Treatment and Control Groups in a Dynamic Setting

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1 Introduction

Thinking about effects of some specified factor often proceeds in terms of comparing a treatment and a control group. The treatment group consists of units exposed to the factor, the control group consists of units not exposed to the factor. The comparison concerns the distributions of an outcome variable in the two groups. A quantity derived from the two distributions, often the difference between their mean values, is then taken as a measure of the effect.

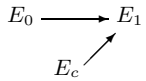
While the basic idea is straightforward, difficulties can arise in a dynamic setting. These difficulties concern the definition of a control group. Assume that the treatment is defined by experiencing a specific kind of event. So one can define a treatment group as a set of units who experienced the treatment in a specified temporal location, say t_c. But how to define a control group? Some authors have proposed that a control group should consist of all units who did not experience the treatment until, and including, $t_c + \delta$. But how to choose δ? Sianesi (2004) has proposed to choose $\delta = 0$ if one is interested in the contrast between experiencing the treatment 'now' and 'waiting' (see also Fitzenberger et al. 2013, Fredriksson and Johansson 2008). In contrast, Kohler et al. (2012) have used a relatively large value of δ, and Brand and Xie (2007) proposed a 'composite of counterfactuals' based on a reference to several values of δ.

In this paper, I start from hazard functions which allow defining effects in a temporally local way and therefore avoid fixing δ at some particular value. This approach conforms to thinking of 'causation' as a relationship between an event, or a temporally locatable state, and probabilities of subsequent 'outcomes' whose specification depends on the interest of a researcher.

In Section 2 I define effect shapes which compare hazard functions for treated and not treated units in a temporally local way, and I contrast this approach with Sianesi's proposal. In Section 3 I discuss how effects defined by a comparison of treated and not treated units can be given a causal interpretation. I propose a notion of 'comprehensive treatment effects' which does not require an actual or fictitious random assignment of treatments and can therefore be used, in particular, to understand self-selected treatments. In Section 4 I consider 'temporally extended effects' which concern event probabilities in an extended time interval after the treatment. Section 5 provides a discussion.

2 Temporally local effects

I consider a situation which basically has the following structure:



There is a specified event, E_0, whose occurrence creates the situation σ_0 in which E_1 events, which may be of different type, can occur. While this situation endures (= until the occurrence of an E_1 event), an event E_c, subsequently called 'the treatment', might occur, and we are interested in the effects of this event on the probabilities of E_1 events.

In order to refer to events I presuppose a discrete time axis, $\mathcal{T} := \{0, 1, 2, \ldots\}$, elements of this set will be called 'temporal locations' (e.g. days or months). E_1 events will be represented by a duration variable (T_1, E_1) where $E_1 \in \{1, \ldots, m\}$ specifies the type of the event and $T_1 \in \mathcal{T}$ records the temporal location in which the event occurs. For E_c it suffices to use a duration variable, T_c, recording the temporal location in which the treatment occurs ($T_c = \infty$ if a treatment never occurs).

I assume that the interest concerns effects of the treatment on the

occurrence of an event $E_1 = j$. One can begin with a hazard function

$$r_j^1(t_c, d) := \Pr(T_1 = t_c + d, E_1 = j \,|\, T_1 \geq t_c + d, T_c = t_c) \quad (1)$$

This is the probability of $E_1 = j$ occurring at $t_c + d$, conditional on having experienced the treatment at t_c and still being in the situation σ_0 at $t_c + d$. There is then the question of how to define a sensible comparison. I propose to begin with a comparison that depends both on t_c and d, and require that the treatment did not occur until $t_c + d$.[1] The hazard function to be used for the comparison can be written as

$$r_j^0(t_c, d) := \Pr(T_1 = t_c + d, E_1 = j \,|\, T_1 \geq t_c + d, T_c > t_c + d) \quad (2)$$

The condition entails that the treatment did not occur until, and including, $t_c + d$, but does not exclude that this event might occur later. (In order to ease a comparison with $r_j^1(t_c, d)$, I use the notation $r_j^0(t_c, d)$ although this hazard function depends only on $t_c + d$.)

An effect of the treatment which depends both on the treatment time, t_c, and on the time since the occurrence of the treatment, d, can be defined by

$$\Delta_j(t_c, d) := r_j^1(t_c, d) - r_j^0(t_c, d) \quad (3)$$

These are temporally local effects relating to temporal locations $t_c + d$ (for $d = 0, 1, 2, \ldots$). A sequence of such effects, that is, $\Delta_j(t_c, d)$ considered as a function of d, will be called an *effect shape* (of the treatment w.r.t. the development of the hazard of $E_1 = j$).

