Assessing the effects of societal injury control interventions

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Abstract

Injuries have emerged as one of the biggest public health issues of the 21st century. Yet, the causal effects of injury control strategies are often questioned due to a lack of randomized experiments. In this thesis, a set of quasi-experimental methods are applied and discussed in the light of causal inference theory and the type of data commonly available in injury surveillance systems. I begin by defining the interrupted time series design as a special case of the regression-discontinuity design, and the method is applied to two empirical cases. The first is a ban on the sale and production of non-reduced ignition propensity (RIP) cigarettes, and the second is a tightening of the licensing rules for mopeds. A two-way fixed effects model is then applied to a case with time-varying starting dates, attempting to identify the causal effects of municipality-provided home help services for the elderly. Lastly, the effect of the Swedish bicycle helmet law is evaluated using the comparative interrupted time series design and synthetic control method. The results from the empirical studies suggest that the stricter licensing rules and the bicycle helmet law were effective in reducing injury rates, while the home help services and RIP cigarette interventions have had limited or no impact on safety as measured by fatalities and hospital admissions. I conclude that identification of the impact of injury control interventions is possible using low cost means. However, the ability to infer causality varies greatly by empirical case and method, which highlights the important role of causal inference theory in applied intervention research. While existing methods can be used with data from injury surveillance systems, additional improvements and development of new estimators specifically tailored for injury data will likely further enhance the ability to draw causal conclusions in natural settings. Implications for future research and recommendations for practice are also discussed.
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**Author contributions** The papers included in this doctoral thesis are the result of collaborative efforts between the authors. However, the majority of the work from study initiation, the formulation of research questions, data collection, statistical analysis, and
writing of the initial manuscripts were carried out by the main author. Co-supervisor Finn Nilson participated in the interpretation of the results, initiation of the papers and contributed to the discussion and conclusions. Main supervisor Ragnar Andersson participated in the planning process, interpretation of the results and writing of the final versions of the manuscripts. Co-author Johanna Gustavsson contributed to the discussion, methods and interpretation of the results in Study IV, and co-author Anders Jonsson was responsible for the data collection, participated in the interpretation of the analyses, and co-wrote the manuscript in Study I. Co-author Niklas Jakobsson contributed in the planning process, interpretation of results and writing of the manuscript for Study V.
1 Introduction

According to the most recent global burden of disease study, injuries are one of the top ten causes of death and disability in the world (Ärnlöv and Larsson 2014). During the last century, many prevention strategies have been introduced. For instance, most countries have by now adopted seat belt and drunk driving laws, and bicycle helmet legislation, fire-safe cigarette laws and other safety reforms are becoming more and more prevalent. But what actually happens when society intervenes on safety issues? Can we be certain that the right interventions are prioritized, or that they even affect injury outcomes the way that our policymakers and institutions hope? Are the large reductions in injury rates observed in most industrialized countries primarily caused by improved safety policy and other societal interventions, or are they simply artifacts of unrelated changes to the state of the world?

At the macro level, we know that injury rates tend to follow some temporal trend over time (see e.g. Oppe 1991, Moniruzzaman and Andersson 2008, Nilson 2014). While the causes of these changes are not fully known, it appears that the rates correlate with socioeconomic, demographic and technological development, similar to a ’societal learning curve’ (Oppe 1989). Disentangling the causal effects of specific interventions from other co-occurring changes is therefore required in order to correctly answer the questions posed above.

It is well known that the most credible method for effect identification is the randomized experiment, because random assignment of individuals to treatment and control groups will ensure that all other confounding variables are equal between the groups. However, a glance at the Cochrane systematic reviews of injury control interventions indicate that the number of randomized experiments conducted in injury control research is small (see e.g. Thompson et al. 1999, Macpherson and Spinks 2008, Kwan and Mapstone 2009, Owen et al. 2011, Mulvaney et al. 2015). Instead, quasi-
experimental and observational evidence is much more prevalent (Robertson 2007). There are several potential explanations for this lack of randomized trials, but a probable explanation is that the injury outcome is rare in small groups, which would require either very large samples or decades of follow-up time before obtaining sufficient statistical power to identify an effect. Such experiments are therefore very costly. Furthermore, many interesting interventions are introduced by societal bodies (such as governments), usually with no element of randomization, leaving quasi-experiments and observational studies as the only viable option (Heckman and Smith 1995).

Despite this, it appears that the quality of most intervention research in injury epidemiology is judged based on the quality of evidence-ladder used in most medical research fields, that is; non-randomized studies are crudely considered to be less valuable than randomized trials without much specification as to why this is the case. Furthermore, the study design selection criteria for systematic reviews used by Cochrane review groups appear very limited, as they list only two possible non-randomized designs that are of high enough quality to be included in a Cochrane systematic review: (i) controlled before-after studies (CBA) and (ii) the increasingly prevalent interrupted time series (ITS) design (Oxman 2011, Higgins and Green 2011).¹ Nowhere do the guidelines, which are highly adopted in many non-Cochrane systematic reviews of health care interventions as well (Rockers et al. 2012), make any clear mention of arguably stronger and often more valid quasi-experimental methods such as the difference-in-differences (Angrist and Pischke 2014) and regression-discontinuity designs (Lee and Lemieux 2009), or more generalized panel data methods such as the synthetic control method (Abadie et al. 2010) or fixed effects regression analysis (Heckman and Robb 1985). Not only can this exclusion ham-

¹The Cochrane Injuries Group do not specifically state which non-randomized studies to include. However, the study inclusion criteria from all recently published reviews of societal injury control interventions limit the search to CBA and ITS studies.
per the quality of the evidence of systematic reviews that include non-randomized evidence, but will likely also deter injury epidemiologists from using these methods, which have become standard designs for policy evaluation in many of the social sciences (Morgan and Winship 2014). In addition to being fairly easy to implement, these methods for causal inference in observational settings are also well suited for the type of secondary data that is available in abundance to epidemiologists looking to evaluate the impacts of societal interventions, in the many high-quality injury surveillance and reporting systems available in many parts of the world (Driscoll et al. 2004).

While there exists some papers and books detailing causal inference theory in relation to observational research in economics (see e.g. Angrist and Pischke 2014) and other social sciences, such as education research (Schlotter et al. 2011), there appears to be no current texts clearly detailing the underlying assumptions and applications of methods for causal inference in observational settings in specific relation to injury control research. In fact, most texts that stem from the medical sciences (e.g. in epidemiology and public health) appear to focus primarily on classic correlational designs when referring to observational research, and text books that specifically cover injury control research do not elaborate much on the analysis of quasi-experiments either (see e.g. McClure et al. 2004, Haas et al. 2007, Robertson 2007, Rivara et al. 2009). Some recent papers also argue that the evidence culture in public health should move from strictly focusing on randomization to also better include more rigorous quasi-experimental evidence (Victora et al. 2004), but papers that present and discuss alternative methods barely scratch the surface of the state-of-the-art designs that can be found in the evaluation literature (Bonell et al. 2011, Cousens et al. 2011, Pronyk et al. 2012). Many of these texts also lack a critical component in understanding why some methods are likely to produce more valid estimates than others, which is the explicit description and use of counterfactual theory.
1.1 Purpose and objectives

The general purpose of this thesis is to assess the strengths and limitations of a set of methods suitable for injury control research by first addressing them theoretically, and by subsequently applying them to empirical cases using data from injury surveillance systems. The specific objectives of the empirical studies included in the thesis are to identify the effects of four interventions on injury outcomes: (i) a ban on the sale and production of non-fire safe cigarettes on fatal fires (Study I), (ii) stricter licensing rules for mopeds on traffic injury accidents involving young moped drivers (Study II), (iii) municipal provision of a home help services for the elderly population on fall injury incidence (Study III), and (iv) the Swedish bicycle helmet law on bicycle-related head injuries and helmet use (Studies IV & V). The scope of the text is mainly limited to quantitative outcome evaluation and discussions of internal validity.

1.2 Organization of the thesis

The remainder of the thesis proceeds as follows: Section 2 introduces and discusses the theory of causation in relation to the type of data most commonly available in injury surveillance systems. Section 3 then uses this theory to detail and discuss the validity of a set of study designs with examples and results from the appended studies (Study I to V). Finally, Section 4 concludes the thesis with a discussion regarding perceptions of the value of observational evidence, suggestions for future research and implications for practice. The main text is stripped of details from each empirical study that do not relate directly to causal inference and methodology. Brief descriptions of the data used and a short introduction to each studied intervention will be given along with the presentation of the studies in Section 3, but for further details, including detailed presentation of the empirical results and discussions of external validity (generalizability), the reader is referred to the appended papers.
2 Theory

2.1 Causation

In the introduction to their book on causal reasoning, Shadish et al. (2002) note that causal reasoning, and therefore also the act of inferring causality, is inherently a qualitative feat. This notion may come as a surprise to some, as most of the scientific literature on causal inference heavily focuses on mathematical concepts and quantitative estimators. However, the act of understanding the underlying assumptions of each quantitative estimator, and expressing these clearly using mathematical notation, can help guide us when attempting to interpret and infer causality from numbers and figures (Angrist and Pischke 2014). The counterfactual model, which will be heavily drawn from in this chapter, forces researchers to consider potential outcomes under different conditions. In a sense, the starting point is to pose simple counterfactual questions; e.g. what if public policy intervention $I$ was not implemented in jurisdiction $A$ in year $B$? What would the state of $A$ have been with regards to outcome $Y$, which $I$ is supposed to affect, in absence of the intervention? As will be seen below, it is in the subsequent quest for the answer to these questions that many empirical challenges arise, and in order to systematically tackle these, an understanding of the requirements for a correct identification of a causal effect in observational settings is key (Pearl 2000).

