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Estimating causal effects in the presence of partial interference using multivariate Bayesian structural time series models

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Abstract

Researchers regularly use synthetic control methods for estimating causal effects when a subset of units receive a single persistent treatment, and the rest are unaffected by the change. In many applications, however, units not assigned to treatment are nevertheless impacted by the intervention because of cross-unit interactions. This paper extends the synthetic control methods to accommodate partial interference, allowing interactions within predefined groups, but not between them. Focusing on a class of causal estimands that capture the effect both on the treated and control units, we develop a multivariate Bayesian structural time series model for generating synthetic controls that would have occurred in the absence of an intervention enabling us to estimate our novel effects. In a simulation study, we explore our Bayesian procedure’s empirical properties and show that it achieves good frequentist coverage even when the model is misspecified. Our work is motivated by an analysis of a marketing campaign’s effectiveness by an Italian supermarket chain that permanently reduced the price of hundreds of store-brand products. We use our new methodology to make causal statements about the impact on sales of the affected store-brands and their direct competitors. Our proposed approach is implemented in the CausalMBSTS R package.

Keywords— Causal Inference, Partial Interference, Synthetic Controls, Bayesian Structural Time Series
1 Introduction

Synthetic control methods have recently gained a lot of popularity for obtaining estimates of causal effects from panel data with a single intervention (e.g., Abadie and Gardeazabal (2003); Abadie et al. (2010, 2015); Brodersen et al. (2015)). Unlike traditional difference-in-difference methods, synthetic controls provide more flexibility framework as they directly impute the unobserved outcome for treated time series by combining data from multiple control series that were not directly impacted by the treatment but are, nevertheless, correlated with the counterfactual outcome (O’Neill et al., 2016). For example, when studying the effectiveness of a supermarket chain’s policy to permanently reduce the price of store-branded cookies’ on daily sales, a control series could be the sales of bread. Bread sales are unlikely to be impacted by the price of cookies; instead, they capture daily and weekly trends, which are useful in modeling how the sales of cookies would have evolved had we not reduced their price. More broadly, these methods have been successfully applied to evaluate the effectiveness of policy changes in healthcare (Kreif et al., 2016; Papadogeorgou et al., 2018; Viviano and Bradic, 2019), economics (Billmeier and Nannicini, 2013; Abadie et al., 2015; Dube and Zipperer, 2015; Gobillon and Magnac, 2016; Ben-Michael et al., 2018), marketing and online advertising (Brodersen et al., 2015; Li, 2019), amongst others.

Typically, synthetic control methods assume that there is no interference between experimental units; that is, the assignment any unit receives has no bearing on the outcome of any other unit (Cox, 1958). However, there are many applications where this assumption is violated (e.g., Hudgens and Halloran (2008); Tchetgen and VanderWeele (2012), and Basse et al. (2019)). In the cookies example, the price reduction is likely to have a direct impact on competitor brands of cookies, and vice-versa. In this paper, we extend the synthetic control framework to the setting of where units interfere within predefined groups without interfering across these groups; this is known as partial interference (Sobel, 2006).

Partial interference occurs in many applications and has been extensively studied within the cross-sectional causal inference literature (e.g., Rosenbaum (2007), Hudgens and Halloran (2008), and Forastiere et al. (2020)). In the panel setting, like the one we are considering, partial interference has received relatively less attention, partly because of the added complications induced by the temporal component. In practice, authors often sidestep the issue by aggregate units that are likely to interfere with each other, generating a single treated time series that now satisfies the no-interference assumption (Bojinov et al., 2020a). One obvious downside of this approach is the inherent loss of information and a decreased ability to detect heterogeneous treatment effects.

To tackle this issue directly, we consider the extended potential outcomes that directly allow both spillovers across units and time (Robins, 1986; Robins et al., 1999; VanderWeele, 2010; Bojinov and Shephard, 2019; Bojinov et al., 2020b). We then define three new classes of causal effects that capture the impact of an intervention on both the unit that received it and the units within the same group. To perform inference, we derive the multivariate version of the popular Bayesian structural time-series model for causal inference introduced in Brodersen et al. (2015). Like its univariate counterpart, our model allows for a great deal of flexibility due to its ability to incorporate trends and seasonality effects. To perform inference, we provide a Markov chain Monte Carlo algorithm and describe how the resulting draws can be used to estimate our new causal effects. We then use a small simulation study to investigate the frequentist properties of our proposed approach and our ability to use posterior predictive checks to assess our findings’ robustness.

Our work is motivated by an analysis of the effectiveness of a policy change implemented by an Italian supermarket chain. In particular, on October 4th, 2018, the Florence branch of a supermarket chain store permanently lowered the price of 707 store brands in several product categories. The hope was that this intervention (or treatment) would expand its customer base and increase sales. For each product, the supermarket chain had identified a direct competitor brand, namely, a direct substitute differing primarily in the brand name. Traditional economic theory suggests that, if two goods are perfect substitutes, lowering the price of one
of them should impact the sales of the other (Nicholson and Snyder, 2012). In other words, the intervention applied to the store brand may affect the competitor brand’s outcome generating interference between the two units. To address this issue, we treat every store-competitor pair jointly, allowing us to model this group-specific interference directly. We also estimate the causal effect at different time horizons to determine whether the effect persists over time or quickly disappears. The results show that the policy change had a minor, short-term impact on the sales of store brands; interestingly, we do not detect significant effects on the competitor brands, suggesting that price may not be the only relevant sales driver in such a market. We also show that addressing the same problem by aggregating the units leads to information loss and misleading results.

