# Treatment and Control Groups in a Dynamic Setting

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**Summary.** 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. It is proposed to think of effects as being dependent on both the time when and the time since the treatment occurred. The paper develops corresponding definitions of treatment and control groups, and proposes a notion of 'comprehensive treatment effect' that takes into account how treatment and control groups are generated. Based on this notion, the paper discusses causal interpretations which do not presuppose a potential outcomes framework.

*Keywords:* Treatment group; control group; effect shape; comprehensive treatment effect; causal interpretation

## 1 Introduction

Thinking about effects of some specified factor often proceeds by 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.

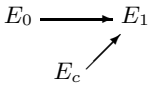
While the basic idea is straightforward, difficulties can arise in a dynamic setting. These difficulties concern the definition of a control group. Assume that the treatment is defined by experiencing a specific kind of event. So one can define a treatment group as a set of units who experienced the 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 $\delta$? 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 & Johansson, 2008). In contrast, Kohler et al. (2012) have used a relatively large value of $\delta$, and Brand & Xie (2007) proposed a 'composite of counterfactuals' based on a reference to several values of $\delta$.

In this paper, I start from hazard functions which allow defining effects in a temporally local way and therefore avoid fixing $\delta$ 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 time-dependent 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 consider 'temporally extended effects' which concern event probabilities in an extended time interval after the treatment, and I show that such effects can suggest wrong interpretations due to competing risks. In Section 4 I discuss how effects defined by a comparison of treated and not treated units can be given a causal interpretation without presupposing a potential outcomes framework. This means that I avoid postulating the existence of variables representing counterfactual quantities, and I do not presuppose joint probability distributions for such variables. Instead, I only use definitions based on observable variables. I propose a notion of 'comprehensive treatment effect' which does not require an actual or fictitious random assignment of treatments and can therefore be used, in particular, to understand effects of self-selected treatments. Section 5 concludes.

## 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 $\sigma_0$ 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.

As an example, one can think of consensual unions. The event $E_0 = 1$ is defined as the beginning of a consensual union. The outcome event describes how the union ends: in a marriage ($E_1 = 1$), in a separation ($E_1 = 2$) or through the death of one of the partners ($E_1 = 3$). The question is how the probability of a marriage depends on the occurrence of a pregnancy ($E_c = 1$).[1]

In order to refer to events, I presuppose a discrete time axis, $\mathcal{T} := \{0, 1, 2, \ldots\}$, measuring time since the occurrence of $E_0$ (so the calendar time of the occurrence of $E_0$ is not used in subsequent considerations). Elements of $\mathcal{T}$ will be called 'temporal locations' (e.g. days, months or years). $E_1$ events will be represented by a duration variable $(T_1, E_1)$ where $E_1 \in \{1, \ldots, m_1\}$ 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 focusses on the occurrence of an event $E_1 = j$. As will be shown in sections 3 and 4, it is nevertheless important to take into account competing risks as assumed by the domain of $E_1$. One can begin with a hazard function

$$r_j^1(t_c, d) := \Pr(T_1 = t_c + d, E_1 = j \mid T_1 \geq t_c + d, T_c = t_c) \tag{1}$$

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 $\sigma_0$ at $t_c + d$.

[1] For an empirical illustration see Blossfeld et al., 1999.

There is then the question of how to define a sensible comparison. I follow the idea to begin with a comparison that depends both on $t_c$ and $d$, and require that the treatment did not occur until $t_c + d$.[2] The hazard function to be used for the comparison can be written as

$$r_j^0(t_c, d) := \Pr(T_1 = t_c + d, E_1 = j \mid T_1 \geq t_c + d, T_c > t_c + d) \tag{2}$$

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 $t_c + d$.)

Effects of the treatment which depend both on the treatment time, $t_c$, and on the time since the occurrence of the treatment, $d$, can be defined by

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

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*

A reference to temporally local effects requires that treatment and control groups must be defined in a time-dependent way. With respect to (3), one can define a treatment group $\mathcal{R}^1(t_c, d)$ as a set of units who experienced the treatment at $t_c$ and are still in the situation $\sigma_0$ at $t_c + d$, and a control group $\mathcal{R}^0(t_c, d)$ as a set of units who are still in $\sigma_0$ at $t_c + d$ and did not experience the treatment until, and including, $t_c + d$. With corresponding event sets $\mathcal{E}_j^s(t_c, d)$ consisting of 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.)