The rise of the causal inference theory in the social sciences can be largely attributable to the need for more careful and systematic analysis and thinking regarding the causal mechanisms and pathways through which changes in one variable causes a change in another (Morgan and Winship 2014). While the groundwork for this way of thinking about causal relationships can be attributed to many scientists from many different disciplines over the course of the last century (e.g. Neyman 1923, Rubin 1974, Heckman and Robb 1985, Imbens and Angrist 1994), the computer scientist Judea Pearl (2000) is often credited as the first to provide a formal, and general,
theory of causation (Morgan and Winship 2014). The essence of their combined works were theoretical formalizations of the conditions under which an estimated effect can be considered defensibly causal, and under what formal assumptions the effect of an intervention has been empirically identified. Through these formalizations, it has been clearly shown and mathematically proven that causal effects can be estimated even in observational settings by sometimes fairly simple means, given that the right type of data exists or can be collected. Some of these methods will be discussed theoretically here in Section 2, and then subsequently applied in Section 3, but first I will begin by defining some important quantities and expressions.

2.2 Causal effect

Consider two parallel states: one where an intervention exists at some point in time and one where the intervention does not exist at the same point in time. Let \( Y(1) \) denote the outcome of interest in the state in which the intervention exists, and \( Y(0) \) denote the no-intervention state. Given that all else is equal (e.g. setting, population, other causal variables of the outcome of interest), the causal effect of an intervention can be defined as \( Y(1) - Y(0) \), i.e. the difference in outcome between the two potential states. This is the general quantity of interest when estimating the effects of interventions, and the most common definition found in the causal inference literature (Morgan and Winship 2014).

2.3 Injury control interventions

In most cases, a societal injury control intervention will attempt to modify some or several variables which change the risk of injury in some target population. Consider these to be intermediate variables in a causal relationship between the intervention under study to the causal effect we are attempting to quantify. To simplify notation, I will denote undefined interventions by \( D \) and the intermediate vari-
able(s) by $M$. The theoretical causal chain can then be written as $D \rightarrow M \rightarrow Y$.

### 2.4 Energy damage

The standard model in the epidemiological study of infectious diseases consists of three key components, (i) the host, defined as the infected person, animal or carrier of the disease; (ii) the environment, defined as the external environment in which the disease exists, the characteristics of which may influence the probability of contraction or severity of the disease, and (iii) the agent, defined as the microorganism that causes the disease; i.e. the only sufficient and necessary condition for its existence. These three components constitute the *epidemiological triad*, which in a sense is a causal model of how these three components interact given a certain disease (Bonita et al. 2006).

Gordon (1949) is often credited as first person to successfully apply the epidemiological triad to the injury problem. His correct identification of the causal agent of injury, along with the works of prominent researchers in biomechanics (e.g. DeHaven 1944, Stapp 1957), paved the way for some truly effective injury prevention strategies (Haddon Jr 1980, Robertson 2007). As they have convincingly proven, the *only* necessary, causal agents of injury are various forms of energy, which can either be mechanical, thermal, chemical, electrical or radiant.

Taking into account that the only necessary condition for an injury to occur is the transfer of energy between the environment and the host, we can then define any $D$ or $M$ as effective in eliminating or reducing the risk and or severity of injuries *only* if it by some direct or intermediate mechanism eliminates or reduces the probability of the transfer of energy to the human body, alternatively if it decreases the exposure-time and intensity of the energy transferred so that the human tissue can withstand it.\(^2\)

\(^2\)In the special cases of drowning, asphyxiation and hypothermia, where it is
2.5 Quantities of interest

With regards to the causal chain detailed above ($D \rightarrow M \rightarrow Y$), the only way to reduce the risk or severity of any injury in a population is to somehow modify variables that cause energy damage to human tissue. Thus, the arrow between $M \rightarrow Y$ must contain a causal effect on energy exposure in order for intervention $D$ to be effective.

**Proposition 1:** $M$ must cause a change in energy exposure in the target population in order to change injury risks.

An additional requirement for an effective injury control intervention is also that the arrow between $D \rightarrow M$ contains a causal effect of the intervention on the exposure to one or several intermediate variables that fulfill Proposition 1.

**Proposition 2:** $D$ must cause a change in population-level exposure to one or more intermediate variables that modify energy exposure.

If these two propositions hold, $D$ should have some effect on injury rates in the population. However, the size and direction of this effect are important considerations for decision-makers looking to compare different interventions, yet the above only captures the causal direction of events. Let us then consider in greater detail the three most important quantities of interest relating to these aspects.

2.5.1 Treatment effect on the treated

First, there is the quantity captured in the arrow between $M \rightarrow Y$, which is sometimes defined as the treatment effect on the treated (TT) (Imbens and Angrist 1994). As indicated above, this quantity will be directly proportional to the amount or intensity of energy damage prevented by $M$. For instance, if $M$ is a bicycle helmet, the arrow would capture the causal effect of bicycle helmets on $Y$, which is governed by the ability of the helmet to reduce the intensity of energy damage to the human body.
sity of the force that reaches human tissue upon impact. Let \( \tau \) denote this TT effect, which in the case of binary treatments can be defined mathematically as:

\[
\tau = E[Y|M = 1] - E[Y|M = 0]
\]  

(2.1)

where \( E[Y] \) is the expected average value of \( Y \) in the population given different states of exposure to the mediator \( M \).

### 2.5.2 Intervention effect on treatment exposure

If we still consider \( M \) to be binary, the quantity captured in the arrow between \( D \rightarrow Y \) can be defined as the causal effect of intervention \( D \) on population-level exposure to \( M \). Continuing the bicycle helmet example, this would amount to the effect of a bicycle helmet law on helmet use in the target population. Let \( \theta \) denote this quantity, which can be defined as (Angrist and Pischke 2014):

\[
\theta = E[M|D = 1] - E[M|D = 0]
\]  

(2.2)

that is, the population average difference in the probability of exposure to \( M \) given different intervention states. This is analogous to what is often referred to as compliance or adherence in the medical literature (McKay and Verhagen 2016). I choose to instead call this quantity treatment exposure, since this term is more general in its nature as it does not make any statements regarding the active choices of individual actors, only that they are by some mechanism exposed to its protective effects. Thus, the term also captures cases in which an individual is passively exposed to a treatment or intervention without their prior knowledge or consent, e.g. if they drive through a re-constructed intersection. In essence, however, they are mathematically equal.\(^3\)

\(^3\)The causes of treatment adherence or passive exposures to treatment are of course a more complex subject matter than a mathematical equality, but detailing these aspects are beyond the scope of this thesis (see e.g. Haddon Jr 1980, Reason 2000, Gielen and Sleet 2003, Lund and Aarø 2004, Robertson 2007, 2007,
2.5.3 Intervention effect on the outcome

The third quantity of interest is the causal effect of $D$ on $Y$, i.e. the impact of the intervention on injury rates, which we can call $\rho$. In the case of a single mediating variable, the intervention effect is simply given by the product of Equation (2.1) and (2.2) (Angrist and Pischke 2014):

$$\rho = \theta \times \tau$$  \hspace{1cm} (2.3)

and in the case of multiple mediating variables, the total impact can instead be defined as:

$$\rho' = \sum_{m=1}^{M} (\theta_m \times \tau_m)$$

where the full set of $M$ mediators associated with the intervention are summed according to the interventions causal effect on the population-level exposure to each $m$ along with the corresponding causal effect of each $m$ on $Y$. It is thus clear that the impact of any injury control intervention is simply the causal effect of $M$ on $Y$, reduced directly in proportion to the causal effect of $D$ on $M$. Readers familiar with medical terminology will likely recognize this as the intention-to-treat effect (Gupta 2011), while econometricians will probably see that this is analogous a reduced-form effect (Angrist and Pischke 2014). Following the same example as above, this quantity could be the impact of a bicycle helmet law on injury rates, which is dependent on (i) its effect on the proportion of helmet users, as well as (ii) the effect of bicycle helmets on the average impact force to the head in the event of a bicycle accident.