Treatment and control groups

The proposal requires that also treatment and control groups must be defined in a time-dependent way. With respect to (3), one can define a

[1] This is similar to what has been called a 'timing of events approach' in labor market research, see, e.g., Abbring and van den Berg (2003), Fredriksson and Johansson (2008), Lalive et al. (2008), Crépon et al. (2008). My discussion departs from this approach by not starting from a presupposition of counterfactual entities.

Table 1 Fictitious data for numerical illustration.

unit	T_c	T_1	unit	T_c	T_1
1	∞	2	11	2	2
2	∞	3	12	2	3
3	∞	4	13	2	4
4	∞	5	14	2	5
5	∞	5	15	3	5
6	∞	6	16	3	7
7	∞	6	17	3	8
8	∞	7	18	4	7
9	∞	8	19	4	8
10	∞	9	20	5	9

treatment group $\mathcal{R}^1(t_c, d)$:= a set of units who experienced the treatment at t_c and are still in the situation σ_0 at $t_c + d$, and a control group $\mathcal{R}^0(t_c, d)$:= a set of units who are still in σ_0 at $t_c + d$ and did not experience the treatment until, and including, $t_c + d$. With corresponding event sets $\mathcal{E}_j^s(t_c, d)$:= all members of $\mathcal{R}^s(t_c, d)$ who experienced the event $E_1 = j$ at $t_c + d$ ($s = 0, 1$), one can use $\#\mathcal{E}_j^s(t_c, d) / \#\mathcal{R}^s(t_c, d)$ to estimate (1) and (2), respectively. (# is used for the number of elements in a set.)

To illustrate, I use the fictitious data in Table 1. There are 20 units. Units 1 – 10 do not experience the treatment, units 11 – 20 experience this event in temporal locations given in column T_c. There are no competing risks and no censored observations, all units eventually experience the outcome event $E_1 = 1$ as indicated in column T_1. For $T_c = 2$, one can immediately derive:

d	$\mathcal{R}^1(2, d)$	$\mathcal{E}_1^1(2, d)$	$r_1^1(2, d)$	$\mathcal{R}^0(2, d)$	$\mathcal{E}_1^0(2, d)$	$r_1^0(2, d)$
0	$11 - 14$	11	1/4	$1 - 10, 15 - 20$	1	1/16
1	$12 - 14$	12	1/3	$2 - 10, 18 - 20$	2	1/12
2	$13, 14$	13	1/2	$3 - 10, 20$	3	1/9
3	14	14	1	$4 - 10$	$4, 5$	2/7
4	$\emptyset$	$\emptyset$	?	$6 - 10$	$6, 7$	2/5

If $d \geq 4$, the risk set $\mathcal{R}^1(2, d)$ is empty and the effect $\Delta_1(2, d)$ cannot be estimated.

The hazard function for comparison

The hazard function $r_j^0(t_c, d)$, which is used for the comparison, relates to a control group, $\mathcal{R}^0(t_c, d)$, consisting of all units which did not experience the treatment until, and including, t_c+d. In other words, units in $\mathcal{R}^0(t_c, 0)$ are excluded as soon as they experience the treatment. Using such a dynamic control group can be justified with two postulates:

Postulate 1: The control group for the temporal location t_c+d should not contain units who received a treatment before $t_c + d$.

Postulate 2: There should be no conditioning on the future. This requires that the control group for the temporal location $t_c + d$ must not exclude units receiving a treatment later than $t_c + d$.

The postulates entail that, for each temporal location t_c+d, the definition of $r_j^0(t_c, d)$ is completely based on information about the units which do not have experienced the treatment until, and including, $t_c + d$. No assumptions are required about the behavior of units in $\mathcal{R}^0(t_c, 0)$ and their possible treatments in temporal locations later than $t_c + d$.

Postulate 2 simply means that a researcher (observer), in order to define and estimate treatment effects at t_c+d, does not use any knowledge (if available) about treatments which occurred later than t_c+d. One therefore does not need any assumptions about the joint distribution of T_1 and T_c for $T_1 > t_c + d$ (not even assume its existence). The postulate does not entail that units of the process under consideration cannot anticipate treatments or, if they can, this will not influence their current behavior. Some authors have suggested that this assumption is required in order to identify counterfactual outcomes (e.g., Abbring and van den Berg, 2003). However, in many applications, in particular when treatments are completely or partially self-selected by human individuals, a theoretical framework entailing this assumption would contradict the process under investigation. I therefore propose that this assumption should not be considered as an essential part of the theoretical framework.