[2] This is similar to what has been called a 'timing of events approach' in labor market research, see, e.g., Abbring & van den Berg (2003), Fredriksson & Johansson (2008), Lalive et al., (2008), Crépon et al., (2009), Vikström (2014). 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 | $\infty$ | 2 | 11 | 2 | 2 |
| 2 | $\infty$ | 3 | 12 | 2 | 3 |
| 3 | $\infty$ | 4 | 13 | 2 | 4 |
| 4 | $\infty$ | 5 | 14 | 2 | 5 |
| 5 | $\infty$ | 5 | 15 | 3 | 5 |
| 6 | $\infty$ | 6 | 16 | 3 | 7 |
| 7 | $\infty$ | 6 | 17 | 3 | 8 |
| 8 | $\infty$ | 7 | 18 | 4 | 7 |
| 9 | $\infty$ | 8 | 19 | 4 | 8 |
| 10 | $\infty$ | 9 | 20 | 5 | 9 |

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 $T_c$. In this simplified example, 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.[3] Using such a dynamic

[3]Several researchers have used this idea before, see e.g. Crépon et al. (2009), Hernán et al. (2008), Gran et al. (2010), Vikström (2014).

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, available not later than $t_c + d$, about the units which do not have experienced the treatment until, and including, $t_c + d$. No assumptions are made about the behavior of units in $\mathcal{R}^0(t_c, d)$ 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. This is important because in many applications, in particular when treatments are completely or partially self-selected by human individuals, making this assumption would contradict the process under investigation. This will be further considered in the discussion of models to be used for the definition and interpretation of effects.

### *A contrast between 'now' and 'waiting'?*

Sianesi motivated her approach by referring to a choice between receiving a treatment 'now' and 'waiting' (Sianesi, 2004, 2008; see also Fitzenberger et al., 2013; Fredriksson & Johansson, 2008). A hazard function representing 'waiting' might be defined by

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

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 a possibly interesting quantity, it seems not possible to interpret $\Delta_j^*(t_c, d)$ as representing a causal effect of the treatment occurring at $t_c$. This is reasonable because 'waiting' simply leaves it open what will happen subsequently. It only has a definite meaning for 'now' ($d = 0$), and then $\Delta_j^*(t_c, 0) = \Delta_j(t_c, 0)$. On the other hand, if $d > 0$, $\Delta_j^*(t_c, d)$ and $\Delta_j(t_c, d)$ differ. In order to compare both definitions, one needs a hazard function for the occurrence of treatments in the control group, say

$$h(t_c, k) := \Pr(T_c = t_c + k \,|\, T_1 \geq t_c + k, T_c \geq t_c + k) \tag{5}$$

For example, in the temporal location $t_c + 1$, the relationship is

$$\Delta_j^*(t_c, 1) = \Delta_j(t_c, 1) \; - h(t_c, 1)\, \Delta_j(t_c + 1, 0) \tag{6}$$

Assume $r_j^1(t_c, 1) = r_j^1(t_c + 1, 0) = 0.2$; $r_j^0(t_c, 1) = 0.1$, and $h(t_c, 1) = 0.05$. Then $\Delta_j(t_c, 1) = 0.1$, but $\Delta_j^*(t_c, 1) = 0.1 - 0.05 \cdot 0.1 = 0.095$. This illustrates how Sianesi's contrast not only depends on treatments occurring later than $t_c$, but also reflects treatment effects defined for different temporal locations.

# 3 Temporally extended effects

Effect shapes make effects dependent on both the time of treatment occurrence and the duration since the treatment occurrence. 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 $\sigma_0$ 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 (beginning at $t_c$) for still being in $\sigma_0$ until $t_c + d$, given that a treatment occurred at $t_c$:

$$G^1(t_c, d) := \prod_{k=0}^{d-1} (1 - r^1(t_c, k)) \tag{7}$$

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

$$p_j^1(t_c, \delta) = \sum_{d=0}^{\delta} r_j^1(t_c, d)\, G^1(t_c, d) \tag{8}$$

Correspondingly, one can define a treatment group $\mathcal{R}^1(t_c, 0)$ as a set of units who experienced the treatment at $t_c$. With an event set $\mathcal{E}_j^1(t_c, 0:\delta)$, consisting of all members of $\mathcal{R}^1(t_c, 0)$ who experienced the event $E_1 = j$ in $[t_c, t_c+\delta]$ while still being in $\sigma_0$, one can use $\#\mathcal{E}_j^1(t_c, 0:\delta)/\#\mathcal{R}^1(t_c, 0)$ to estimate (8). 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 (8) 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. 2009). One can begin with a survivor function paralleling (7):