Two papers consider our setup of partial-interference on panel or time series data; Cao and Dowd (2019) and Grossi et al. (2020). Cao and Dowd (2019), develops a model that requires that the impact of an intervention on one unit to the other is linear with an unknown parameter. Our paper imposes no such restriction, making it much more generally applicable. Grossi et al. (2020) formulation focuses on a context where only a single unit is intervened on, while the others are assigned to control. Since the treatment received by that unit may affect the outcomes of the other units, they rely on the partial interference assumption (Sobel, 2006) and identify different clusters such that the units belonging to different clusters do not interfere with each other. The inference is restricted to the group containing the treated unit, while the others form the “donor pool” used to construct synthetic controls by combining the donor outcomes. Our work presents a generalization of their specific context. Again, we study partial interference, but we allow for the existence of multiple treated units and different combinations of treatments within groups. By extending the univariate Bayesian structural time series model to the multivariate setting, we can also model the interference between units in the same cluster by explicitly modeling their dependence structure while allowing for a transparent way to deal with the surrounding uncertainty.

The paper is structured as follows. In Section 2, we present our causal framework, defining the treatment assignments, potential outcomes, causal effects, and our underlying assumptions. In Section 3, we introduce the multivariate Bayesian structural time series model and explain how to apply it to our setting. In Section 4, we detail a simulation study that tracks the performance of our approach. In Section 5, we present an empirical analysis of the effectiveness of an Italian supermarket’s new pricing policy. The final section, then presents our concluding remarks.

2 Causal framework

In this section, we outline our framework for estimating the causal effect of an intervention in a time series setting with partial interference among statistical units. Throughout, we illustrate key concepts and definitions by leveraging our analyses of the new price policy introduced by the Italian supermarket chain in all the stores located in the city of Florence. In our empirical example, the statistical units are grouped into pairs, and so we begin by introducing the notation for a bivariate outcome variable; we then extend the notation to the general group sizes. We conclude the section by deriving three classes of causal effects.

2.1 Notation

Throughout, we use a superscript $s$ to denote the store brand and $c$ the competitor brand. At time $t \in [1, T]$, for each pair $j \in \{1, \ldots, J\}$, let $W_{j,t}^{(s)} \in W$ be the treatment assignment for the store brand, $W_{j,t}^{(c)} \in W$ be treatment assignment for the competitor brand, and $W_{j,t} = (W_{j,t}^{(s)}, W_{j,t}^{(c)}) \in W^2$ the pair assignment. We mostly focus on the binary treatment case, where $W = \{0, 1\}$; following convention, we refer to “1” as treatment and “0” as control. In our application, each pair is assigned to one of four possible treatments: no permanent price reduction $W_{j,t} = (0, 0)$, both receive a permanent price reduction $W_{j,t} = (1, 1)$, store brand receive a permanent price
reduction only \( W_{j,t} = (1, 0) \), or competitor brand receive a permanent reduction only \( W_{j,t} = (0, 1) \). We then define the assignment path for each pair as the matrix \( W_{j,1:T} = (W_{j,1}, \ldots, W_{j,T})' \in W^{2 \times T} \), and the assignment panel that captures the assignments of all units throughout the study as \( W_{1:J,1:T} = (W'_{1,1:T}, \ldots, W'_{J,1:T}) \in W^{2J \times T} \). We will use this vector and matrix notation for other variables, but will sometimes drop the subscript if the dimensions are obvious from the context. Realizations of random variables will be denoted by their lower case; for example, \( w_{j,t} \) will denote a sample from \( W_{j,t} \).

In the panel set up, the pairs can change their assignment at any point in time, but to keep our notation less cumbersome, we only focus on the case when there is a single persistent policy change, as was the case in our empirical application.

**Assumption 1 (Single intervention)** We say pair \( j \) received a single intervention, if there exists a \( t^*_j \in [1, T] \) such that for all \( t \leq t^*_j \) we have \( W_{j,t} = (0, 0) \) and for all \( t, t' > t^*_j \) we have \( W_{j,t} = W_{j,t'} \). If all pairs receive a single intervention, then we say the study is single intervention panel study. For simplicity, we also assume that the intervention happen simultaneously, that is, \( t^*_j = t^*_j' = t^* \).

We maintain Assumption 1, which allows us to drop the \( t \) subscript from the treatment assignment so that \( W_j = (w^{(s)}_{j}, w^{(c)}_{j}) \in \{0, 1\}^2 \) for all \( t > t^* \) and \( W_j = (0, 0) \) for \( t \leq t^* \).

### 2.1.1 Potential outcomes

We now define the potential outcomes that describe what would be observed for a particular pair at a fixed point in time given assignment panel. Generally, the potential outcomes are a function of the full treatment panel (e.g., Bojinov et al. (2020b)); however, restricting our attention to non-anticipating potential outcomes\(^1\) and Assumption 1 somewhat simplify the setup.

Assuming the intervention occurred at time \( t^* + 1 \), for each pair \( j \in \{1, \ldots, J\} \) at time \( t \in [1, t^*] \), we observe an outcome \( Y_{j,t} = (Y^{(s)}_{j,t}, Y^{(c)}_{j,t}) \), where \( Y^{(s)}_{j,t} \) is the outcome of the store brand and \( Y^{(c)}_{j,t} \) is the outcome of the competitor brand. In our application, the outcome of interest is the average hourly sales for each product.