2.6 Identification

The main issue discussed in most texts relating to the empirical estimation of causal effects is the issue of confounding, which arises for more comprehensive discussions regarding theories of behavior change and structural modifications in relation to injury control interventions).
when there exists some other unobserved causal variables that correlate with the causal variable of interest and the outcome of interest, resulting in spurious empirical relationships if not handled effectively. As noted in the introduction, conducting a randomized experiment will ensure that all confounding variables will be equally distributed between an intervention group and a control group. Identifying the quantity $Y(1) - Y(0)$ can then be done simply by calculating the average difference in $Y$ between the two groups (Angrist and Pischke 2014). When randomization is impossible or inefficient, steps must be taken in order to credibly identify causal effects in observational data, and for causal interpretation of the derived effect estimates, we must be willing to make some additional assumptions (Morgan and Winship 2014).

The assumptions will differ depending on the identification strategy (i.e. study design, methods of statistical analysis, etc), the available data, and the nature of what causal mechanisms generate the outcome of interest and the intervention itself. However, a simple, and unifying generalization can be made, which is that quantity $Q$ will be identified if all back-door paths from the causal variable of interest to the outcome of interest that operate through confounding variables can be blocked (Pearl 2000). This fundamental identifying assumption is called the conditional independence assumption, which is upheld if the outcome $Y$ is independent of the causal variable $X$ within each stratum of the confounding variables $S$, so that adjusting for $S$ in observational data will result in an unbiased effect estimate. Again, this is achieved by design in randomized experiments because random treatment assignment will ensure that all $S$ are (on average) equal between the treatment and control groups. In observational settings, it may sound as if this amounts to matching or controlling for all variables that may confound the causal relationship of interest, which would be a dire feat considering that not all confounding variables may be known or observable. However, credible effect identification can be achieved in observational settings even in the presence of unmeasured confounders given a small set of additional assumptions, which will be elaborated on in the coming
sections, along with some methods that can be used to control for unobservables when simple conditioning (e.g. regression analysis) is not enough for effect identification. In essence, however, most (if not all) of these will implicitly contain the conditional independence assumption in that they state under which conditions this assumption will hold even if some important confounder is unobserved. It should be noted here that these assumptions are often untestable, and it is thus up to the researcher to decide and defend the plausibility of these given each empirical case (Morgan and Winship 2014).

In brief, Morgan and Winship (2014) summarize these ’special case’ strategies as (i) conditioning on a prior value of the outcome variable, (ii) using an unconfounded instrumental variable (IV) to estimate the effects of a confounded treatment and (iii) estimating an isolated and exhaustive mechanism (or set of mechanisms) that relates the causal variable to the outcome variable through causal mediation analysis, and (iv) estimating causal effects by repeated observations of the outcome in same unit (e.g. a state or country). This chapter will focus primarily on point number (iv), which includes discontinuous functions of time and time-series cross-sectional (panel) data estimators as examples of repeated measurement strategies. The reason for this is primarily because such data is readily available in most injury surveillance systems, while the type of data required for regular regression estimation, or estimation by points (i-iii) is much less prevalent.

2.7 The unobservable state in time series data

Since empirical evidence and theory suggests that injury rates will change over time as part of some societal learning process (Oppe 1989), comparing a country to itself before and after some legislation is enacted may yield biased effect estimates if the counterfactual state is not correctly modeled. Here, I will shortly consider and define the components of the unobservable state in time series data in order to facilitate discussions of causality in the empirical studies below. Without much loss of generality, I will assume that
the unobservable state of interest is a state in which an actual intervention unit (e.g. a country) is unexposed to the intervention of interest in the post-intervention period, $T_1$, which consists of all post-intervention time points. Let $t$ denote the time, counting sequentially from $1, 2, \ldots, T$, where $T$ is the total number of time points available in the series. Assuming the intervention of interest is implemented during some time point $t_x$ in the series, and that it is not abolished during the series, we can define and code a time-varying intervention variable ($D_t$) according to the pre- ($T_0$) and post-intervention ($T_1$) periods:

$$D_t = \begin{cases} 0 & \text{if } t < T_1 \\ 1 & \text{if } t \geq T_1 \end{cases} \quad (2.4)$$

Recall from previous sections that the intervention effect is defined as $\rho_{it} = Y_{it}(0) - Y_{it}(1)$. The observed outcome at time $t$ in intervention unit $i$ is thus:

$$Y_{it}(1) = Y_{it}(0) + \rho_{it}D_{it} \quad (2.5)$$

In order to find an unbiased estimate of $\rho_{it}$ using observational time series data, we must thus separate it from the unobservable counterfactual state $Y_{it}(0)$. This imposes some explicit requirements. According to Abadie et al. (2010), the components of this counterfactual can be given by the following factor model:

$$Y_{it}(0) = \kappa_t + \gamma_tS_i + \epsilon_{it} \quad (2.6)$$

where $\kappa_t$ is a vector of unknown factors contributing to a (sample) universal common secular trend; $S_i$ is an $(r \times 1)$ vector of either observed or unobserved confounding variables at the unit level; $\gamma_t$ is a $(1 \times r)$ vector of time-varying parameters with unknown factor loadings associated with these confounders, and $\epsilon_{it}$ are random errors at the unit level. The contents of this state has some important impli-

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4Their original factor model includes two separate terms for observed and unobserved confounders. However, since for the majority of this thesis, all confounders are considered unobserved, the model was simplified by merging these terms into one.
cations for estimation of intervention effects in time series data. For instance, if changes over time are simply driven by the common factors $\kappa_t$, a set of unaffected control units can be used to estimate the evolution of $Y_{it}(0)$ in the intervention unit (Angrist and Pischke 2014). However, this is a strong assumption considering that the trajectory of $Y$ may also be driven by an evolution of unobserved covariates that are *specific to the intervention unit*. Autonomous units that select to implement the intervention at a certain point in time will likely differ to some unknown extent from other units, which might introduce some bias of unknown magnitude into effect estimates based solely on adjustments for common factors $\kappa_t$. In other words, if concurrent events that affect $Y$ occur in the $T_1$ period only in the intervention unit, and/or the evolution of $\gamma_tS_i$ is specific to that same unit, an effect estimate based on such comparisons will by definition be biased to some extent. Letting $STB$ denote this general secular trends bias, the effect estimate will become $\hat{\rho} = \rho + STB$. Because this bias term is by definition impossible to observe since it is constructed by unobservable factors, we might conclude that the causal effect of the intervention is impossible to estimate, or if evidence so suggests, conclude that the bias is very small so that $\hat{\rho} \approx \rho$ given the chosen strategy to remove the influence of secular trends on $\hat{\rho}$. The ability to draw causal inferences then relies heavily on theoretical and empirical evidence that the remaining secular trend bias is small or non-existent, so that the estimated effect is at least defensibly causal and not entirely spurious (Morgan and Winship 2014). In the next section, I will consider at set of strategies which under some circumstances can remove this bias from the empirical estimation.

3 Empirical strategies and applications

In most injury surveillance systems there is high quality data on injury rates from different causes, often stratified by at least age and sex. However, the prevalence of observable covariates that can be used for valid regression-based adjustment for secular trends is not
very high. Furthermore, we may not even be aware of all potential variables that may cause this bias. Because of this, I will only consider methods that can deal with unobservable confounders.

3.1 Interrupted time series

I will begin by considering empirical cases in which no valid controls are available, which may occur if there are no regions or countries that are remotely comparable to the intervention unit, or no comparable outcome data is available due to differences in injury surveillance systems (which may be a particular issue for interventions at the national level). In these cases, the use of a single-case design, called interrupted time series (ITS), might be considered as an alternative to control-based methods (which will be discussed later in Section 3.3). However, the ITS design imposes some modeling challenges and strong underlying assumptions regarding the functional form of the effect, which will be shown below. To my knowledge, no theoretical literature exists that detail the identifying assumptions of the ITS design from a causal inference perspective. Thus, I will now attempt to define these criteria.

Here, it becomes important to note that the subscript \( t \) was added to \( \rho \) in Equation (2.5), indicating that the intervention effect may be time-varying in nature. There may thus be some \( D \times t \) interaction effect, the functional form of which may be known or unknown based on prior knowledge of the interventions causal effect on treatment exposure in the population over time. While this interaction is possible to model in a regression framework (Wagner et al. 2002), it may be hard to sufficiently be convinced that changes that occur at time points far from the start of the intervention period are part of the intervention effect and not artifacts of other concurrent changes, or changes in unobservable time-varying confounders (Glass 1997).