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

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 $\sigma_0$

without experiencing a treatment. The probability for comparison can then be defined as

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

and the temporally extended effect can be defined as

$$\Delta_j^e(t_c, \delta) := p_j^1(t_c, \delta) - p_j^0(t_c, \delta) \tag{11}$$

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 (10) 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 (10). 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.

### *Interpretation of extended effects*

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

$$\Delta_j^p(t_c, d) := r_j^1(t_c, d)\, G^1(t_c, d) - r_j^0(t_c, d)\, G^0(t_c, d)$$

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

$r_j^s(t_c, d)$, but also depends on surviving in the situation $\sigma_0$. This is relevant for interpreting the extended effect because surviving in $\sigma_0$ also depends on other 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 competing events. To illustrate, assume $\Delta_j(t_c, d) = 0$ for all $d$. As shown by

$$\Delta_j^e(t_c, \delta) = \sum_{d=0}^{\delta} \Delta_j(t_c, d)\, G^1(t_c, d) + \big[G^1(t_c, d) - G^0(t_c, d)\big] r_j^0(t_c, d)$$

there can well be an extended effect for $E_1 = j$ due to different probabilities of surviving for 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 in favor of considering effect shapes, in addition to temporally extended effects, 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.[4]

# 4 Causal interpretations

Treatments are here 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 'becoming married', 'finding a job', '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

[4] 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).

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 many 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 sections, 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 $\sigma_0$ 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 $t_c$.

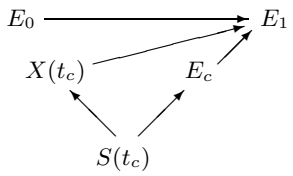
Effects concern the occurrence of $E_1$ events in temporal locations $t_c + d$. So one can distinguish between instantaneous effects ($d = 0$) and temporally remote effects ($d > 0$). I begin with considering instantaneous effects.

### *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 this 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 a unit 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. However, the model should be understood as presupposing a fixed group of units, $\mathcal{R}(t_c, 0)$, which can possibly be selected for treatment (represented by the arrow leading from $S(t_c)$ to $X(t_c)$). The model is silent about the generation of this group and, in particular, does not exclude that processes of self-selection, also based on the anticipation of possible treatments, play a relevant role in its generation.

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 $X(t_c)$. 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

$$\Pr(X(t_c) = x \mid S(t_c) = s, T_1 \geq t_c) \tag{12}$$

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 $S(t_c)$.

### *Comprehensive treatment effects*

Presupposing model M1, one should start from the effect of the selection variable $S(t_c)$ on the occurrence of $E_1$ events. As the generation of this effect starts from the selection of units for treatment (and not just with the occurrence of the treatment), it will be called a 'comprehensive treatment effect' (CTE). One can begin with the definition

$$\begin{aligned}&\Pr(T_1 = t_c + d, E_1 = j \mid S(t_c) = 1, T_1 \geq t_c + d) - \\ &\quad \Pr(T_1 = t_c + d, E_1 = j \mid S(t_c) = 0, T_1 \geq t_c + d)\end{aligned} \tag{13}$$

The instantaneous CTE (if $d = 0$) is identical with $\Delta_j(t_c, d)$. However, if $d > 0$, the definition is ambiguous because the condition $S(t_c) = 0$ leaves it open whether there might be treatments in temporal locations later than $t_c$. Therefore, in order to allow thinking of temporally remote effects, one needs an explicit definition of the control group in temporal locations later than $t_c$. Based on the two postulates discussed in Section 2, one should add the condition $T_c > t_c + d$ to the second term in (13), which relates to the control group:

$$\begin{aligned}&\Pr(T_1 = t_c + d, E_1 = j \mid S(t_c) = 1, T_1 \geq t_c + d) - \\ &\quad \Pr(T_1 = t_c + d, E_1 = j \mid S(t_c) = 0, T_c > t_c + d, T_1 \geq t_c + d)\end{aligned} \tag{14}$$

So defined, the CTE is equal to $\Delta_j(t_c, d)$ for all $d \geq 0$.