For \( t > t^* \), generally the outcomes depend on the treatment assignment matrix, \( Y_{j,t}(w_{1:j}) = (Y^{(s)}_{j,t}(w_{1:j}), Y^{(c)}_{j,t}(w_{1:j})) \). In our empirical application, the products within each pair are alike and only differ on their brand name and packaging; whereas, brands in different pairs differ on many characteristics (e.g., ingredients, flavor, or weight). Therefore, we assume that a price reduction of one brand will only directly impact its sales and its direct competitor’s sales. Essentially, in our empirical context this is an assumption about consumer behaviour; since each pair represents a different type of cookie (e.g., chocolate, whole grain, cream cookie), we are assuming that consumers’ choice of one pair or the other is not driven by price, but rather by individual preferences.\(^2\) To connect the general interference setting to our empirical application, we assume that there is no interference across pairs.

**Assumption 2 (Partial temporal no-interference)** For all \( j \in \{1, \ldots, J\} \), and \( t \in [t^* + 1, T] \) we assume that for any \( w_{1:j}, w'_{1:j} \in W^{2 \times J} \) such that \( w_{j} = w'_{j} \),

\[
Y_{j,t}(w_{1:j}) = Y_{j,t}(w'_{1:j}) .
\]

---

\(^1\)Following Bojinov and Shephard (2019), we say the potential outcomes are non-anticipating if the outcomes at time \( t \) are not impacted by future treatment assignments. That is, the potential outcomes only depend on past or current treatment assignments. In our empirical setting, for \( t < t^* \), this assumption would be violated if the knowledge of the upcoming price reduction changed present sales. For instance, consumers could have postponed their purchases leading to a decrease in sales before the intervention. We can, however, safely exclude this, as the supermarket chain did not advertise the upcoming discount in advance.

\(^2\)Also, every store brand has its own specific direct competitor, thus the possibility that the same good belongs to more than one pair is ruled out.
This allows us to simplify our notation and write \( Y_{j,t}(w_j) = Y_{j,t}(w_j) \).

In our application, there are four potential outcome paths that can occur, corresponding to the four different assignments. For each store-competitor pair, we can combine the post-treatment outcomes to define four potential outcome paths or potential outcome time series,

\[
Y_{j,t^*+1:T}^*(w_j) = (Y_{j,t^*+1:T}^{(s)}(w_j), Y_{j,t^*+1:T}^{(c)}(w_j)).
\]

Note that, even though we dropped the \( t \) script from the assignment, our setup implicitly assumes that the outcomes at time \( t > t^* \) are a function of the assignment path. This ensures that the potential outcomes at two different points in time correspond to two different treatment paths and are not directly comparable.

To connect the potential outcomes to the observed outcome, we assume that there is full compliance; that is, every pair receives the assigned treatment. In a causal inference setting for panel data, for each unit, there is only one observed potential outcome time series, whereas the others are unobserved. Generally, we will denote the observed treatment as \( w^{obs}_j \) which then leads to the observed outcome \( Y_{j,t^*+1:T} = Y_{j,t^*+1:T}(w^{obs}_j) \).

In our application, the observed outcome is \( Y_{j,t^*+1:T} = Y_{j,t^*+1:T}(1,0) \).

### 2.1.2 Covariates

For each pair and time point, we observe a vector of covariates \( X_{j,t} \in X \) that are not impacted by the intervention. If the covariates were impacted by the treatment, then we would consider them as secondary outcomes.

**Assumption 3 (Covariates-treatment independence)** Let \( X_{j,t} \) be a vector of covariates; for all \( t > t^* \) and for all assignments \( w_j, w'_j \in W^2 \) we assume that

\[
X_{j,t}(w_j) = X_{j,t}(w'_j) \quad \forall j \in \{1, \ldots, J\}.
\]

For the empirical application, our set of covariates for each pair includes: i) weekend and holiday dummies; ii) daily sales of products that are in categories that did not receive the price reduction; iii) the prices of both goods before the intervention. For all of these covariates, Assumption 3 is likely to be satisfied. We include the prior price as it is a good predictor of sales had there not been an intervention. Had we, instead, included the actual daily price after the reduction would have violated Assumption 3. To check if the control series \( X_{j,1:T} \) are genuinely unaffected by the intervention, we can test if the time series exhibits a change at the intervention time.

### 2.1.3 Assignment mechanism

We now define the class of assignment mechanism (i.e., conditional distributions of the assignment given the set of potential outcomes, covariates, and past assignments) that will allow us to identify and estimate the causal effects defined in the subsequent section. Our assumption has two parts. The first requires the assignment is individualistic; that is, the treatment of one pair has no bearing on another. The second requires the assignment is non-anticipating; that is, the assignment in a given period does not depend on future outcomes or covariates.

**Assumption 4 (Non-anticipating individualistic treatment)** The assignment mechanism is independent across pairs, at time \( t^* + 1 \) for the \( j \)-th pair depends solely on its past outcomes and past covariates.
\[
\Pr(W_{1:J,t^*+1} = w_{1:J,t^*+1}|W_{1:J,1:T}, Y_{1:J,1:T}(w_{1:J,1:T}), X_{1:J,1:T}) = \prod_{j=1}^J \Pr(W_{j,t^*+1} = w_{j,t^*+1}|Y_{j,1:T}(w_{j}), X_{j,1:T}).
\]

The non-anticipating treatment assumption is the extension of the unconfounded assignment mechanism in a cross-sectional setting (Imbens and Rubin, 2015; Bojinov et al., 2020b). Assumption 4 is essential in ensuring that, conditional on past outcomes and covariates, any differences in the outcomes are attributable to the intervention.