First, consider the simple case of no time-effect interaction, which Glass (1997) defines as an abrupt and permanent effect. This implies that by dropping the subscript \( t \) on \( \rho_t \), the effect is no longer
assumed to be time-dependent. This in turn suggests that the intervention reaches its full potential in terms of changing treatment exposure at the first time point in the \( T_1 \) period and remains constant for the remainder of the available time series. Assume also that there is some secular trend in the data so that \( \gamma_{STT0} \neq \gamma_{STT1} \) which will cause a simple comparison of pre- and post-intervention means to contain both \( \rho \) and \( STB \), and that these confounders are unobserved and thus cannot be controlled for using conventional regression methods. Suppose, however, that covariate assignment is as good as random at the most proximate values of \( t \) on either side of the \( T_1 \) cutoff, so that

\[
\rho_{it} = \lim_{t \to c^-} E[Y_{it} | T = t] - \lim_{t \to c^+} E[Y_{it} | T = t] \quad (3.1)
\]

where \( c \) is used to indicate the temporal intervention cut-off (Imbens and Lemieux 2008). If the intervention causes an abrupt change in treatment exposure at \( c \), the intervention effect can be viewed and estimated as a discontinuous function of time:

\[
Y_{it} = \alpha + f(t) + \rho D_{it} + \epsilon_{it} \quad (3.2)
\]

where \( f(t) \) captures the secular trend, and \( \rho \) is the intervention effect estimate at the \( T_1 \) cutoff. If this is true, the ITS design can be regarded as a special case of the regression-discontinuity (RD) design, for which theoretical literature covering the formal identifying assumptions exist. Borrowing from this literature, the assumption that such an abrupt effect ITS estimator identifies the causal effect of the intervention would be (Lee and Lemieux 2009):

**Assumption ITS.1** *Local randomization:* The distribution of time-varying confounders \( S_{it} \) becomes identical on either side of \( t = c \) as smaller and smaller neighborhoods of \( c \) are examined.

This has two important implications for interpretation. Firstly, if the intervention effect is homogeneous over time (i.e. constant), the parameter can be interpreted as the causal effect of the intervention and
extrapolated across all post-intervention time periods. Secondly, if the intervention effect is heterogeneous over time (i.e. changes as a function of time after the intervention), the parameter can only be interpreted as the local causal effect at the $T_1$ cutoff, similar to the local average treatment effect (LATE) discussed in Angrist et al. (1996) and Lee and Lemieux (2009). This suggests that the strategy is best suited if the intervention satisfies the condition that $\rho$ does not include any time-effect interactions, and if there are reasons to assume it does, only interpret the estimated parameter as the local effect at the cutoff (Hahn et al. 2001). This is a very important feature that, as discussed by Heckman and Urzua (2010) in their critique of LATE estimators, limits both the scope and external validity of the estimator to a small window in time. In response to this, however, Imbens (2010) argues that estimates of the LATE are better than biased parameter estimates, which illustrates the issue and trade-off between estimating valid but localized parameters versus less credible but sometimes more policy-relevant generalizations (or as in this case, extrapolation to time points far from the intervention cutoff).

Furthermore, it also implies that if there is no local effect at the cutoff, e.g. if the intervention affects treatment exposure very differently at time points far from the cutoff through gradual changes but has no instantaneous impact, the method may be unsuitable unless this gradual change can be modeled explicitly (Card et al. 2012). Hence, unless there is prior knowledge of delayed abrupt and possibility permanent intervention effects or the treatment exposure variable can be directly observed, the strategy may be unsuitable. To illustrate, a case in which the method would produce an unbiased intervention effect estimate is presented in Figure 1. It thus becomes obvious that the validity of the local randomization assumption is highly dependent on the true functional form of the effect over time, and that the single-case design will be most credible in cases where there is an abrupt effect on treatment exposure at some time $t$ along an observable time series of treatment exposure (Imbens and Lemieux 2008). If treatment exposure cannot be
observed directly, we must add an additional assumption relating to
the functional form of the effect over time:

**Assumption ITS.2** *Abrupt effects*: There exists some known point $c$ along a time series of the outcome $Y$ at which treatment exposure should change abruptly in the form of a known function.

This suggests that the credibility of the design will vary from case to case; if the effect can be assumed to be gradual or take some other complex trajectory over time, much stronger assumptions will be required. For instance, the local randomization assumption will change to an assumption of equal trajectories of confounders $S$ over the entire pre- and post-periods instead of a small window of time, and the functional form (abrupt effects) assumption will change to some arbitrary functional form assumption depending on the assumed form of the change in trend induced by the intervention, which may be hard to model convincingly unless it is observed directly or known with a great degree of certainty (Card et al. 2012).

Interestingly, these particular issues of identification do not appear to be discussed in much detail in papers that promote the use of the ITS design in applied intervention research (Wagner et al. 2002, Penfold and Zhang 2013, Kontopantelis et al. 2015).

For the strategy to be valid, there must also be no concurrent events or shocks that affect the outcome variable at the time point $c$ at which the abrupt discontinuous effect is assumed to exist (Biglan et al. 2000):

**Assumption ITS.3** *No concurrent events*: Only the studied intervention $D$ causes the observed discontinuity in $Y$ at $c$.

The necessity of this assumption to hold should speak for itself, as any violations would cause the estimated effect to contain both the effect of the intervention and the concurrent event(s).
Figure 1. Illustration of a case in which a single case interrupted time series design will yield an unbiased intervention effect estimate at the time of implementation (denoted by $c$). The critical condition is that the temporal intervention assignment (i) causes an abrupt change in the exposure to one (or more) treatment variables (ii) at the time point $c$, that in turn have a concurrent causal effect on the outcome of interest (iii).

3.1.1 Study I

In Study I, we applied the ITS method (Equation (3.2)) to study the abrupt effects of an intervention aimed to reduce the risk of fire-related mortality. In brief, the intervention involved an EU-wide ban on the production and sale of non-reduced ignition propensity (RIP) cigarettes in November 2011, which was hypothesized to greatly reduce the rate of cigarette-related fire deaths (The European Commission 2011). We studied monthly data on four different outcomes; (i) the number of residential fires, (ii) the number of fatal residential fires, (iii) the number of smoking-related fires and (iv) the number of fatal smoking-related fires. The time series are displayed visually in Figure 2. As seen in the graphs, seasonality was present in all cases, and seasonal terms were thus added to the models (see Study I for details). We used non-parametric smoothing splines for the trend parameter ($f(t)$) to avoid making any subjective input into the choice of functional form of the underlying trend. This was done to reduce the risk of non-linearity bias (see Figure 3 for an example of this). A last modeling consideration before settling on the final models, which is general for most time series analyses (see e.g. Box and Jenkins 1976, for details), was to check the residuals (i.e. the difference between observed values and model predictions) for autocorrelation, which can increase the risk of false positive results if present and left untreated. These modeling aspects are cov-
ered extensively elsewhere (see e.g. Box and Jenkins 1976, Wagner et al. 2002, Bonander 2015, or the appended studies), and will not be elaborated upon much further here.

The resulting estimates of $\rho$ at the cutoff in this case showed no statistically significant abrupt effects of the intervention in any of the outcome variables studied. Because no evidence of an effect was found, it seems unwarranted to discuss the validity of the local randomization and no concurrent events assumptions here. A more important consideration may be the validity of the abrupt effects assumption, which in this case would require an abrupt jump in the population-level exposure to RIP cigarettes at the time of intervention (recall Panel (ii) in Figure 1). While later evidence from 2013 indicates that the cigarettes sold on the Swedish market are RIP cigarettes (Larsson and Bergstrand 2015), the introduction may have been more gradual if producers and retailers gradually switched to RIP cigarettes prior to November 2011 as an anticipatory response to the intervention. However, if the treatment effect on the treated ($\tau$) was strong, any anticipatory effect should have been visible in the time series during the months prior to the intervention. Based on visual inspection of Figure 2, this does not appear to be the case.

After the study was published, Larsson and Bergstrand (2015) conducted a set of laboratory tests where they exposed lit RIP cigarettes to realistic environments (such as textiles, sofas) as opposed to the standard filter-paper test used by the EU. They found evidence that while the cigarettes pass the standard test almost flawlessly, they still smolder their full length when dropped in more realistic environments, which indicates that the effect of RIP cigarettes might be smaller than expected by the European Commission when they introduced the ban. As shown in Equation (2.3), if $\tau$ is small, the intervention effect ($\rho$) is likely to be even smaller. Two probable and possible reasons for the lack of evidence of an effect found here may therefore be that $\rho$ is too small to be found given the standard errors of the estimates, or that RIP cigarettes simply do not work as intended.
Figure 2. Time series graphs from January 1999 to December 2013, with estimated values from the GAMLSS/GARMA models, for residential fires, fatal residential fires, residential fires in which smoking is a known cause and fatal residential fires in which smoking is a known cause. The vertical line indicates when the ban on all non-reduced ignition propensity, or fire-safe, cigarettes was introduced.
While some questions still remain regarding the effects of intervention, the RIP cigarette case illustrates the necessity of carefully considering the shape of the intervention effect over time in any ITS analysis, which includes both the causal effect of the intervention on treatment exposure, as well as the causal effect of the treatment on the outcome of interest.