A contrast between 'now' and 'waiting'?

Sianesi motivated her approach with being interested in a contrast between receiving a treatment 'now' versus 'waiting' (Sianesi 2004, 2008; see also Fitzenberger et al. 2013). A hazard function representing 'waiting' might be defined by

$$r_j^*(t_c, d) := \Pr(T_1 = t_c + d, E_1 = j \,|\, T_1 \geq t_c + d, T_c > t_c) \tag{4}$$

Here it is only required that there is no treatment until, and including, t_c (not until $t_c + d$ as required in the definition of $r_j^0(t_c, d)$). The contrast of interest is then defined as $\Delta_j^*(t_c, d) := r_j^1(t_c, d) - r_j^*(t_c, d)$. This quantity depends on the distribution of treatments between t_c and $t_c + d$. Therefore, while possibly interesting, it seems not possible to interpret $\Delta_j^*(t_c, d)$ as representing a causal effect of the treatment. This is understandable because 'waiting' simply leaves it open what will happen subsequently. It only has a definite meaning for 'now' ($d = 0$), and then $\Delta_j^*(t_c, 0) = \Delta_j(t_c, 0)$.

In order to avoid the indeterminacy of 'waiting' one could compare treatments experienced in different temporal locations. In terms of hazard functions, one would compare $r_j^1(t_c, d)$ and $r_j^1(t'_c, d)$. However, thinking of causation as a relationship between an event, or a temporally locatable state, and probabilities of subsequent outcomes, the difference of the two hazard functions cannot be interpreted as a causal effect.

3 Causal interpretations

My discussion concerns treatments, conceptualized as events, which influence a process that might lead to an event, $E_1 = j$, at some future date. To think of a causal effect of a treatment therefore requires a conceptualization of the process leading to outcomes. I consider outcome events whose occurrence, at least to some extent, depends on the behavior of the units under consideration. I therefore presuppose that these are behavioral units (most often human individuals), subsequently be called 'primary agents'.

As examples of outcome events one can think of 'finding a job', 'becoming married', 'getting a cold', 'visiting a dentist', 'becoming involved in a traffic accident'. Obviously, there are different scopes of influencing the occurrence of an outcome event. In any case, the fact that a primary agent can influence the outcome must be taken into account when interpreting the causal effect of a treatment. In many applications, in particular in social research, at least a part of the causal effect of a treatment must be viewed as being mediated through an agent's behavior.

I start from the definition of treatment effects proposed in Section 2 which is based on a comparison of a treatment and a control group. A causal understanding of these effects must take into account how treatments, and thereby a treatment and a control group, come into being. A basic distinction can be made between self-selected and heteronomous treatments. A self-selected treatment comes into being by the primary agent to which the treatment applies. In contrast, I distinguish between three kinds of heteronomous treatments: (a) The treatment is generated by an experimenter who is able to treat a primary agent as an experimental object; (b) the process leading to a treatment originates from an institution (defined in a broad way, including, e.g., medical offices and labor market agencies); (c) the treatment is not generated by a human agent or institution. In most cases, depending on the kind of treatment and institutional regulations, the primary agent can build expectations about a future treatment and often has some scope for influencing the occurrence of the treatment.

I refer to treatments which might occur in the temporal location t_c (as in the previous section, all considerations are conditional on starting from a fixed t_c). The process generating such treatments concerns the members of a set, $\mathcal{R}(t_c, 0)$, consisting of all units who, at t_c, are still in the situation σ_0 and did not experience a treatment before t_c. I also assume a vector of covariates, say $X(t_c)$, describing the units in $\mathcal{R}(t_c, 0)$. Components of $X(t_c)$ can be time-constant characteristics of the units, or variables recording events which occurred before t_c.

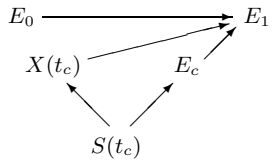
Effects concern the occurrence of E_1 events in temporal locations $t_c + d$.

So one can distinguish between instantaneous effects ($d = 0$) and temporally remote effects ($d > 0$) which also depend on processes occurring after the treatment. I begin with considering instantaneous effects.

Intentionally generated heteronomous treatments

I first consider treatments generated by an experimenter or institution (treatments not generated by a human agent or institution will not be discussed in the present paper). The situation can be depicted by the following diagram.