### 2.1.4 Multivariate case

Our framework easily generalizes to groups of size \(d_j > 2\). For \(j \in \{1, \ldots, J\}\), let \(W_j^i \in W\) be the treatment status of the \(i\)th unit inside the \(j\)th group, and let \(W_j = (W_j^{(1)}, \ldots, W_j^{(d_j)}) \in W^{d_j}\) be the treatment status of the \(j\)-th group. Again, Assumption 1, allowed us to drop the subscript for time. We then define the outcome to be a \(d_j\)-variate vector, \(Y_t = (Y_t^{(1)}, \ldots, Y_t^{(d_j)})\), for \(t \leq t^*\). Assuming that there is only partial interference, Assumption 2, the potential outcomes for \(t > t^*\) for any \(w_j \in \{0, 1\}^{d_j}\) are

\[
Y_{j,t}(w_j) = (Y_t^{(1)}(w_j), \ldots, Y_t^{(d_j)}(w_j)).
\]

Throughout the rest of the paper we will use the more compact notation to denote the potential outcome time series as \(Y_{j,t^*+1:T}(w_j)\). All other assumptions and definitions easily extend to the multivariate case.

### 2.2 Causal estimands

In a panel setting, the number of causal estimands increases substantially, as any contrast of potential outcomes has a causal interpretation. In this section, we develop three classes of causal effects; for each, we can define a contemporaneous effect (i.e., an instantaneous effect at each time point after the intervention), a cumulative effect (i.e., a partial sum of the contemporaneous effect), and an average temporal effect (i.e., a normalization of the cumulative effect). Our primary objective is to obtain an estimate for each group. To simplify our notation, we will drop the subscript \(j\) that identifies the group and focus on analyzing each multivariate time series separately; \(d\) will then indicate the group size. Even though our goal is to estimate the heterogeneous effect on each pair, the definitions below are given for a general multivariate case where units define groups of size \(d > 2\).

Since we are following the potential outcome approach to causal inference, we restrict \(t > t^*\) so that the causal effects are defined as comparisons between two potential outcomes.

**Definition 1** For \(w, \tilde{w} \in W^d\), the general causal effect of an assignment \(w\) compared to an alternative assignment \(\tilde{w}\) is

\[
\tau_t(w, \tilde{w}) = (\tau_t^{(1)}(w, \tilde{w}), \ldots, \tau_t^{(d)}(w, \tilde{w})) = (Y_t^{(1)}(w) - Y_t^{(1)}(\tilde{w}), \ldots, Y_t^{(d)}(w) - Y_t^{(d)}(\tilde{w})) = (Y_t(w) - Y_t(\tilde{w}))
\]

The cumulative general causal effect at time point \(t' > t^*\) is

\[
\Delta_{t'}(w, \tilde{w}) = \sum_{t=t^*}^{t'} \tau_t(w, \tilde{w})
\]
The temporal average general causal effect at time point $t'$ is

$$
\bar{\tau}_t(w, \bar{w}) = \frac{1}{t' - t^*} \sum_{t=t^*+1}^{t'} \tau_t(w, \bar{w}) = \frac{1}{t' - t^*} \Delta_t(w, \bar{w})
$$

(3)

In a general $d$-variate case, the total number of general causal effects that we can estimate is $C_{2^d}^d$.

**Example 1** In our empirical application we have a bivariate outcome, with $d = 2$, and $W = \{(0, 0), (0, 1), (1, 1), (1, 0)\}$. Then, $\tau_t((1, 0), (0, 0)) = Y_t(1, 0) - Y_t(0, 0)$ is the change in units sold when only the store brand gets a discount compared to the alternative scenario where none of them receive a discount.

We can combine the general causal effects to define the marginal causal effect that captures the impact of changing a single unit within a group across all possible treatment combinations the group could have received. For simplicity, from now on we assume that $W = \{0, 1\}$. Generalizing to multiple treatments is easy, but makes the notation more cumbersome.

**Definition 2** Let $A \subset \{0, 1\}^d$ be the subset of all treatment paths $w$ such that $w^{(i)} = 1$ and $B_i \subset \{0, 1\}^d$ be the subset of all treatment paths $\bar{w}$ such that $w^{(i)} = 0$. The marginal causal effect on the $i$th series is the sum of the $i$th elements of $\tau_t(w, \bar{w})$ computed across all the possible realizations in $A_i \times B_i$,

$$
\tau_t(i) = \sum_{(w, \bar{w}) \in A \times B} \tau_t^{(i)}(w, \bar{w})
$$

(4)

The cumulative marginal causal effect at time point $t' > t^*$ is

$$
\Delta_t(i) = \sum_{t=t^*+1}^{t'} \tau_t(i)
$$

(5)

The temporal average marginal causal effect at time point $t'$ is

$$
\bar{\tau}_t(i) = \frac{1}{t' - t^*} \sum_{t=t^*+1}^{t'} \tau_t(i) = \frac{1}{t' - t^*} \Delta_t(i)
$$

(6)

Now, let $N_{A_i \times B_i}$ denote the total number of possible assignments in $A_i \times B_i$; the mean marginal causal effect can be defined as,

$$
\tau_t(i, N_{A_i \times B_i}) = \frac{1}{N_{A_i \times B_i}} \sum_{(w, \bar{w}) \in A_i \times B_i} \tau_t^{(i)}(w, \bar{w})
$$

(7)

The cumulative and temporal average mean marginal effects can be then derived as in equations (5) and (6).