Figure 3. Fictional example of when a non-linear time trend has been mistaken for a structural break in a time series. The results from a segmented linear regression model in the graph to the left indicate a significant intervention effect, while the non-linear curve in the graph to the right indicate no discontinuity at the time of intervention (which is indicated by the vertical line).

3.1.2 Study II

The second empirical application of the ITS design involves a case in which we were able to identify an abrupt discontinuity in injury rates at specified points in time. Here, I will therefore instead focus more on discussing the abrupt effects, local randomization and no concurrent events assumptions than in the previous case.

The intervention under study involved a tightening of licensing rules for Class 1 mopeds in Sweden, which was implemented in October 2009. The new licensing rules requires prospective drivers to pass a new, longer course which now involves four hours of traffic-based driving practice alongside an 8-hour theory course.5

5Eight hours of theoretical education was also required before the intervention in order to receive a so-called provisional moped license.
This change greatly increased the price of obtaining a moped license (from roughly 2000 SEK to 5000 SEK), which according to vehicle registration data appears to have greatly reduced the number of newly registered mopeds in traffic and thus decreased moped use. The intervention therefore involves at least two different mediators ($M$ variables) which could potentially affect the risk of moped-related injuries: (i) decreased exposure in terms of moped use, (ii) improved traffic behavior via increased knowledge. An additional aspect relating to point (ii) is also that the new licenses may now be revoked in the event of a severe traffic violation, which may also affect risk-taking behavior.

An important aspect, which relates to the shape of the intervention effect over time, is that individuals are only required to pass the new course if they do not already possess a previous moped license issued prior to October 2009. Likewise, holders of any other type of driving license are not required to obtain an AM license to operate a Class 1 moped. Because of this, we might assume that the intervention will primarily affect those who were younger than 15 years$^6$ at the time of the intervention and thus had no other way of operating a moped legally than by passing the new course. This would likely produce a gradual effect over time if the outcome studied was the overall moped-related injury rate, as the population most affected by the intervention (the cohort born after August 1994) would grow larger over time, resulting in a gradual treatment exposure effect in the entire population of moped drivers. Such an evolution over time would violate the abrupt effects assumption detailed above. However, an abrupt effect should be identifiable in an age-stratified time series of moped injury events involving 15-year-old drivers, which is the population that should be most immediately affected by the intervention. By setting the temporal cut-off ($c$) to October 2009 in this stratified series and running an ITS analysis as detailed above, we found evidence of a 40% reduction in moped-related injuries involving this age group of drivers at this time point.

$^6$In Sweden, the minimum age requirement for driving a moped is 15 years.
To convince ourselves further that this was not due to any concurrent events, we also considered following the growth of the treatment cohort further by extending the analysis to age-stratified series of injury events involving 16- and 17-year-old moped drivers. We hypothesized that if the reduction found is a causal effect of the intervention, similar reductions should be found by lagging the intervention variable ($D$) to $c + 12$ months and $c + 24$ months in analysis of the stratified series of 16- and 17-year-olds, respectively. Furthermore, no abrupt effects should be found at the original $c$ among 16-year-olds, and no effects should be found at $c$ or $c + 12$ among 17-year-olds because they would not be affected at that time. The results returned virtually the same effects at the delayed time points, and as expected, we found no evidence of any discontinuities in $Y$ at the original $c$ in these groups. The results, along with model predictions of the counterfactual state, are presented in Figure 4.

Because the intervention estimates are almost identical, it should be safe to conclude that this is a causal effect of the intervention under study. However, there are still some potential threats to the validity of the estimate. The first is that we basically follow the same cohort over time, and cohort-specific effects therefore cannot be ruled out (the cohort may have, for instance, been less likely to drive a moped even without the intervention). Furthermore, the estimate might reflect a local effect at the time of the intervention and may not be generalizable to all time points after the intervention if the effect is time-varying. In addition to this, the many potential causal mediators generate a black box problem that disallows direct inference regarding which specific parts of the intervention were effective.\(^7\)

### 3.2 The fixed effects estimator

A main weakness of the abrupt effects ITS estimator discussed above is that it imposes some very strong assumptions and restrictions regarding the shape of the intervention effect over time, which limits

\(^7\)Nonetheless, we provide some hypotheses regarding this issue in the discussion of the appended paper (Study II).
Figure 4. Time series of the number of injury events involving teenage (15-17 years) Class 1 moped drivers reported by the Swedish police stratified by age of the driver, with estimates from the negative binomial GAMLSS models and predicted values (with 95% confidence intervals) had the intervention not taken place based on the reciprocal of the intervention effect (1/IRR) from the incidence model.
its use mainly to interventions where an abrupt effect can be expected. In this section, I will detail a strategy that can effectively relax this assumption. However, the strategy imposes some greater requirements on the available data and the details of the empirical case. To illustrate, consider a dataset that contains $I$ intervention units, where $I > 1$, all of which contain repeated measurements of $Y_{it}$ before and after the time of intervention $c$. If $c$ also varies by intervention unit, we can exploit the time series cross-sectional (panel) nature of the dataset to adjust for secular trends without making any explicit assumptions regarding the functional form of the effect (other than that the effect must manifest some time within the observable time series) (Allison 1994). Recall from Equation (2.6) that we are aiming to estimate an unobservable state that can be decomposed into (i) unobservable time-varying factors common to all $I$ in the sample, and (ii) unobservable time-varying factors that are specific to the intervention unit(s). To obtain plausible estimates of $\rho$ using panel data, we can then estimate the following fixed effects (FE) model:

$$Y_{it} = \alpha_i + \rho D_{it} + \gamma T_t + \epsilon_{it}$$

(3.3)

where $\alpha_i$ is a full set of dummy indicators for each unit (called fixed effects) and $\gamma T_t$ is a set of time point fixed effects. These parameters will capture two types of unobservable confounders: $\alpha_i$ will contain baseline differences (time invariant confounders), and $\gamma T_t$ will capture sample common time effects (see point (i) above). The remaining sources of bias in $\hat{\rho}$ can therefore only relate to unit-specific time-varying confounders, e.g. concurrent events or diverging trends (for more technical details, see e.g. Baltagi 2008, Wooldridge 2010).

Expressed more intuitively, the FE estimator captures the average of the within-unit difference in post- and pre-intervention means of $Y$ while removing all static differences between units. If both unit and time fixed effects are included, as in Equation (3.3), the model is called a two-way FE model (Baltagi 2008). This model removes, in addition to static differences, the effects of unobserved confounders that vary over time, but are common to all units in the
analyzed sample (e.g. national trends). Hence, the main identifying assumption of the two-way model becomes:

**Assumption FE.1** *Common trends:* The evolution of time-varying confounders $S_{it}$ is common to all units in the sample.

While this assumption may appear quite strong, some tests and robustness checks can be applied in order to probe the plausibility of the results, e.g. by adding unit-specific trends to the model (Morgan and Winship 2014) or by testing for effects at other points in time where no intervention effect should be present (see e.g. Clark et al. 2008).

As with the ITS estimator, we must also impose a no concurrent events assumption in order for the conditional independence assumption to hold, albeit in a slightly relaxed form:

**Assumption FE.2** *No concurrent events:* No confounding variables change systematically in conjunction with the interventions under study.

Violations of this assumptions could, for instance, include cases where the existence of intervention itself is systematically caused by a confounding variable or when simultaneous interventions are often introduced along with the intervention of interest. In practice, this would produce a parameter estimate that captures both the effect of the interventions and the effects of the confounding variables similar to standard regression adjustment where omitted variables bias is present (Halaby 2004).

Furthermore, it should be noted that in order for the model to be informative about the effects of the interventions under study, it is also implied that these interventions are homogeneous enough to be meaningfully expressed as an average of the unit-specific effects, as opposed to a distribution of effects (Heckman et al. 1997).
can for instance be present if contexts in which the interventions are implemented differ greatly, if the rate of compliance (i.e. treatment exposure) differs systematically between the studied settings, or if the interventions themselves are incomparable. While violations of this assumption are not directly associated with the identification of the average causal effect of the interventions, it may hamper policy-relevant interpretation of the resulting parameter estimate. The issue can for instance be present if contexts in which the interventions are implemented differ greatly, if the rate of compliance (i.e. treatment exposure) differs systematically between the studied settings, or if the interventions themselves are incomparable (Morgan and Winship 2014).8

3.2.1 Study III

In Study III, we applied the two-way FE estimator to study the effects of municipal provision of a home help service for the elderly. The intervention involves helping elderly people with complicated tasks around the home, such as changing light bulbs, hanging paintings and carrying things to the attic, with the purpose of reducing fall injury risks. The intervention has gradually spread across Sweden between 2000-2010, which allowed us to apply the model in Equation (3.4). By the year 2013, 191 (of 291) municipalities reported providing this service to their elderly inhabitants according to a survey by Bernfort et al. (2014). Using data from the same survey, we could identify the starting date (year and month) in 74 municipalities, which constituted our analysis sample.