Model M1



The process generating treatments starts from the variable $S(t_c)$, with domain $\{0, 1\}$, representing the action of the experimenter or institution in the temporal location t_c. As part of the diagram, $S(t_c)$ is a 'decision node' as described by Dawid (2002). The action is in two steps. In a first step, the experimenter or institution selects a unit for treatment (if $S(t_c) = 1$) or control (if $S(t_c) = 0$). In the model, this is represented by the selection of a value of the variable $X(t_c)$. Then, in a second step, the treatment is applied to units selected for treatment. To simplify the discussion, I assume a deterministic relationship between $S(t_c) = 1$ and the treatment without a temporal delay. This entails that a unit cannot avoid treatment if selected for treatment, but does not exclude that the treatment could be anticipated.[2]

Note that there is no arrow from $X(t_c)$ to E_c. It is a human agent (experimenter or institution) who generates the treatment, not the variable

[2] A discussion of models for situations in which primary agents can influence the occurrence of heteronomous treatments (to be distinguished from 'avoiding', or 'not self-selecting', a treatment) is outside the scope of the present paper.

$X(t_c)$. By selecting units for the treatment, this agent generates a treatment group, $\mathcal{R}^1(t_c, 0)$, and a control group, $\mathcal{R}^0(t_c, 0) = \mathcal{R}(t_c, 0) \setminus \mathcal{R}^1(t_c, 0)$. Furthermore, the agent generates the distributions

$$\Pr(X(t_c){=}x \,|\, S(t_c) = s, T_1 \geq t_c) \tag{5}$$

which relate, respectively, to the treatment group (if $s = 1$) and to the control group (if $s = 0$). As a special case one can consider randomized treatment assignments which are defined by the independence of $X(t_c)$ and $S(t_c)$.

Separating treatment and selection effects

Given model M1, one should start from the effect of the selection variable $S(t_c)$, defined by $\tilde{\Delta}_j(t_c, d) := \tilde{r}^1_j(t_c, d) - \tilde{r}^0_j(t_c, d)$ where the hazard functions on the right-hand side are defined by

$$\tilde{r}^s_j(t_c, d) := \Pr(T_1{=}t_c + d, E_1{=}j \,|\, S(t_c){=}s, T_1 \geq t_c + d)$$

(for $s = 0, 1$). This will be called a 'comprehensive treatment effect' (CTE).

Given a deterministic relationship between the selection variable $S(t_c)$ and the treatment, as was assumed above, one can likewise use the earlier notation $\Delta_j(t_c, d) = r^1_j(t_c, d) - r^0_j(t_c, d)$ and interpret this as a comprehensive treatment effect. In any case, it is only the comprehensive treatment effect which can be observed by comparing outcomes in the treatment and the control group, and only this CTE has an immediate causal interpretation in model M1.

It is possible, however, to decompose the instantaneous CTE, $\Delta_j(t_c, 0)$, into a selection effect and (a version of) a 'hypothetically pure' treatment effect. I use the notations $r^1_j(t_c, d; x)$ and $r^0_j(t_c, d; x)$, defined by adding the condition $X(t_c) = x$ on the right-hand side of (1) and (2), respectively. The hazard functions from which the instantaneous CTE is derived can be written as

$$r^s_j(t_c, 0) = \sum\nolimits_x r^s_j(t_c, 0; x) \Pr(X(t_c){=}x \,|\, S(t_c){=}s, T_1 \geq t_c)$$

(for $s = 0, 1$). Using then $\Delta_j(t_c, d; x) := r^1_j(t_c, d; x) - r^0_j(t_c, d; x)$, one can write:

$$\begin{aligned}\Delta_j(t_c, 0) = & \\ &\sum\nolimits_x \big[\Pr(X(t_c){=}x \,|\, S(t_c){=}1, T_1 \geq t_c) - \\ &\qquad \Pr(X(t_c){=}x \,|\, S(t_c){=}0, T_1 \geq t_c)\big]\, r^0_j(t_c, 0; x) + \\ &\sum\nolimits_x \Delta_j(t_c, 0; x) \Pr(X(t_c){=}x \,|\, S(t_c){=}1, T_1 \geq t_c)\end{aligned} \tag{6}$$

The first term on the right-hand side can be interpreted as a selection effect, resulting from the selection of units for treatment. So it must be viewed as part of the causal effect of the action of the experimenter or institution. The second term on the right-hand side can be interpreted as an average treatment effect, where the average is with respect to the distribution of covariates in the treatment group. Note that this effect is a theoretical construction and, in general, not equal to $\Delta(t_c, 0)$ which, as an observable effect, is to be interpreted as a CTE. Equality only holds if there is no selection effect.