The following discussion is based on this understanding, that is, $\Delta_j(t_c, d)$ is interpreted as a comprehensive treatment effect which begins with the selection of units for treatment. I first consider instantaneous, then temporally remote effects.

### *Separating selection and treatment effects*

The instantaneous CTE, $\Delta_j(t_c, 0)$, can be decomposed into a selection effect and (a version of) a balanced treatment effect. I use the notations $r_j^1(t_c, d; x)$ and $r_j^0(t_c, d; x)$, 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

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

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

$$\begin{aligned}\Delta_j(t_c,0) = & \qquad (15)\\ &\sum\nolimits_x \big[\Pr(X(t_c){=}x \mid S(t_c){=}1, T_1 \geq t_c) - \\ &\qquad \Pr(X(t_c){=}x \mid S(t_c){=}0, T_1 \geq t_c)\big]\, r_j^0(t_c,0;x) + \\ &\sum\nolimits_x \Delta_j(t_c,0;x)\,\Pr(X(t_c){=}x \mid S(t_c){=}1, T_1 \geq t_c)\end{aligned}$$

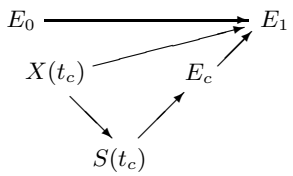
The first term on the right-hand side can be interpreted as a selection effect, resulting from the selection of units for treatment. The second term on the right-hand side can be interpreted as an average treatment effect, where the average is with respect to the distribution of covariates in the treatment group.

Both parts play a role in a causal interpretation of the CTE that shows how the CTE is generated. As suggested by model M1, the first part of the decomposition can be interpreted as an indirect effect of $S(t_c)$ which is mediated by the selection of a value of $X(t_c)$. Due to the deterministic relationship between $S(t_c) = 1$ and $E_c = 1$, the second part of the decomposition can be interpreted as a version of a direct effect of $S(t_c)$. In general, one has to suppose that the conditional effects, $\Delta_j(t_c,0;x)$, depend on $x$, and the definition of the direct effect therefore depends on the distribution of the covariates which is used for averaging. This remains true even if units are randomly selected for treatment. In observational studies there is the further problem that one cannot presuppose that one has observed all covariates which are causally relevant for the outcome event and whose distributions differ in the treatment and the control group. In any case, the balanced treatment effect, that is, the direct effect of $S(t_c)$, must be interpreted as a theoretical construct resulting from a particular decomposition of the observed CTE.

*Self-selected treatments*

Self-selected treatments arise 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 to the following model.

Model M2




In contrast to M1, an arrow now leads from $X(t_c)$ to $S(t_c)$. Moreover, 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, a member of $\mathcal{R}(t_c, 0)$ 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 $S(t_c)$.[5] Removing 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 variable; and this model is therefore incompatible with the idea of a randomized treatment assignment.

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 $S(t_c)$

[5] This has been suggested as a requirement for definitions of a 'causal effect', see, e.g., Pearl (2009). Then, however, only model M1, not M2, could be used as a framework for causal considerations.

can ease the understanding.

Although M1 and M2 are quite 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 as an essential part of the process leading to outcome events. In model M1, this is done by the experimenter or institution, in model M2, this is a result of the primary agents' behavior. Therefore, also M2 can be used to derive conditional distributions of $X(t_c)$ having an interpretation comparable with (12):

$$\Pr(X(t_c)=x \mid S(t_c)=s, T_1 \geq t_c) = \frac{\Pr(S(t_c)=s \mid X(t_c)=x, T_1 \geq t_c)\Pr(X(t_c)=x \mid T_1 \geq t_c)}{\sum_{x'} \Pr(S(t_c)=s \mid X(t_c)=x', T_1 \geq t_c)\Pr(X(t_c)=x' \mid T_1 \geq t_c)} \tag{16}$$

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, (16) only requires the reference to a function $x \longrightarrow \Pr(S(t_c)=s \mid X(t_c)=x, T_1 \geq t_c)$ describing the self-selection of primary agents. So also model M2 allows one to think that the conditional distributions of $X(t_c)$ result from the event variable $S(t_c)$.