The marginal causal effect captures the impact of assigning the $i$th unit to treatment, averaged over all possible interventions that could have been applied to the other units. Thus, the marginal effect extends to the time series setting the average distributional shift effect in Sävje et al. (2020), with the difference that it is averaged across units whereas the marginal effect is individual-specific and, in its temporal average version, averaged across times. We could make this effect slightly more general by introducing non-stochastic weights in the summation to up-weight or down-weight particular treatment combinations. However, this makes the notation somewhat more cumbersome without adding new insights.

**Example 2** Suppose that we are interested in estimating the marginal effect of the active treatment on the store brand, then $A = \{(1, 0), (1, 1)\}$, $B = \{(0, 0), (0, 1)\}$, and $A \times B = \{(1, 0) \times (0, 0); (1, 0) \times (0, 1); (1, 1) \times (0, 0); (1, 1) \times (0, 1)\}$.
Furthermore, \( \tau_t(w, \tilde{w}) = (\tau^{(s)}_t(w, \tilde{w}), \tau^{(c)}_t(w, \tilde{w})) \) and hence,
\[
\tau_t(s) = \tau^{(s)}_t((1,0),(0,0)) + \tau^{(c)}_t((1,0),(0,1)) + \tau^{(c)}_t((1,1),(0,0)) + \tau^{(s)}_t((1,1),(0,1)).
\]
Finally, the mean marginal effect of the active treatment on the store brand is \( \tau_t(4, 4) = 1/4 \cdot \tau_t(s) \).

A special case of the general causal effect is the conditional causal effect that fixes the treatments for all units within the group except for the \( i \)th unit.

**Definition 3** For \( w \in W^{d-1} \), the **conditional causal effect** is the effect of assigning the \( i \)th series to treatment as opposed to control, fixing the treatments of the other series to equal \( w \)
\[
\tau^i_t(i, w) = Y_t((w_1, \ldots, w_{i-1}, 1, w_i, \ldots, w_{d-1})) - Y_t((w_1, \ldots, w_{i-1}, 0, w_i, \ldots, w_{d-1}))
\]
Similarly to the marginal and mean marginal causal effects, we can define the cumulative and temporal average conditional causal effect at time point \( t'<t^* \).

The conditional effect can also be seen as the generalization to the time-series setting of the assignment-conditional unit-level treatment effect in Sævje et al. (2020).

**Example 3** The general effect defined in Example 1 is already a conditional effect, since it measures the impact of the permanent reduction on the store brand given that the competitor is always assigned to control. However, we may also be interested in the conditional effect of the permanent price reduction on the store brand when the competitor brand is discounted as well, that is, \( w^1 = (1,1), \tilde{w}^1 = (0,1) \) and \( \tau^i_t(s, (1,1)) = Y_t(1,1) - Y_t(0,1) \).

## 3 Multivariate Bayesian Structural Time Series

We now outline our approach for estimation and inference of the causal effects defined in Section 2.2. We begin by deriving the multivariate Bayesian structural time series models (MBSTS), which are the multivariate extensions of the models used by Brodersen et al. (2015) and Papadogeorgou et al. (2018). Like their univariate versions, MBSTS models are flexible and allow for a transparent way to deal with uncertainty. Flexibility comes from our ability to add sub-components (e.g., trend, seasonality, and cycle) that encapsulate the characteristics of a data set. Uncertainty is quantified through the posterior distribution, which we derive and provide a sampling algorithm.

Estimation in this approach has two steps: first, we estimate an MBSTS model for each pair in the period up to the intervention, \( t \in [1, t^*] \); then, we estimate the target causal effects by forecasting the unobserved potential outcomes in the period following the intervention, \( t \in [t^* + 1, T] \). This section mirrors the two steps by first describing the model priors and posterior inference followed by the forecast and inference step.

Throughout this section, we employ random matrices to simplify the notation and subsequent posterior inference by allowing us to avoid matrix vectorization. Recalling the notation introduced by Dawid (1981), let \( Z \) be an \((n \times d)\) matrix with standard normal entries, then \( Z \) follows a **standard matrix Normal distribution**, written \( Z \sim \mathcal{N}(I_n, I_d) \), where \( I_n \) and \( I_d \) are \((n \times n)\) and \((d \times d)\) identity matrices. Thus, throughout the rest of paper, \( Y \sim \mathcal{N}(\mathbf{M}, \mathbf{A}, \mathbf{S}) \) indicates that \( Y \) follows a matrix normal distribution with mean \( \mathbf{M} \), row variance-covariance matrix \( \mathbf{A} \) and column variance-covariance matrix \( \mathbf{S} \). Finally, a \( d \)-dimensional vector \((n = 1)\) following a multivariate standard Normal distribution will be indicated as \( Z \sim \mathcal{N}_d(0, I_d) \) and \( IW(\nu, S) \) will denote an Inverse-Wishart distribution with \( \nu \) degrees of freedom and scale matrix \( S \).