The selection effect becomes particularly clearly visible if, actually or hypothetically, there is no treatment effect, meaning that $r^1_j(t_c, 0; x) = r^0(t_c, 0; x)$ for all x. The initial selection effect can then be written as

$$\begin{aligned}&\Pr(T_1{=}t_c, E_1{=}j \,|\, S(t_c){=}1, T_1 \geq t_c) - \\ &\qquad \Pr(T_1{=}t_c, E_1{=}j \,|\, S(t_c){=}0, T_1 \geq t_c)\end{aligned}$$

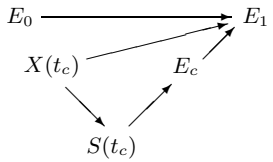
The two terms simply refer to the probability of $E_1 = j$ in the treatment and the control group, respectively.

The second term in (6) is one version of a balanced effect which, hypothetically, compares two groups having approximately the same distribution of covariates. A balanced formulation is often supposed to be required for a causal interpretation. However, the CTE circumvents this requirement. This is possible by viewing the CTE as resulting from a process consisting, not only of treatments, but also of events which assign treatments to units. The argument is, of course, that also the latter events play a causally relevant role.

Self-selected treatments

Self-selected treatments come into being through the behavior of the units in $\mathcal{R}(t_c, 0)$. One cannot refer to an agent (experimenter or institution) who, based on consideration of $X(t_c)$ and/or by using a random generator, creates a partition of $\mathcal{R}(t_c, 0)$ into a treatment and a control group, and the model M1 can therefore not be used. For each primary agent, values of $X(t_c)$ are given, and the selection can only concern the treatment. Of course, one should assume that these selections depend in some way on $X(t_c)$, and this leads one to the following model.

Model M2



In contrast to M1, an arrow now leads from $X(t_c)$ to $S(t_c)$, but also the meaning of the 'decision node', $S(t_c)$, has changed. In model M1, $S(t_c)$ represents the behavior of an experimenter or institution. In model M2, $S(t_c)$ represents the behavior of a primary agent defined by being a member of $\mathcal{R}(t_c, 0)$ and characterized by a particular value of $X(t_c)$.

Note that, in model M2, one cannot 'hypothetically dismiss' the arrow from $X(t_c)$ to $S(t_c)$.[3] Dropping this arrow would lead to an essentially different model which entails the assumption that $X(t_c)$ does not play a role in the primary agents' selection of treatments. When concerned with model M2 for self-selected treatments, $S(t_c)$ must be considered as an endogenous variables.

In the following I assume again a deterministic relationship between $S(t_c) = 1$ and the treatment without a temporal delay. So one could also consider a simplified version of M2 in which $S(t_c)$ is omitted and there is a single arrow from $X(t_c)$ to E_c. However, an explicit reference to $S(t_c)$ can ease the understanding.

[3] This has been suggested as a requirement for definitions of a 'causal effect', see, e.g., Pearl (2000). Then, however, only model M1, not M2, could be used as a framework for causal considerations.

Although M1 and M2 are essentially different models, they are in an important respect comparable. Both models can be considered as describing the generation of a treatment and a control group. In model M1, this is done by the experimenter or institution, in model M2, this is a result of individual behavior. Therefore, also M2 can be used to derive conditional distributions of $X(t_c)$ having an interpretation comparable with (5):

$$\Pr(X(t_c)=x \mid S(t_c)=s, T_1 \geq t_c) = \frac{\Pr(S(t_c)=s \mid X(t_c)=x, T_1 \geq t_c)\Pr(X(t_c)=x \mid T_1 \geq t_c)}{\sum_{x'} \Pr(S(t_c)=s \mid X(t_c)=x', T_1 \geq t_c)\Pr(X(t_c)=x' \mid T_1 \geq t_c)} \tag{7}$$

In both models, the initial distribution of $X(t_c)$ (conditional on $T_1 \geq t_c$) is fixed at the beginning of t_c. In addition, (7) only requires the reference to a function $x \longrightarrow \Pr(S(t_c)=s \mid X(t_c)=x)$ describing the self-selection of primary agents.

Given this comparability, the partition (6) can also be used for model M2. One can distinguish a selection effect resulting from the primary agents' self-selection into a treatment and a control group and, in addition, a hypothetically pure treatment effect. Moreover, also the CTE has a comparable meaning in both models: in M1, it is the effect of the behavior of the experimenter or institution, in M2 it is the result of the behavior (decisions) of the primary agents.