It follows that the notion of a CTE which starts from $S(t_c)$ can also be used for interpreting model M2. In temporal location $t_c$, the effect compares the probabilities of outcome events $E_1 = j$ between units having chosen a treatment and units not having chosen a treatment. The decomposition (15) can again be used to distinguish a selection effect and a balanced treatment effect. As before, in order to interpret the balanced part as a 'pure' treatment effect, $X(t_c)$ should include all relevant confounding variables which influence both the self-selection of treatments and the outcome event.

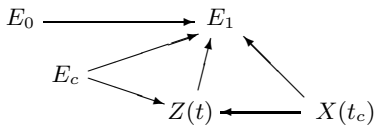
### *Temporally remote effects*

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 $t_c+d$. 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 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.

In order to represent events which may occur after the treatment I use a time-varying covariate, $Z(t)$, possibly consisting of several components, which is defined for $t \geq t_c$ and may depend on $X(t_c)$ and the treatment status. It is assumed that all units have the same value $Z(t_c) = z_0$. I refer to the following model where the relationship between $X(t_c)$ and $E_c$ can be specified as in model M1 or M2.

Model M3




It is assumed that values of $Z(t)$ are realized at the beginning of temporal locations, depending on the situation at the end of the previous location, so that the occurrence of an $E_1$ event in a temporal location $t_c + d$ depends on the history of $Z(t)$ until, and including, $t_c + d$. This history will be denoted by $\underline{Z}(t_c+d)$.

The following discussion concerns the interpretation of $\Delta_j(t_c, d)$, a temporally remote CTE. To begin with, I consider temporally remote effects conditional on given values of the covariates $X(t_c)$:

$$\Delta_j(t_c, d; x) = r_j^1(t_c, d; x) - r_j^0(t_c, d; x) \tag{17}$$

Both hazard functions depend on values of $\underline{Z}(t_c + d)$. For units in the treatment group one can write

$$r_j^1(t_c, d; x) = \sum_{\underline{z}_d} r_j^1(t_c, d; x, \underline{z}_d) \Pr(\underline{Z}(t_c+d)=\underline{z}_d \mid T_1 \geq t_c+d, T_c=t_c, X(t_c)=x) \tag{18}$$

Values of $\underline{Z}(t_c+d)$, for $d > 0$, are generated sequentially:

$$z_0 \longrightarrow z_1 \longrightarrow z_2 \longrightarrow z_3 \longrightarrow \cdots \longrightarrow z_d$$

according to

$$\Pr(\underline{Z}(t_c{+}d){=}\underline{z}_d \,|\, T_1 \geq t_c{+}d, T_c{=}t_c, X(t_c){=}x) = \tag{19}$$
$$\prod_{k=0}^{d-1} \Pr(Z(t_c{+}k{+}1){=}z_{k+1} \,|\, T_1 \geq t_c{+}k{+}1, T_c{=}t_c,$$
$$X(t_c){=}x, \underline{Z}(t_c{+}k) = \underline{z}_k) \, \frac{1 - r^1(t_c, k; x, \underline{z}_k)}{1 - r^1(t_c, k; x)}$$

The factor $[1-r^1(t_c,k;x,\underline{z}_k)]/[1-r^1(t_c,k;x)]$ is required for updating the distribution of $X(t_c)$, in temporal location $t_c{+}k$, on which the distribution of $Z(t_c{+}k{+}1)$ depends. Note that the hazard functions in this factor relate to all kinds of $E_1$ events:

$$r^1(t_c, k; x) := \Pr(T_1 = t_c{+}k \,|\, T_1 \geq t_c{+}k, T_c{=}t_c, X(t_c) = x) \tag{20}$$
$$r^1(t_c, k; x, \underline{z}_k) :=$$
$$\Pr(T_1 = t_c{+}k \,|\, T_1 \geq t_c{+}k, T_c{=}t_c, X(t_c) = x, \underline{Z}(t_c{+}k) = \underline{z}_k)$$

This entails that hazard functions for the focal event $E_1 = j$ also depend on hazard functions for competing risks. Nevertheless, since all hazard functions and conditional probabilities on the right-hand side of (19) are defined by model M3, $r^1_j(t_c, d; x)$ can be interpreted as a hazard function, averaged w.r.t. all possible values of $\underline{Z}(t_c{+}d)$, which only depends on the treatment status and $X(t_c) = x$.

