of the remaining observations. In addition we also impute fictitious durations to treatment for agents in policy regime $Z = 0$. The DGP effect of the ex-post baseline effect is therefore guaranteed to be 0.

Figures 6-8 and Table 3 show the results when applying our estimation method. In Figure 6-8 we present the estimated ex-ante, ex-post baseline, and ex-post interaction effects of the probability of exit within $s + \triangle$ fixed at 90 (so $\triangle$ decreases as s increases) for the surviving treated at each of $s = 1,\ldots,30$ using a sample of 2500 observations from the full population of 5000. The ex-ante effect is the effect of being exposed to the risk of treatment on the probability of exiting prior to s. The ex-post baseline effect is the effect of receiving treatment when $Z = 0$ for agents who would have survived and received treatment at s under policy regime $Z = 1$. The ex-post interaction effect is the effect of actual treatment at s in policy regime $Z = 1$ on the probability of exit between s and $s + \triangle$. The estimator seems to perform quite well relative to the DGP values (blue more sinuous line). We observe quite narrow confidence bands (in grey) which is due to the proportionality assumption which permits the use of all observations in the maximum likelihood estimation procedure, as opposed to conditioning on untreated survivors at s.

Table 3 provides further statistics on the performance of our estimator when estimating average effects over the treatment times $s = 1,\ldots,30$. The average total policy design effect in the table sums the effect for the population of surviving treated at s under $Z = 1$. The three top panels present the estimated effects, their bias, variance and mean squared error when specifying the duration dependence to four constant terms, one for each of $Z = 0, 1$, pre and post treatment. The estimator performs relatively well with 2500 and 1000 observations despite the simplicity of the model, but the bias and variance increase starkly even with 500 observations. When we increase the complexity of the duration dependence terms by setting three cutoffs at $s = 20, 60, 250$ we find a strong reduction in the bias but a very high variance.

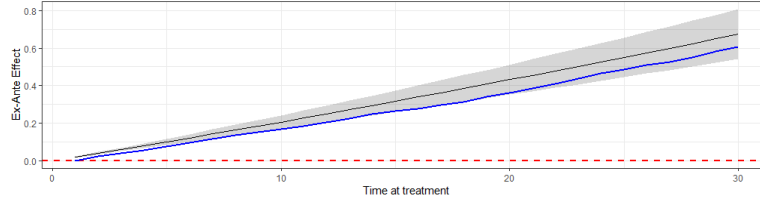Effect Decomposition of dynamic discrete choice model

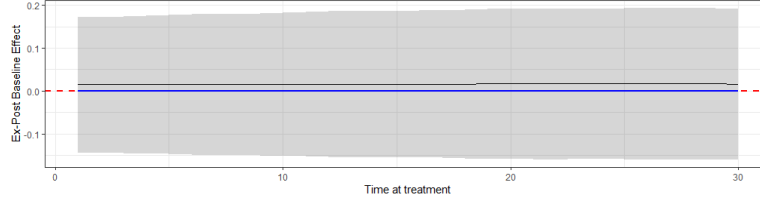Figure 6: Ex-Ante Effects on Survivors (ATS)

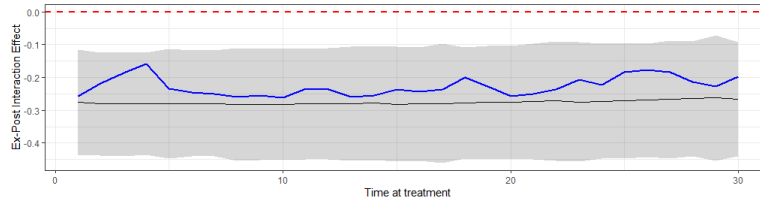Figure 7: Ex-Post Baseline Effects on Survivors (ATS)

Figure 8: Ex-Post Interaction Effects on Survivors (ATS)

Table 3: Simulation results of dynamic discrete choice model, s from 1,…,30 and $s + \triangle$ fixed at 90

	ATS	Bias	Variance	MSE
N=2500				
$\hat{\Delta}^{ATS}_{ex-ante}(1,s)$	0.231	0.013	0.000	0.001
$\hat{\Delta}^{ATS}_{ex-post\ base}(1,s,\triangle)$	0.016	0.016	0.007	0.007
$\hat{\Delta}^{ATST}_{ex-post\ int.}(1,s,\triangle)$	-0.278	-0.045	0.007	0.009
N=1000				
$\hat{\Delta}^{ATS}_{ex-ante}(1,s)$	0.231	0.013	0.001	0.001
$\hat{\Delta}^{ATS}_{ex-post\ base}(1,s,\triangle)$	0.071	0.071	0.026	0.031
$\hat{\Delta}^{ATS}_{ex-post\ int.}(1,s,\triangle)$	-0.338	-0.104	0.026	0.037
N=500				
$\hat{\Delta}^{ATS}_{ex-ante}(1,s)$	0.234	0.015	0.002	0.003
$\hat{\Delta}^{ATS}_{ex-post\ base}(1,s,\triangle)$	0.292	0.292	3.310	3.395
$\hat{\Delta}^{ATS}_{ex-post\ int.}(1,s,\triangle)$	-0.514	-0.281	1.384	1.463
N=2500, higher order duration dependence				
$\hat{\Delta}^{ATS}_{ex-ante}(1,s)$	0.223	0.005	0.001	0.001
$\hat{\Delta}^{ATS}_{ex-post\ base}(1,s,\triangle)$	-0.008	-0.008	0.802	0.803
$\hat{\Delta}^{ATS}_{ex-post\ int.}(1,s,\triangle)$	-0.237	-0.003	0.779	0.779