Table 1 shows the intervention effect estimates under different model specifications. Column I displays the model in Equation (3.4), which adjust for unobservable municipality and year fixed effects. As indicated by the very small confidence interval, we can reject the presence of effects larger than ±5 percent on the incidence of hospital

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8If there is reason to suspect that different subgroups of interventions may differ on important variables that moderate the effect size, subgroup analysis of homogeneous sets of interventions can be performed instead.
Table 1. Results from negative binomial panel regression models, showing the estimated average effects of the home help services intervention on the incidence of fall-related hospitalizations among elderly people in Swedish communities under different model specifications.

<table>
<thead>
<tr>
<th>Model</th>
<th>I</th>
<th>II</th>
<th>III</th>
<th>IV</th>
<th>V</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intervention effect (%)</td>
<td>-.80</td>
<td>1.45</td>
<td>.058</td>
<td>2.16</td>
<td>-.02</td>
</tr>
<tr>
<td>Linear trend change (%)</td>
<td>-.06</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Municipality fixed effects</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Year fixed effects</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Seasonality</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Linear trend</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Linear trend change</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Municipality-specific linear trend</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Quadratic trend</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Cubic trend</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Municipality-specific quadratic trend</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Municipality-specific cubic trend</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
</tbody>
</table>

Notes: Incidence rate ratios (IRR) from the models were transformed into relative effects using the following formula: \((\text{IRR} - 1) \times 100\). Confidence intervals (in parenthesis) were calculated using cluster robust standard errors. N clusters (municipalities) = 74. N observations = 12432 (168 per cluster).
admissions due to falls in municipalities that offer the home help service. The model in Column II replaces the common year effects term with a municipality-specific trend, relaxing the assumption of common trends by allowing for a municipality-specific linear time trend. Nonetheless, the results remain virtually the same. However, by adding this linear trend, we are essentially estimating a pooled ITS model and thereby only testing for abrupt effects, which may not be present here since the implementation and actual use of the service within municipalities is likely to be gradual. Therefore, the model in Column III adds a linear trend change parameter to test for average differences in slope between the pre- and post-intervention periods in order to test for a linear gradual effect. Still, the results appear robust to model specification, and remain unchanged. The models in Columns IV-V probe the linear functional form assumption of the municipality-specific trends, but still produce similar results. Visual inspection of Figure 5, which plots the proportion of the sample that provides the service over time alongside the sample average fall-related injury rates, also shows no indication of a correlation between the two variables.

Figure 5. Monthly incidence of fall-related hospital admissions per 1000 population among elderly persons (65+ years) (gray line, left Y-axis) in a sample of Swedish municipalities that offer home help services for the elderly (n = 74). The proportion of communities (black line) providing the service at different time points are displayed on the right Y-axis.

Because of the relative strength of the fixed effects design with time-varying implementation (as discussed above), it appears safe to conclude that there is no average intervention effect on community-level fall-related injury rates. However, there are still some poten-
tial weaknesses that should be considered. First, there may be hidden effects on more specific types of fall-related injuries that cannot be captured using aggregated fall-injury admissions as the outcome variable. Furthermore, there may be some differences in effect size depending on municipality-specific variables, such as the level of usage by the elderly treatment population, the composition of the services provided (which vary slightly by municipality), or the occupation and skill of person(s) performing the service. In addition to this, there may also be other positive side effects on treated homes/individuals relating to quality of life and well-being, even if the actual effects on fall-related injury rates appear to be small.

3.3 Control-based estimators

The estimators discussed in the previous sections have only concerned scenarios in which all units in the sample are affected by the intervention. Of course, it is also highly relevant to consider cases where unaffected control series are available. In this section, I will discuss two different estimators while applying them to the same empirical case: the Swedish bicycle helmet law for children.

The topic of the effects of bicycle helmets and helmet legislation has an especially troubled past in the empirical literature (see Olivier et al. 2014, for an overview). In summary, the effects of helmets on the risk of head injury has been questioned by authors who claim that observational evidence from case-control studies is insufficient to draw causal conclusions due to the high risk of violation of the conditional independence assumption (Curnow 2005, Robinson 2007). Also, inconsistencies in the evidence from studies of bicycle helmet legislation has been used to further question the efficacy of helmets. For instance, Dennis et al. (2013) found no abrupt effects of provincial helmet laws in Canada on hospital admissions due to cycling-related head injuries using an ITS design, despite observed increases in helmet use (Dennis et al. 2010), and Robinson (2007) points to concurrent decreases in pedestrian head injuries and argues that effects observed in countries that enforce helmet use may be artifacts.
of concurrent events or secular trends. However, since helmets directly target the causal agent of injury, as supported by both theory and experimental biomechanical studies (e.g. Cripton et al. 2014), it is unclear how to interpret these findings. Are helmets really not effective in reducing the risk and severity of head injuries in the event of a bicycle accident, or can the consistencies be explained by poor identification strategies? To probe the credibility of these claims, the next sections will assess and analyze the effects of the Swedish helmet law using different empirical strategies, while discussing the relative validity of these designs. First, I will discuss and apply an extension of the ITS design that tests for differences in discontinuities between a case and a comparison series, similar to what has been done in previous studies of helmet laws (e.g. Walter et al. 2011, Dennis et al. 2013). After this, the case is analyzed using the synthetic control method (Abadie et al. 2010), which has some more desirable properties from a causal inference perspective.

3.3.1 Study IV

Recall that a central assumption of the abrupt effects ITS estimator applied in Study I and II is that no concurrent events other than the intervention itself causes any observed discontinuities in the time series of the outcome variable. A way to test this assumption is to study an unaffected comparison series that should also be affected by these concurrent events (Somers et al. 2013). In Study IV, we used adult cyclists as a comparison to the intervention population (children under the age of 15 years). To integrate this test in a regression framework, we used the following difference-in-discontinuities model:

$$Y_{it} = \alpha + f(t) + \lambda D_t + \beta I_i + (I_i \times f(t)) + \rho (I_i \times D_t) + \varepsilon_{it}, \quad (3.4)$$

in which a set of interaction terms, \((I_i \times f(t))\) and \((I_i \times D_t)\), and a dummy indicator for the intervention group, \(I_i\), were added to the standard abrupt effects ITS model detailed in Equation (3.2). Here, \(I_i\) and \((I_i \times f(t))\) allow the intercept and trend to vary between the
case and comparison series, and the parameter associated with the 
\( (I_i \times D_t) \) term, \( \rho \), tests for any differences in discontinuity at the 
start of the intervention. Applying this model to time series data on 
the proportion of cyclists admitted to hospitals within the child and 
adult groups,\(^9\) we found some inconsistent results, which if inter-
preted at face value could be in support of the anti-helmet claims 
cited above. Firstly, evidence of an abrupt effect was only present 
among male children (Figure 6), and this jump appears to mainly 
have been driven by an increase in non-head injury rates (see Study 
IV for details). A potential explanation for the gender difference 
could have been that the law only increased helmet use among boys, 
but Bonander (2015) showed that there are clear jumps of roughly 
15-20 percentage points in helmet use at the time of the interven-
tion among both female and male children using both observational 
and emergency department data. Since helmets directly target the 
causal agent of injury, and if the treatment effect on the treated is as 
large as the literature suggests (Attewell et al. 2001, Cripton et al. 
2014), I cannot find any plausible explanation for why the true ef-
fect of the law should be moderated by sex. A probable explanation 
for the inconsistent results could be that the comparative ITS de-
sign was unable to identify the effects of the increase in helmet use 
using hospital admissions data for two reasons: (i) the low event 
count produced large variability in the time series, which is espe-
cially prevalent among female children since they are less likely to 
be involved in bicycle accidents, and (ii) the proportion of head in-
jury admissions may be a crude proxy for the outcome of interest, 
which is arguably better measured as the average severity of head 
injuries (which we use in Study V below). Notwithstanding these 
inconsistencies in the empirical case, the test detailed in Equation 
(3.4) serves as a simple validity check for other concurrent events at 
the time of intervention in ITS studies. For this estimate to be valid,

\[^9\]The outcome variable is given by

\[ Y_{it} = \frac{n \text{ head injury patients}_{it}}{N \text{ patients}_{it}} \]

(3.5)
we make the same assumptions as in the standard ITS case, but with partial relaxation of the concurrent events assumption given that:

**Assumption CITS.1** *Capture of concurrent events:* The control series captures the same confounding effects of concurrent events that might otherwise bias the uncontrolled ITS estimate.