### 3.1 The model

Two equations define the MBSTS model. The first one is the “observation equation” that links the observed data \( Y_t \) to the state vector \( \alpha_t \) that models the different components in the data (such as, trend, seasonal, or
The multivariate local level model is characterized by a trend component evolving according to a simple random walk, no seasonal is present, and both the disturbance terms are assumed to be Normally distributed.

$$\mathbf{Y}_t = \mathbf{Z}_t \alpha_t + \mathbf{X}_t \beta + \mathbf{e}_t, \quad \mathbf{e}_t \sim N_d(0, H_t \Sigma)$$

$$\alpha_{t+1} = T_t \alpha_t + \mathbf{R}_t \eta_t, \quad \eta_t \sim N(0, \mathbf{C}_t), \quad \alpha_1 \sim N(a_1, P_1, \Sigma) \quad (9)$$

Where, for all $t \leq t^*$, $\alpha_t$ is matrix of the $m$ states of the $d$ different time series and $\alpha_1$ is the starting value; $\mathbf{Z}_t$ is a vector selecting the states entering the observation equation; $\mathbf{X}_t$ is a vector of regressors; $\beta$ is matrix of regression coefficients; and $\mathbf{e}_t$ is a vector of observation errors. For the state equation, $\eta_t$ is a matrix of the $r$ state errors (if all states have an error term, then $r = m$); $T_t$ is a matrix defining the equation of the states components (e.g. in a simple local level model $T_t = 1$); and $\mathbf{R}_t$ is a matrix selecting the rows of the state equation with non-zero error terms. Under our specification, we assume that $\mathbf{e}_t$ and $\eta_t$ are mutually independent and independent of $\alpha_1$. We denote variance-covariance matrix of the dependencies between the time series by

$$\Sigma = \begin{bmatrix} \sigma_1^2 & \cdots & \sigma_1 \sigma_d \\ \sigma_2 \sigma_1 & \cdots & \sigma_2 \sigma_d \\ \vdots & \ddots & \vdots \\ \sigma_d \sigma_1 & \cdots & \sigma_d^2 \end{bmatrix}.$$  

$H_t$ is the variance of the observation error at time $t$; to simplify notation we can also define $\Sigma_e = H_t \Sigma$. Finally, $\mathbf{C}_t$ is an $(r \times r)$ matrix of dependencies between the states disturbances and since we are assuming that different states are independent, $\mathbf{C}_t$ is a diagonal matrix. Indeed, we can also write $\eta_t \sim N_d(0, \mathbf{Q}_t)$ where $\mathbf{Q}_t$ is the Kronecker product of $\mathbf{C}_t$ and $\Sigma$, denoted by $\mathbf{Q}_t = \mathbf{C}_t \otimes \Sigma$. Furthermore, different values in the diagonal elements of $\mathbf{C}_t$ allows each state disturbance to have its own $(d \times d)$ variance-covariance matrix $\Sigma_e$.  

In short,

$$\mathbf{Q} = \mathbf{C}_t \otimes \Sigma_e = \begin{bmatrix} c_1 \Sigma & 0 & \cdots & 0 \\ 0 & c_2 \Sigma & \cdots & 0 \\ \vdots & \vdots & \ddots & \vdots \\ 0 & 0 & \cdots & c_d \Sigma \end{bmatrix} = \begin{bmatrix} \Sigma_1 & 0 & \cdots & 0 \\ 0 & \Sigma_2 & \cdots & 0 \\ \vdots & \vdots & \ddots & \vdots \\ 0 & 0 & \cdots & \Sigma_r \end{bmatrix}.$$  

To build intuition for the different components of the MBSTS model, we find it is useful to consider an example of a simple local level model.

**Example 4** The multivariate local level model is characterized by a trend component evolving according to a simple random walk, no seasonal is present, and both the disturbance terms are assumed to be Normally distributed.

1Notice that this parametrization assumes the same set of regressors for each time series but still ensures that the coefficients are different across the $d$ time series.

2The notation $H_t \Sigma$ and $c_i \Sigma$ allows to understand that the dependence structure between the $d$ series is the same for both $\mathbf{e}_t$ and $\eta_t$; furthermore, when $H_t$ and $\mathbf{C}_t$ are known, the posterior distribution of $\alpha_t$ is available in closed form (West and Harrison, 2006). Instead, we employ a simulation smoothing algorithm to sample from the posterior of the states and in Section 3.1.2 we derive posterior distributions for $\Sigma_e$ and $\Sigma_e$ in the general case of unknown $H_t$ and $\mathbf{C}_t$.  

---

9
Figure 1: The plot shows 200 observations (e.g., number of units sold of a specific item) sampled from a multivariate local level model with $d = 2$. 

\[ Y_t = \mu_t + \varepsilon_t \quad \varepsilon_t \sim N_d(0, H_t \Sigma) \]

\[ \mu_{t+1} = \mu_t + \eta_{t, \mu} \quad \eta_{t, \mu} \sim N_d(0, c_1 \Sigma) \]

We can recover the general formulation outlined in (9) by setting $\alpha_t = \mu_t$ and $Z_t = T_t = R_t = 1$. Figure 1, provides a graphical representation of how a sample from this model would look like when $d = 2$.

3.1.1 Prior elicitation

The unknown parameters of Model (9) are the variance-covariance matrices of the error terms and the matrix of regression coefficients $\beta$. Since we assume that both the observation and state errors are normally distributed, for their variance-covariance matrices, we choose the conjugate Inverse-Wishart distributions. Generally, the MBSTS model can handle dynamic covariate coefficients. However, in our empirical application we believe that the relationship between covariate and the outcome is stable over time, and so we use a matrix normal prior, $\beta \sim N(b_0, H, \Sigma_\varepsilon)$. In many applications, we have a large pool of possible controls but believe that only a small subset is useful. We can incorporate such a sparsity assumption by setting $b_0 = 0$ and introducing a selection vector $\varrho = (\varrho_1, \ldots, \varrho_P)'$ such that $\varrho_p \in \{0, 1\}$, $p \in [1, \ldots, P]$. Then, $\beta_p = 0$ when $\varrho_p = 0$, meaning that the corresponding row of $\beta$ is set to zero and that we are eliminating regressor $X_p$ from our model. When $\varrho_p = 1$ then $\beta_p \neq 0$, meaning that we are including regressor $X_p$ in our model. This is known as Spike-and-Slab prior and it can be written as

\[ \Pr(\beta, \Sigma_\varepsilon, \varrho) = \Pr(\beta_\varrho | \Sigma_\varepsilon, \varrho) \Pr(\Sigma_\varepsilon | \varrho) \Pr(\varrho). \]

We assume each element in $\varrho$ to be an independent Bernoulli distributed random variable with parameter $\pi$.