Temporally remote effects

I now consider treatment effects in temporal locations $t_c + d$ for $d > 0$. I assume that these effects result from what is actually the case at the beginning of the temporal location $t_c + d$ (no 'action at a distance'). The main question then concerns how the treatment at t_c has contributed to the development of the situation at the beginning of $t_c + d$. One obvious idea is that a unit who experienced a treatment at t_c is thereby changed in some way, and this new feature of the unit and/or her environment endures for temporal locations beyond t_c. A complementary idea is that the treatment

influences the occurrence of further events after the treatment which then in turn influence the occurrence of outcome events.

Since the effect definition is based on comparing outcomes in a treatment and a control group one also has to take into account selection effects occurring after the treatment. These effects can be considered by using a partition analogous to (6) for temporal locations beyond t_c:

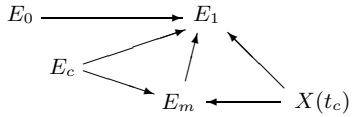
$$\begin{aligned}\Delta_j(t_c, d) = \quad & (8)\\ & \sum\nolimits_x \big[\Pr(X(t_c){=}x \mid S(t_c){=}1, T_1 \geq t_c + d) - \\ & \qquad \Pr(X(t_c){=}x \mid S(t_c){=}0, T_1 \geq t_c + d)\big]\, r_j^0(t_c, d; x) + \\ & \sum\nolimits_x \Delta_j(t_c, d; x) \Pr(X(t_c){=}x \mid S(t_c){=}1, T_1 \geq t_c + d)\end{aligned}$$

The second term on the right-hand side can again be interpreted as an average treatment effect, where the average now is with respect to the distribution of covariates in the set of treated units who are still in σ_0 at $t_c + d$. The first term on the right-hand side represents an effect resulting from a difference in the distributions of covariates in the treatment and the control group at $t_c + d$. This difference has two sources. First, a difference at t_c when the treatment occurred. A second source are E_1 events which depend on the covariates and therefore change their distributions differently in the treatment and the control group. This also shows that an important part of the selection effect cannot be eliminated by a random assignment of treatments at t_c.

Both sources of the selection effect can be causally interpreted. As I argued above, the first source results from treatment assignment done by an experimenter or institution (in model M1) or from the behavior of primary agents (in model M1). The second source results from the occurrence of E_1 events. Of course, these events change both the treatment and the control group and consequently the respective conditional distributions of $X(t_c)$. However, the development of the *difference* between these two distributions can be traced back to the selection and application of treatments at t_c.

I now consider events which might occur after the treatment and influence the occurrence of outcome events. To fix ideas, I refer to a single event, represented by the binary event variable E_m. Since the treatment can only occur once, this is not a time-dependent confounder but can be viewed as a variable mediating the treatment effect:

Model M3



The relationship between $X(t_c)$ and E_c can be specified as in model M1 or M2.

There are three possible approaches. (a) One can consider E_m as a variable mediating the comprehensive treatment effect. (b) One can be interested in the effect of E_m on the occurrence of E_1 events, conditional on whether there was a treatment before. (c) One can consider (E_c, E_m) as representing sequences of possible events and investigate how the occurrence of E_1 events depends on such sequences.[4]

If the interest mainly concerns effects of the treatment, one can follow the first approach. Of course, given that E_m also depends on $X(t_c)$, E_m must be considered as mediating a joint effect of the treatment and the covariates. However, the dependence of E_m on $X(t_c)$ and E_c can be viewed like the dependence of E_c on $X(t_c)$; E_c is simply a further component in the vector of covariates relevant for the occurrence of E_m. Moreover, because one is interested in effects of the treatment, it seems not necessary to apply the distinction between self-selected and heteronomous treatments also to E_m. Independent of how E_m events come into being, one can think of E_m as mediating the CTE of the treatment.

[4] A version of this approach was proposed by Lechner and Miquel (2010).

4 Temporally extended effects

Effect shapes make effects dependent on both the time when the treatment occurred and the duration since the treatment occurred. One also might be interested in effects which concern the probability of an outcome event ($E_1 = j$) in a time interval after the occurrence of the treatment.