Furthermore, as with all control-based estimators, we must assume the standard stable unit treatment value assumption (Rubin 1978). That is, that the control unit(s) are not also affected by the intervention status of the intervention unit in the form of spillover or contamination effects.

### 3.3.2 Study V

In this section, I will detail some control-based estimators that pose less restrictive assumptions regarding the functional form of the effect (which is an issue in both the ITS and CITS designs). Consider a panel dataset similar to that in the home help services case, but that also contains a set of \( j \) unaffected control units as well as one or more intervention units. Formally, these units should be strictly exogenous in the sense that they are not affected by the intervention under study (as above). It is not necessary for these units to be equal on pre-intervention confounders or levels of the outcome variable. Rather, the trends in the outcome variable \( Y \) should be generated by the same unobservable causal processes that generate the trends in \( Y \) in the intervention unit(s). To capture and remove the secular trends bias from the the intervention effect estimate, we could then estimate the following difference-in-differences model:

\[
Y_{it} = \alpha + \gamma T_1 + \lambda D_{it} + \rho (D \times T_1)_{it} + \varepsilon_{it}
\]

where \( \alpha \) and \( \gamma \) are the pre- and post-intervention outcomes in the control units, respectively; \( \lambda \) is the pre-intervention outcome in the intervention unit(s) and \( \rho \) is the difference between the estimated
Figure 6. Average annual proportion of head injuries among cyclists admitted to hospitals in Sweden from January 1998 to December 2012 stratified by age group and sex with observed values and model predictions. The vertical line indicates when the bicycle helmet law for children came into effect.
counterfactual state constructed using the evolution of $Y$ in the controls and the observed state in the intervention unit(s) (Angrist and Pischke 2014). However, if no such set of controls exist that satisfy the common trends assumption, there may be some weighted combination of unaffected controls that does. Abadie et al. (2010) offer a solution to this problem using a matching strategy that selects this weighted combination of controls from a larger dataset and reduces it to the best fitting control series based on pre-intervention fit, assigning small or zero weight to controls with low predictive value so that these do not contribute (greatly) to the control unit. The resulting control unit, called a synthetic control, is then extrapolated into the post-intervention period, whereby the effect estimate is simply given by the difference between the weighted outcome in the synthetic control to the intervention series:

$$\rho_{1t} = Y_{1t} - \sum_{j=2}^{J+1} w_j Y_{jt}$$

(3.7)

where $j = 1$ denotes the intervention unit, $j = 2, \ldots, J + 1$ denotes the pool of potential controls. The goal of the matching procedure is to minimize the root mean squared prediction error (RMSPE) of the synthetic control unit with respect to the observed outcome in intervention unit in the time period before the intervention. The weights $w$ are selected based on the predictive value of the series in the pre-intervention period. As shown by Abadie et al. (2010), synthetic control-based estimators are likely to yield unbiased estimates if the synthetic control series can accurately predict the original series across a large number of pre-intervention time points. Given that such a combination of controls can be found in the empirical data, the method can produce estimates that are likely to be shielded against, but not immune to, violations of the common trends assumption. For instance, violations of the identifying assumptions of this estimator include still concurrent events specific to the intervention unit, or spillover effects into one or many of the

10 Formally, the outcome variable in the intervention unit must lie within the convex hull of the outcomes in the donor pool (Abadie et al. 2010).
units in the donor pool, especially if these receive large weights.

In Study V, we revisited the bicycle helmet law case using the synthetic control method (SCM). We began by studying the impact of the law on average head injury severity scores using emergency department data from the Swedish Traffic Accident Data Acquisition (STRADA), using all available cyclist and pedestrian 15-year age groups as potential controls, which allowed for much more flexibility in the construction of the control unit than in Study IV. As seen in Figure 7, Panel B, the synthetic control unit closely mimics the intervention group in the pre-intervention period, which is a sign that the model will produce valid effect estimates. The resulting counterfactual suggest an average post-intervention effect of -.061, or a relative reduction of -27%. Unlike the results from Study IV, we find no evidence of any sex-specific differences in effect size, which also appears more plausible.

**Figure 7.** Estimated effect of the Swedish bicycle helmet law on average head injury severity among child cyclists. The black line shows the intervention group, and dashed lines represent the sample average (in A), the synthetic control unit (in B), placebo studies (in C) and results from alternate models (in D).
Because the SCM does not allow for standard statistical inference (see Abadie et al. 2010; 2015, for details), we performed a large set of permutation tests and sensitivity analyses to probe the robustness of our results. First, we ran a set of placebo studies where we re-assigned the intervention to all other units in the donor pool, measuring the probability of finding an effect while taking the design effect into account. To do this, Abadie et al. (2010) suggests performing a graphical test that compares (3.7) from the actual intervention unit to the placebo effects from the donor pool. The placebo gaps in Figure 7, Panel C shows the difference between the observed average head injury severity scores in all groups and their synthetic counterparts, and it is clear that the effect in the intervention group (solid line) is large compared to the placebo effects. Following Abadie et al. (2015), we also performed a leave-one-out analysis in which the most influential controls were iteratively removed from the donor pool until only a single potential donor remained (Figure 7, Panel D). The median effect estimate from the alternate models was -.054, which corresponds well with the original estimate.

After also conducting a large set of placebo tests on other age groups and injury severity scores to other body parts, it appears safe to conclude that this is not a chance finding. Nonetheless, even though the synthetic control method has emerged as one of the most credible quasi-experimental designs given its ability to control for unobservable time-varying confounding and estimate intervention effects without prior assumptions of the shape of the effect over time, there are still some threats to the validity of the design that may be present despite the robustness of the results. For instance, there could still exist some post-intervention differences that are specific to the intervention unit, which therefore cannot be sufficiently captured by the temporal evolution of the synthetic control unit. For instance, there may still be some other concurrent interventions that

11To facilitate visual interpretation of the placebo gaps (Figure 7, Panel C) and leave-one-out plots (Figure 7, Panel D), the resulting effect estimate/control unit was assigned a transparency weight based on the rank of the models pre-intervention fit (darker = lower RMSPE).
apply only to the intervention group (such as school-based helmet use campaigns) that I am unaware of. Furthermore, since the counterfactual is entirely constructed based on controls, it is paramount that none of the controls that receive weights are also affected by the intervention through spillover effects. Since it is not entirely foreign to conceive that adult cyclists could also change their mind about wearing a helmet when a law for children is put in place, we also performed a sensitivity analysis in which we excluded other cyclist age groups from the donor pool, using only pedestrians as controls. Still, we find evidence of a similar intervention effect, which suggests that spillover bias is not a problem in this case.

**Connecting the quantities**

A final question that should be asked here is how plausible the size of the effect estimate derived above is. To answer this, we attempted to estimate the two other quantities of interest listed in Section 2.5, i.e. the effect of the law on helmet use and the treatment effect on the treated. First, we checked whether or not, and how much, the helmet law increased helmet use through the self-reported helmet use variable available in STRADA. Unfortunately, we could not use the SCM for this since helmet use was already much higher in the intervention group prior to the helmet law, which means that there is no weighted average of the donor pool that can approximate the outcome in the pre-period.\(^\text{12}\) We could, however, still attempt to estimate the effects using a difference-in-differences (DD) model (Equation (3.6)), which estimates the effect of the law under the assumption of common trends in absence of the law. Comparisons of the self-reported helmet use between the treated and control unit in four different age spans are presented in Figure 8. After estimating four different DD models based on these spans we found that, while the size of the effect estimate is somewhat dependent on the selection of age span, the results still consistently showed an increase in

\(^\text{12}\)As noted above, the intervention unit must lie within the convex hull of the outcomes in the donor pool in order to successfully identify a match (Abadie et al. 2010). I discuss possible solutions to this issue below.
helmet use of 13-22 percentage points in the post-period.

![Graph showing helmet use percentage over time for different age groups.]

**Figure 8.** Comparisons of self-reported helmet use by cyclists treated at emergency departments over time between the intervention group (solid line) and a comparison group (dashed line) allowing different age spans (data source: the Swedish Traffic Accident Data Acquisition).

After this, we tried to obtain some information regarding the final parameter of interest, i.e. the effect of helmet use on average head injury severity scores in the child cyclist population. Using ordinary least squares regression with adjustment for observable confounders, we found statistically significant effects on the severity of head injuries, but also on several unshielded body parts, which suggests that the helmet variable may be confounded by unobservable variables. To better deal with these unobservables, we instead generated a municipality-year pseudo panel dataset (Heckman and Robb 1985) in the hope of obtaining an unbiased estimate after adjustment for municipality fixed effects, time trends and variations in average severity scores to all other available body parts below the

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We included age dummies, accident type dummies and gender effects in the regression model.
neck area. Doing so, we found point estimates in the range of -.21 to -.25, which correspond to relative reductions of approximately 55 to 60%. We also ran a set of placebo models with severity scores to other body parts as outcome variables, finding statistically significant evidence of an effect only on head injury severity, which lends credibility to the empirical strategy. Furthermore, we found no evidence of an effect on pedestrian head injury scores in the treatment population.