Indicating with $\theta = (\nu_c, \nu_r, S_c, S_r, X_{1:t^*})$ the vector of known parameters and matrices and denoting with $X_q$ and $H_q$ the selected regressors and the variance-covariance matrix of the corresponding rows of $\beta$, the full set of prior distributions at time $t \leq t^*$ is,
\[ \varrho | \theta \sim \prod_{p=1}^{P} \varrho_p (1 - \pi)^{1 - \varrho_p}, \]

\[ \Sigma_{\varepsilon} | \varrho, \theta \sim IW(\nu_{\varepsilon}, S_{\varepsilon}), \]

\[ \beta_{\psi} | \Sigma_{\varepsilon}, \varrho, \theta \sim N(0, H_{\psi}, \Sigma_{\varepsilon}), \]

\[ \alpha_t | Y_{1:t-1}, \Sigma_{\varepsilon}, \varrho, \theta \sim N(a_t, P_t, \Sigma), \]

\[ \Sigma_{r} | \theta \sim IW(\nu_r + t^*, S_S_{r}). \]

For the elicitation of prior hyperparameters, Brown et al. (1998) suggest setting \( \nu_{\varepsilon} = d + 2 \), which is the smallest integer value such that the expectation of \( \Sigma_{\varepsilon} \) exists. We use a similar strategy for \( \nu_r \). As for the scale matrices of the Inverse-Wishart distributions, in our empirical analysis we set \( S_{\varepsilon} = S_r = \begin{bmatrix} s_1^2 & s_1 s_2 \rho \\ s_1 s_2 \rho & s_2^2 \end{bmatrix} \), where, \( s_1^2, s_2^2 \) are the sample variances of the store and the competitor brand respectively and \( \rho \) is a correlation coefficient that can be elicited by incorporating our prior belief on the dependence structure of the two series. Finally we set \( H_{\varrho} = (X_{\varrho}' X_{\varrho})^{-1} \), which is the Zellner’s g-prior (Zellner and Siow, 1980).

### 3.1.2 Posterior Inference

Let \( \tilde{Y}_{1:t^*} = Y_{1:t^*} - Z_{1:t^*} \alpha_{1:t^*} \) indicate the observations up to time \( t^* \) with the time series component subtracted out. We can derive the following full conditionals distributions as,

\[ \beta_{\psi} | \tilde{Y}_{1:t^*}, \Sigma_{\varepsilon}, \varrho, \theta \sim N(M, W, \Sigma_{\varepsilon}), \quad (11) \]

\[ \Sigma_{\varepsilon} | \tilde{Y}_{1:t^*}, \varrho, \theta \sim IW(\nu_{\varepsilon} + t^*, S_S_{\varepsilon}), \quad (12) \]

\[ \Sigma_{r} | \eta_{1:t^*}^{(r)}, \theta \sim IW(\nu_r + t^*, S_S_{r}), \quad (13) \]

where \( M = (X_{\varrho}' X_{\varrho} + H_{\varrho}^{-1})^{-1} X_{\varrho}' \tilde{Y}_{1:t^*} \), \( W = (X_{\varrho}' X_{\varrho} + H_{\varrho}^{-1})^{-1} \), \( SS_{\varepsilon} = S_{\varepsilon} + \tilde{Y}_{1:t^*} \tilde{Y}_{1:t^*}' - M' W^{-1} M \), \( SS_{r} = S_r + \eta_{1:t^*}^{(r)} \eta_{1:t^*}^{(r)'} \), and \( \eta_{1:t^*}^{(r)} \) indicates the disturbances up to time \( t^* \) of the \( r \)-th state. Full proof of relations (11),(12) and (13) is given in Appendix B.1.

To sample from the joint posterior distribution of the states and model parameters we employ a Gibbs sampler in which we alternate sampling from the distribution of the states given the parameters and sampling from the distribution of the parameters given the states (see Algorithm 1 in Appendix B.1).

### 3.1.3 Prediction and estimation of causal effects

Given the draws from the joint posterior distribution of states and model parameters, we can use them to make in-sample and out-of-sample forecasts by drawing from the posterior predictive distribution. This process is particularly straightforward for in-sample forecasts.

Let \( \vartheta = (\alpha_{1:t^*}, \beta_{\psi}, \Sigma_{\varepsilon}, \Sigma_{r}, \varrho) \) be the vector of states and model parameters. To sample a new vector of observations \( Y_{1:t^*}^{\text{new}} \) given the observed data \( Y_{1:t^*} \), we note that,
\[ \Pr(Y_{1:t'}^{\text{new}} | Y_{1:t'}) = \int \Pr(Y_{1:t'}^{\text{new}} | \theta) \Pr(\theta | Y_{1:t'}) d\theta \]

(14)

\[ = \int \Pr(Y_{1:t'}^{\text{new}} | \theta) \Pr(\theta | Y_{1:t'}) d\theta \]

(15)

where the last equality follows because \( Y_{1:t'}^{\text{new}} \) is independent of \( Y_{1:t'} \), conditional on \( \theta \). We can, therefore, obtain in-sample forecasts from the posterior predictive distribution by using the draws from \( \Pr(\theta | Y_{1:t'}) \) that were obtained through the Gibbs sampler and substitute them in the model equations (9). We typically use in-sample forecasting for performing model checking.