Event probabilities for treated units

Given a treatment at t_c, one has to consider $p_j^1(t_c, \delta) :=$ the probability of the occurrence of $E_1 = j$ in the time interval $[t_c, t_c + \delta]$. Since the situation σ_0 could end by events different from $E_1 = j$, this probability cannot simply be derived from knowing the hazard function $r_j^1(t_c, d)$ defined in (1). One also needs the survivor function for still being in σ_0 until $t_c + d$, given that a treatment occurred at t_c:

$$G^1(t_c, d) := \prod_{k=0}^{d-1} (1 - r^1(t_c, k)) \tag{9}$$

where $r^1(t_c, k) := \sum_{j>0} r_j^1(t_c, k)$ and $G^1(t_c, 0) := 1$. So one can write

$$p_j^1(t_c, \delta) = \sum_{d=0}^{\delta} r_j^1(t_c, d)\, G^1(t_c, d) \tag{10}$$

Correspondingly, one can consider a treatment group $\mathcal{R}^1(t_c, 0) :=$ a set of units who experienced the treatment at t_c. With an event set $\mathcal{E}_j^1(t_c, 0{:}\delta) :=$ all members of $\mathcal{R}^1(t_c, 0)$ who experienced the event $E_1 = j$ in $[t_c, t_c{+}\delta]$, one can use $\#\mathcal{E}_j^1(t_c, 0{:}\delta)/\#\mathcal{R}^1(t_c, 0)$ to estimate (10). This can be illustrated with the data in Table 1. Assuming $t_c = 2$ and $\delta = 2$, one finds

d	$r_1^1(2, d)$	$G^1(2, d)$
0	1/4	4/4
1	1/3	3/4
2	1/2	2/4

Summing up, the probability defined in (10) is 3/4. The same value results from the treatment group $\mathcal{R}^1(2, 0) = \{11, 12, 13, 14\}$ and the event set $\mathcal{E}^1(2, 0:2) = \{11, 12, 13\}$.

Event probabilities for the comparison

There is no obvious way to define the probability of the occurrence of $E_1 = j$ in the time interval $[t_c, t_c + \delta]$ for not treated units. I use a definition that is consistent with the definition of effects in terms of hazard functions (see also Lalive et al. 2008, Crépon et al. 2008). One can begin with a survivor function paralleling (9):

$$G^0(t_c, d) = \prod_{k=0}^{d-1} (1 - r^0(t_c, k)) \tag{11}$$

where $r^0(t_c, k) := \sum_{j>0} r_j^0(t_c, k)$ and $G^0(t_c, 0) := 1$. $G^0(t_c, d)$ can be interpreted as the time-dependent probability of staying in the situation σ_0 without experiencing a treatment. The probability for comparison can then be defined as

$$p_j^0(t_c, \delta) := \sum_{d=0}^{\delta} r_j^0(t_c, d)\, G^0(t_c, d) \tag{12}$$

and the temporally extended effect can be defined as

$$\Delta_j^e(t_c, \delta) := p_j^1(t_c, \delta) - p_j^0(t_c, \delta) \tag{13}$$

To illustrate with the data in Table 1, and assuming again $t_c = 2$ and $\delta = 2$, one finds:

d	$r_1^0(2, d)$	$G^0(2, d)$
0	1/16	1.000
1	1/12	0.938
2	1/9	0.859

Summing up, the probability defined in (12) is 0.236, and the temporally extended effect is $\Delta_1^e(2, 2) = 0.750 - 0.236 = 0.514$.

Note that one cannot define just one control group for estimating (12). In particular, one cannot use a set of units who did not experience the treatment until, and including, t_c. In our example, this would be the set $\{1 - 10, 15 - 20\}$, and the corresponding event set would be $\{1, 2, 3\}$, resulting in 3/16. Likewise, one cannot use a set of units who did not

experience the treatment until, and including, $t_c+\delta$. In our example, this would be the set $\{1-10,20\}$, and the corresponding event set would be $\{1,2,3\}$, resulting in 3/11.

Causal interpretation of extended effects

Thinking of causation as a temporally local relationship, $\Delta^e_j(t_c,\delta)$ should be considered as resulting from a process, extending from t_c to t_c+d, generating $E_1=j$ events. This can be made explicit by writing $\Delta^e_j(t_c,\delta)=\sum_{d=0}^{\delta}\Delta^p_j(t_c,d)$, where

$$\Delta^p_j(t_c,d) := r^1_j(t_c,d)\,G^1(t_c,d) - r^0_j(t_c,d)\,G^1(t_c,d) \tag{14}$$

This shows that the extended effect is not a simple summary of the temporally local effects which are defined by a reference to the hazard functions $r^s_j(t_c,d)$, but also depends on surviving in the situation σ_0. This is relevant for interpreting the extended effect because surviving in σ_0 also depends on concurrent events as specified in the domain of E_1. One cannot easily interpret an extended treatment effect with respect to a specified event, $E_1=j$, without taking into account how the treatment influences concurrent events. To illustrate, assume $\Delta_j(t_c,d)=0$ for all d. However, as shown by

$$\Delta^e_j(t_c,\delta)=\sum_{d=0}^{\delta}\Delta_j(t_c,d)\,G^1(t_c,d)+\big[G^1(t_c,d)-G^0(t_c,d)\big]r^0_j(t_c,d)$$

there can well be an extended effect for $E_1=j$ due to different surviving probabilities of treated and not treated units. A temporally extended effect is therefore not a sufficient evidence for there being a treatment effect for a specified outcome event.