For units in the control group one can write:

$$r^0_j(t_c, d; x) = \tag{21}$$
$$\sum\nolimits_{\underline{z}_d} r^0_j(t_c, d; x, \underline{z}_d) \Pr(\underline{Z}(t_c{+}d){=}\underline{z}_d \,|\, T_1 \geq t_c{+}d, T_c{>}t_c{+}d, X(t_c){=}x)$$

The sequential generation of values of $\underline{Z}(t_c{+}d)$ is shown by

$$\Pr(\underline{Z}(t_c{+}d){=}\underline{z}_d \,|\, T_1 \geq t_c{+}d, T_c{>}t_c{+}d, X(t_c){=}x) = \tag{22}$$
$$\prod_{k=0}^{d-1} \Pr(Z(t_c{+}k{+}1){=}z_{k+1} \,|\, T_1 \geq t_c{+}k{+}1, T_c{>}t_c{+}k, X(t_c){=}x,$$
$$\underline{Z}(t_c{+}k) = \underline{z}_k) \, \frac{1 - r^0(t_c, k; x, \underline{z}_k)}{1 - r^0(t_c, k; x)} \, \rho(t_c, k+1; x, \underline{z}_{k+1})$$

In parallel to the definitions in (20), the hazard functions $r^0(t_c, k; x)$ and $r^0(t_c, k; x, \underline{z}_k)$ are defined by changing the condition $T_c = t_c$ into $T_c > t_c + k$. In contrast to (19), there is the additional factor

$$\rho(t_c, k; x, \underline{z}_k) := \frac{1 - h(t_c, k; x, \underline{z}_k)}{1 - h(t_c, k; x)} \tag{23}$$

which depends on the hazard functions for the occurrence of treatments in the control group, for $k > 0$ defined by

$$h(t_c, k; x) := \Pr(T_c = t_c{+}k \,|\, T_1 \geq t_c{+}k, T_c \geq t_c{+}k, X(t_c) = x) \tag{24}$$

$$h(t_c, k; x, \underline{z}_k) := \Pr(T_c = t_c{+}k \,|\, T_1 \geq t_c{+}k, T_c \geq t_c{+}k, X(t_c) = x, \underline{Z}(t_c{+}k) = \underline{z}_k)$$

This shows that one has to consider whether, and how, the occurrence of treatments in the control group might affect the distribution of variables which are causally relevant for outcome events and therefore could distort a comparison with the treatment group. One can distinguish three cases. First, the occurrence of treatments in the control group is independent of all variables which are causally relevant for $E_1$. In this case, $\rho(t_c, k; x, \underline{z}_k)$ depends neither on $x$ nor on $\underline{z}_k$, so that outcome probabilities in the treatment and the control group can immediately be compared.

Second, the occurrence of treatments in the control group could depend on values of $X(t_c)$ which are fixed at $t_c$, but does not depend on $Z(t)$ for $t > t_c$. In this case, $\rho(t_c, k; x, \underline{z}_k) = 1$, and treatments in the control group only affect the temporally remote distributions of $X(t_c)$.

Third, the occurrence of treatments in the control group depends on both $X(t_c)$ and $Z(t)$. In general, this should be expected when treatments are self-selected by primary agents. In order to define an unbiased conditional hazard function, one could use

$$\tilde{r}_j^0(t_c, d; x) := \tag{25}$$
$$\sum\nolimits_{\underline{z}_d} r_j^0(t_c, d; x, \underline{z}_d) \frac{\Pr(\underline{Z}(t_c{+}d) = \underline{z}_d \,|\, T_1 \geq t_c{+}d, T_c > t_c{+}d, X(t_c) = x)}{\rho(t_c, k{+}1; x, \underline{z}_{k+1})}$$

The basic idea is similar to reweighting procedures proposed for coping with time-dependent confounders which can influence both sequential treatments and outcomes (Robins et al., 2000; Hernán et al., 2000; Hernán et al., 2008, Gran et al., 2010). However, in the present context the interest concerns effects of a single treatment at $t_c$, and $Z(t)$ is considered as a variable mediating the effect of the treatment. The weights are therefore only intended to avoid selection effects which could result from later treatments in the control group.[6]

[6] Based on a potential outcomes framework, similar reweighting procedures have been discussed by Vikström (2014).