D.4 Reservation utilities in dynamic discrete choice model

Consider the post-treatment Bellman equations from the setting described in section D.

$$V_{0,t} = w_0 - c + \rho\lambda\mathbb{E}_w[\max\{V_1(w), (1-\pi)V_{0,t+1} + \pi V_{0,t+1}^{tr}\}] + \rho(1-\lambda)[(1-\pi)V_{0,t+1} + \pi V_{0,t+1}^{tr}]$$
$$V_{0,t}^{tr} = w_0 - c + \rho\lambda\mathbb{E}_{w^{tr}}[\max\{V_1^{tr}(w), V_{0,t+1}^{tr}\} + \rho(1-\lambda)V_{0,t+1}^{tr}$$

This second equation can be written as,

$$V_{0,t}^{tr} = w_0 - c + \rho\lambda\int_{w^{tr*}}^{\infty}\left(V_1(w^{tr}) - V_{0,t+1}^{tr}\right)dG^{tr}(w) + \rho V_{0,t+1}^{tr}$$

Since all parameters and distributions in the model are time independent, we have a stationary reservation utility strategy. In a stationary strategy $V_{0,t}^{tr} = V_{0,t+1}^{tr} = V_0^{tr}$ for all $t > 0$ Furthermore, the reservation utility w^{tr*} is such that the agent would refuse any offer below it and accept any offer above it so $V_1(w) = \frac{w}{1-\rho}$ if $w \geq w^{tr*}$, V_0^{tr} if $w^{tr} < w^{tr*}$, and $V_1(w^{tr*}) = \frac{w^{tr*}}{1-\rho} = V_0^{tr}$ if $w = w^{tr*}$. It follows that,

$$V_0^{tr} = w_0 - c + \rho\lambda\int_{w^{tr*}}^{\infty}\left(V_1(w^{tr}) - V_0^{tr}\right)dG^{tr}(w) + \rho V_0^{tr}$$

$$= w_0 - c + \frac{\rho\lambda}{1-\rho}\int_{w^{tr*}}^{\infty}\left(w - w^{tr*}\right)dG^{tr}(w) + \frac{\rho w^{tr*}}{1-\rho}$$

Replacing again $V_0^{tr} = \frac{w^{tr*}}{1-\rho}$, rearranging this equation and using integration by parts we obtain the post-treatment reservation utility,

$$w^{tr*} = w_0 - c + \frac{\rho\lambda}{1-\rho}\int_{w^{tr*}}^{\infty}\left(1 - G^{tr}(w)\right)dw$$

Note that this reservation utility does not depend on the policy regime π.

Now consider the reservation utility before treatment with a treatment assignment policy π. Since the problem is still stationary, the agent will again accept any value of w higher than his reservation w^*. We can therefore rewrite
$\mathbb{E}_w[\max\{V_1(w) - (1-\pi)V_{0,t+1} - \pi V_{0,t+1}^{tr}, 0\}] = \int_{w^*}^{\infty} w - w^* dG(w) + (1-\pi)V_{0,t+1} - \pi V_{0,t+1}^{tr}$ which results in the same V_0 value function,

$$V_0 = w_0 - c + \frac{\rho\lambda}{1-\rho}\int_{w^*}^{\infty} w - w^* dG(w) + \rho[(1-\pi)V_0 + \pi V_0^{tr}]$$

Since the agent will accept any value of w higher than his reservation w^* we know that $V_1(w^*) = V_0 = \frac{w^*}{1-\rho}$ which we replace in the above equation and rearrange to get,

$$w^* = \frac{1-\rho}{1-\rho+\rho\pi}(w_0 - c) + \frac{\rho\lambda}{1-\rho+\rho\pi}\left(\int_{w^*(\pi)}^{+\infty} w - w^* dG(w)\right) + \frac{\rho\pi}{1-\rho+\rho\pi}w^{tr*}$$

We can further show prove that w^* is increasing in π if $V_0^{tr} > V_0$:
First we rewrite the previous equation to isolate $\int_{w^*}^{+\infty} w - w^* dG(w)$,

$$\int_{w^*}^{+\infty} w - w^* dG(w) = \frac{1-\rho}{\rho\lambda}[c - w_0 + \frac{1-\rho(1-\pi)}{1-\rho}w^* - \frac{\rho\lambda\pi + \rho(1-\lambda)\pi}{1-\rho}w^{tr*}]$$

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Let us hold w^* constant in the previous equation. w^{tr*} is also constant because it does not depend on π. If we increase π, the derivative of the right-hand side with respect to π is

$$\frac{1-\rho}{\rho\lambda}\left[\frac{\rho}{1-\rho}w^* - \frac{\rho}{1-\rho}w^{tr*}\right] \qquad \leftrightarrow \qquad \frac{1-\rho}{\lambda}[(V_0 - V_0^{tr})]$$

The last equation is negative when $V_0 - V_0^{tr} < 0$, so when the value of treatment is higher than that of no-treatment. Therefore, $\int_{w^*}^{+\infty} w - w^* dG(w)$ is decreasing in π. Furthermore, written as a function of π, with $w^* = w^*(\pi)$, it is also decreasing in w^* which implies that w^* increases in π. The agent is more willing to wait for treatment.

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