A further argument for considering effect shapes, before using temporally extended effects as summaries, concerns that the extended effect can hide important changes in the underlying effect shape. For example, if an extended effect is zero, this could hide an effect shape which is first positive and then negative.[5]

[5] An example dealing with the effect of a women's pregnancy (E_c) on marriage (E_1) in consensual unions was discussed by Blossfeld et al. (1999).

5 Discussion

The paper considers a version of the question of how to define treatment and control groups in a dynamic setting where treatments can occur at any time (but only once). The version considered presupposes that treatments as well as outcomes can be conceptualized as events occurring in temporal locations of a discrete time axis. This motivates to think of effects as being dependent on both the time when and the time since the treatment occurred.

An essential step in the argument concerns that one should start from a temporally local conception of effects. Given that a treatment occurred in temporal location t_c, effects should be defined separately for each temporal location t_c+d ($d=0,1,2,\ldots$). Combining this with the idea that effects should be estimated by comparing a treatment and a control group, also these groups should be defined separately for each t_c+d.

Since treatments can only occur once, a treatment group $R^1(t_c,d)$ can easily be defined as the set of all units who experienced the treatment at t_c and are still at risk for experiencing the outcome event in temporal location t_c+d. Following a temporally local view, the control group, $R^0(t_c,d)$, should not contain units who experienced a treatment before t_c+d (postulate 1), but also should not exclude units who might experience the treatment at a later time (postulate 2). The second postulate can be justified with the argument that one is interested in causally interpretable effects, and their definition for a temporal location t_c+d must not depend on events which might occur later than t_c+d.

In order to find a causal interpretation one has to start from the question of how treatments are generated. This paper considers two models. In model M1, treatments are generated by an experimenter or institution, in model M2, treatments are self-selected by primary agents. In both models, the generation of treatments starts from a selection of units to be treated. Since outcomes depend on properties of the units under consideration, the selection must be viewed as an essential part of the process which eventu-

ally leads to treatment effects. This motivates to consider the difference of outcomes in the treatment and the control group as a 'comprehensive treatment effect' (CTE) resulting from both a division of units into two groups and applying the treatment to the units in one of these groups.

The CTE can be viewed as consisting of two parts: a selection effect and a 'pure' treatment effect. However, the two components cannot be observed separately and their separation must therefore be understood as a theoretical construction. Since the process generating the CTE begins with the selection of treated and not treated units, it seems natural to begin with the definition of the selection part of the CTE.

The selection part of the CTE results from variables which are relevant for the outcome and differently distributed in the treatment and the control group. In order to avoid a confounding with effects of the treatment, its definition should hypothetically assume that the generation of the treatment and the control group is not followed by actually applying the treatment. This can be achieved by using the control group as a reference as done in (6) and (8). The remainder of the CTE can be viewed as a constructed pure treatment effect.

This is consistent with the idea that randomized experiments can provide estimates of pure treatment effects. The argument simply is that such experiments intentionally avoid selection effects. However, such experiments are no substitute for modeling and understanding processes which actually begin with a nonrandom selection of treated and not treated units. Moreover, also treatment effects estimated with randomized experiments depend on the distribution of covariates in the sample of units selected for the experiment. This is true, in particular, when treatments and covariates interact so that conditional treatment effects, $\Delta_j(t_c, d; x)$, depend on $X(t_c) = x$. Therefore, treatment effects estimated with randomized experiments cannot easily be used for partitioning a CTE into a selection and a treatment part.

Accepting the CTE as a notion having a sensible (causal) interpretation also suggests a somewhat different understanding of estimating a pure treatment effect. A pure treatment effect is to be understood as a

theoretically constructed part of a comprehensive treatment effect, and the selection effect is to be understood as an essential complement. Estimation problems due to unobserved components of $X(t_c)$ only concern the partition of the CTE into the two components and do not affect the estimate of the CTE.

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