Taking the uncertainty regarding the point estimates and their resulting standard errors into account, we then ran a set of Monte Carlo simulations to derive a plausible range for the reduced-form intervention effect (\(\rho\)) simply by using our estimates of the intervention effect on helmet use (\(\theta\)) and the treatment effect on the treated (\(\tau\)) (recall from Equation (2.3) that the intervention effect is given by \(\rho = \theta \times \tau\) if helmet use is the only mediating variable that causes the observed effect on head injury severity). The results from this procedure generated estimates that were very close to the effect estimate derived in the SCM analysis, which supports the plausibility of both the effect size and the presence of a causal effect of the law on helmet use, and a causal relationship between helmet use and the severity of children’s head injuries.

4 Concluding discussion

This thesis has attempted to assess and discuss the validity of a set of quasi-experimental methods for outcome evaluation of societal injury control interventions. While answering causal questions is often a complicated task, it has been shown that it is theoretically and practically possible to estimate the effects of interventions even if randomized controlled trials cannot be conducted. The empirical methods used in this thesis were derived by researchers a long time ago, and yet they still do not appear prevalent in injury control research. Perhaps this is because research practice in this field stems from the medical sciences, in which randomized controlled
trials are upheld as the only way to estimate causal effects, while all other non-experimental methods are lumped together into one or two 'less credible' categories of study designs (see e.g. Ho et al. 2008, Burns et al. 2011). As has been shown here, and by many other researchers in the social sciences, this is not the case (Morgan and Winship 2014). Perhaps the greatest barrier to achieving a wide-spread shift in focus from an 'RCT or nothing' mentality is that injury control research is often considered to be part of the medical sciences, and thus subject to the same principles and traditions as, for instance, drug trial research. While the etiology of injuries can be best explained by the natural and medical sciences, the interventions that aim to reduce injury risks are rarely clinical, nor are they introduced by researchers with the goal of valid effect identification; they are most often introduced by societal actors and aimed at non-clinical samples. It is thus becoming increasingly apparent that intervention research in injury epidemiology is largely a part of the social sciences, and should therefore adhere to the standards of credible effect identification that has been developed in this tradition over the course of the last few decades. This should not be interpreted as if the stringent quality-of-evidence ladder used in the medical sciences is not valuable to understanding the quality of research. Rather, in contrast, I argue that non-randomized designs and studies should be subject to more scrutiny than what the somewhat simplified levels of evidence imply, which highlights a greater need for the use of causal inference theories in such empirical investigations. Understanding and discussing the underlying assumptions of causal models in observational settings, instead of simply adding a disclaimer to the discussion stating that all analyses are possibly spurious while using vague (and arguably misleading) terminology like 'associated' or 'correlated' when referring to the effects of interventions, may in fact be critical to the enhancement of causal analysis in injury epidemiology.

While I hope that this thesis will contribute some to this enhancement, there is still much need for future research and development of methods for causal inference that are tailored for the type of data
prevalent in injury surveillance systems, which is often in the form of low, and thus volatile, counts. For instance, I suggest some research into synthetic control-type methods that (i) match on first derivatives (i.e. trends) instead of exact outcomes in order to generate a synthetic difference-in-differences estimator, and (ii) matching methods using filters to reduce noise in volatile series. Point (i) would allow researchers to estimate a more valid difference-in-differences estimate without subjective input into the selection of control units, creating a weighted control that will follow common trends to the intervention unit in the pre-intervention period. It would also allow for the use of the synthetic control method in cases where the intervention unit lies on the tail ends of the outcome distribution, such as the case with the helmet use outcome in Study V. Point (ii) would allow researchers to match on underlying trends while ignoring random noise in the outcome series, which may cause problems when trying to find a good match for the synthetic control unit. Since the random variation in injury data may be large, especially in smaller units of observation (such as municipalities or cities), this type of procedure would likely widen the applicability of the synthetic control method to smaller interventions which may otherwise be impossible to study using the current state of the method.

Similar to previous texts on the subject of causal inference in studies of public health interventions, a limitation to this work is that it fails to give a fully comprehensive review of research designs that can be used to deal with unobservable confounding in non-randomized studies. For instance, I do not detail the instrumental variables approach, which could be used to estimate the causal effects of both interventions and environmental risk factors under specific circumstances (see e.g. Angrist et al. 1996, Schlotter et al. 2011, Angrist and Pischke 2014, Morgan and Winship 2014, for details). I also do not make much mention of the regression-discontinuity design, which exploits abrupt discontinuities along continuous cross-sectional variables to find samples that are as good as randomly allocated to treatment (see e.g. Lee and Lemieux 2009, Schlotter et al.
Another limitation is that the thesis is limited in its scope to the estimation of quantifiable effects. It does not include any considerations into the black box problem, that is; why and how certain interventions work while others do not. While the effects of compliance are implicated in the intention-to-treat estimates (see Section 2.5), the moderating effects of context and population composition on effect modifiers are also very important considerations that have not been touched upon here. The latter aspect is highly relevant for understanding how, when and where to implement certain strategies, how to increase the probability of compliance, and whether or not the results can be generalized to other settings and contexts. While detailing this very complex subject was beyond the scope of this thesis, Nilsen (2006) provides a highly detailed review and analysis of these concepts in relation to community-based injury prevention programs.

Notwithstanding these limitations, the implications for practice of empirical estimation of the outcomes of injury control interventions are the following. If the data allows and a set of unaffected controls exists, researchers should consider control-based methods before turning to single-case designs. As shown in this thesis, the internal validity of the ITS design is highly reliant on some critical assumptions regarding the functional form of the effect, and it is therefore recommended that the method be used mainly if there is a strong reason to believe that there is an abrupt structural break in treatment exposure somewhere along the observable time series. If this assumption cannot be defended, methods that do not impose strong assumptions about the functional form of the intervention effect, such as panel data estimators or synthetic control methods should be considered instead. While the content of this thesis is mainly concerned with retrospective evaluation of interventions in which the

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14 The regression-discontinuity design is actually employed in Study III to serve as a second identification strategy, and the instrumental variables approach is used in Study V in an attempt to estimate the causal effects of helmets. See the appended studies for details.
researcher has no control of the implementation process, it should be noted that if the researcher is in control or otherwise involved, individual, cluster or stepped-wedged randomization is of course preferable to non-random allocation if possible and ethically defensible to do so (Murray et al. 2004, Hemming et al. 2015). In addition to this, as has been pointed out by e.g. Pawson and Tilley (1997) and Nilsen (2006), prospective evaluations should most preferably be conducted using a mix of quantitative and qualitative methods in order to gain a greater understanding of what hides within the black box of pure outcome evaluation.

On a note that has been touched upon by many others before me (e.g. Leamer 1983, Ziliak and McCloskey 2008, Morgan and Winship 2014), I believe that there is value in understanding that much of the uncertainty in drawing causal inferences about the effects of non-randomized interventions comes not from simple statistical inference in the form of p-values and standard errors, but in the form of uncertainty regarding the empirical strategies employed to the specific case at hand. For this reason, scientific journals should push authors to include robustness checks for model specification error, control group selection and other types of placebo falsification tests in order to facilitate transparency and causal interpretation in observational research. Because if the assumptions of the underlying model cannot easily be defended, how can we trust the effect estimates to be causal? How can readers be certain that the model that best fit the authors preconceived notions, or existing theories and paradigms, was not subjectively chosen because it shows what was expected, rather than what might actually be true? By actively attempting to falsify and challenge our own conclusions, by gaining a greater knowledge of the underlying assumptions of each method, and by developing new estimators that are tailored for injury data, we might soon be able to obtain the painfully scrutinizing skepticism that is needed to convincingly infer causality in ways that truly rival the randomized experiment, even in observational settings.
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Assessing the effects of societal injury control interventions

Injuries have emerged as one of the biggest public health issues of the 21st century. Yet, the causal effects of injury control strategies are rarely known due to a lack of randomized experiments. In this thesis, a set of quasi-experimental methods are discussed in the light of causal inference theory and the type of data commonly available in injury surveillance systems. I begin by defining the identifying assumptions of the interrupted time series design as a special case of the regression-discontinuity design, and the method is applied to two empirical cases. The first is a ban on the sale and production of non-fire safe cigarettes and the second is a tightening of the licensing rules for mopeds. A fixed effects panel regression analysis is then applied to a case with time-varying starting dates, attempting to identify the causal effects of municipality-provided home help services for the elderly. Lastly, the causal effect of the Swedish bicycle helmet law is evaluated using a comparative interrupted time series design and a synthetic control design. I conclude that credible identification of the impact of injury control interventions is possible using simple and cost-effective means. Implications for future research and recommendations for practice are discussed.