In the second and third case, the occurrence of treatments in the control group affects the distribution of $X(t_c)$ in temporal locations $t_c + d$. This can be seen as follows:

$$\Pr(X(t_c){=}x \,|\, T_1 \geq t_c{+}d, T_c > t_c{+}d) = \tag{26}$$
$$\Pr(X(t_c){=}x \,|\, T_1 \geq t_c, T_c > t_c) \prod_{k=0}^{d-1} \frac{1 - r^1(t_c, k; x)}{1 - r^1(t_c, k)} \lambda(t_c, k+1; x)$$

where

$$\lambda(t_c, k; x) := \frac{1 - h(t_c, k; x)}{1 - h(t_c, k)} \tag{27}$$

The first term on the right-hand side of (26) represents the initial distribution of $X(t_c)$ in the control group at $t_c$. The following factor takes into account the selection process which is due to the occurrence of $E_1$ events in the control group. Note again, that this factor depends on all competing risks. Finally, $\lambda(t_c, k+1; x)$ becomes relevant when the occurrence of treatments in the control group depends on values of $X(t_c)$. This has to be taken into account when being interested in versions of temporally remote CTEs which are averaged w.r.t. a distribution of $X(t_c)$ (e.g., the initial distribution of $X(t_c)$ in the treatment group). Instead of using $r_j^0(t_c, d)$, one could use a sequentially reweighted hazard function

$$\tilde{r}_j^0(t_c, d) = \sum\nolimits_x \tilde{r}_j^0(t_c, d; x) \frac{\Pr(X(t_c){=}x \,|\, T_1 \geq t_c{+}d, T_c > t_c{+}d)}{\lambda(t_c, k+1; x)} \tag{28}$$

To the extent that one has observed all relevant confounding covariates, the quantity $r_j^1(t_c, d) - \tilde{r}_j^0(t_c, d)$ can be interpreted as a version of a temporally remote CTE that is not distorted by treatments which occurred in the control group.

# 5 Discussion

The paper considers the question of how to define treatment and control groups in a dynamic setting where treatments can occur at any time (but only once). When treatments as well as outcomes are conceptualized as events occurring in temporal locations of a discrete time axis, it is natural 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 depends on 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 $t_c + d$ ($d = 0, 1, 2, \ldots$). As discussed in Section 3, this also helps to understand temporally extended effects as resulting from sequences of temporally local effects.

Combining this approach with the idea that effects should be estimated by comparing a treatment and a control group, 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 $t_c + d$.

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 eventually leads to treatment effects. This motivates the concept of 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 instantaneous CTE (if $d = 0$) can be decomposed into two parts: a selection effect and a balanced 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 (15). The remainder of the CTE can be viewed as a constructed balanced treatment effect.

This is consistent with the idea that randomized experiments can provide estimates of a balanced treatment effect. The argument for randomized experiments 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 decomposing a CTE into a selection and a treatment part.

A further difficulty is related to temporally remote CTEs. This requires to consider time-varying covariates which can change their values after the occurrence of the treatment and can be interpreted as mediating temporally remote effects of the treatment and any further conditions which are fixed at the time when the treatment occurs. One also has to take into account selection effects resulting from the occurrence of all possible outcome events (competing risks). Even if identical at the beginning, distributions of covariates in the treatment and the control group will become different due to outcome events whose occurrence depends on these variables. These selection effects should therefore be considered, not as distorting, but as an essential part of the treatment effect (even if balanced at the beginning). This entails that balancing temporally remote treatment effects does not lead to the construction of a 'pure' treatment effect.

Selection effects which could distort the treatment effect can result, however, from treatments occurring in the control group after its initial formation. This will be the case when such treatments depend on variables which are causally relevant for the outcome events. Treatments occurring in the control group will then change the distribution of these variables in the control group. As a possible approach to coping with this problem, the paper considers a sequential reweighting procedure aiming at the construction of a distribution of covariates corresponding to a hypothetical situation in which treatments in the control group do not occur.

**Acknowledgements.** I thank Ulrich Pötter and two anonymous referees for very helpful comments.

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