To predict the counterfactual time series in the absence of an intervention, we need out-of-sample forecasts. Drawing from the predictive posterior distribution is still relatively straightforward, except the new samples are no longer independent of \( Y_{1:t'} \), given \( \theta \). To see this, consider the vector \( \theta' = (\alpha_{t'+k}, \ldots, \alpha_{t'+1}) \). Then,

\[ \Pr(Y_{t'+k} | Y_{1:t'}) = \int \Pr(Y_{t'+k}, \theta' | Y_{1:t'}) d\theta' = \int \Pr(Y_{t'+k}, \alpha_{t'+k}, \ldots, \alpha_{t'+1}, \theta | Y_{1:t'}) d\theta' = \]

\[ = \int \Pr(Y_{t'+k} | \alpha_{t'+k}, \ldots, \alpha_{t'+1}, \theta, Y_{1:t'}) \Pr(\alpha_{t'+k} | \alpha_{t'+k-1}, \ldots, \alpha_{t'+1}, \theta, Y_{1:t'}) \cdots \]

\[ \cdots \Pr(\alpha_{t'+1} | Y_{1:t'}, \theta) \Pr(\theta | Y_{1:t'}) d\theta' \]

To make out-of-sample forecasts, respecting the dependence structure highlighted above, we substitute the existing draws from \( \Pr(\theta | Y_{1:t'}) \), obtained by the Gibbs sampler, into the model equations (9), thereby updating the states and sampling the new sequence \( Y_{t'+1}, \ldots, Y_{t'+k} \).

### 3.2 Causal effect estimation

We can now estimate the causal effects defined in Section 2.2 by using the MBSTS models to predict the missing potential outcomes. In particular, we derive the posterior distribution of the general causal effect given in equation (1); the other two effects are simply functions or special cases of the general causal effect.

Let \( \Pr(Y_t(w) | Y_t^c(w)) \) and \( \Pr(Y_t(\bar{w}) | Y_t^c(\bar{w})) \) with \( t > t' \) be the out-of-sample draws from the posterior predictive distribution of the outcome under the treatment assignments \( w, \bar{w} \in \mathcal{W}_d \). Then,

\[ \Pr(\tau_t(w, \bar{w}) | Y_{1:t'}, (w), Y_{1:t'}(\bar{w})) = \Pr(Y_t(w) | Y_{1:t'}(w)) - \Pr(Y_t(\bar{w}) | Y_{1:t'}(\bar{w})) \]

(16)

is the posterior distribution of the general causal effect \( \tau_t(w, \bar{w}) \) and it is the difference between the posterior predictive distributions of the outcome under the two alternative treatment paths. Then, the posterior distributions of the cumulative general effect and the temporal average general effect at \( t' > t' \) can be derived from (16) as follows:

\[ \Pr(\Delta_t(w, \bar{w}) | Y_{1:t'}, (w), Y_{1:t'}(\bar{w})) = \sum_{t=t'+1}^{t'} \Pr(\tau_t(w, \bar{w}) | Y_{1:t'}, (w), Y_{1:t'}(\bar{w})) \]

(17)

\[ \Pr(\bar{\tau}_t(w, \bar{w}) | Y_{t'}, (w), Y_{t'}(\bar{w})) = \frac{1}{t' - t'} \Pr(\Delta_t(w, \bar{w}) | Y_{t'}, (w), Y_{t'}(\bar{w})) \]

(18)

Having the posterior distributions of the causal effects, we can easily compute posterior means and 95% credible intervals.

Notice that (16 - 18) do not require \( Y_t(w) \) or \( Y_t(\bar{w}) \) to be observed. However, estimation of unobserved potential outcomes other than \( Y_t(0, \ldots, 0) \) requires a strong set of model assumptions, and as such is often
less reliable. In our application, we are mostly interested in estimating the general effect \( \hat{\tau}(1,0), (0,0) = Y_t(1,0) - Y_t(0,0) \), where \( Y_t(1,0) \) is the observed outcome. The marginal and the conditional effects are of secondary importance and are included in the latter part of the analysis.

In practice, to obtain reliable estimates of the causal effects, the assumed model has to adequately describe the data. We therefore recommend to check model adequacy through the use of posterior predictive checks (Rubin, 1981, 1984; Gelman et al., 2013). Under our setup, we can also show that the above procedure yields unbiased estimates of the general causal effect, and, in turn, of the marginal and conditional effects. A detailed description of posterior predictive checks and the discussion of the frequentist properties of our estimators are given, respectively, in Appendix B.3 and B.2.

### 3.2.1 Combining results

Even though the main goal of the paper is estimating the heterogeneous effect on each cookie pair, it is possible to combine the results of all pairs and estimate an average effect. One way to accomplish this goal is through the use of meta-analysis. Indeed, as the number of time series increases, the estimation of a multivariate Bayesian model becomes computationally inefficient.

Meta-analysis is the statistical synthesis of the results obtained from multiple scientific studies and is often applied in the setting of a single study with multiple independent subgroups (Borenstein et al., 2011). For example, in a study investigating the effect of a drug, the researcher may divide the participants in different groups according to the stage of the disease; in our application, the subgroups are the cookie pairs. We then treat each pair as an independent study and follow the standard steps in a meta-analysis, described below.

One basic meta-analysis approach is computing the summary effect as a weighted average of point estimates (i.e., the results of the individual studies) with weights based on the estimated standard errors. The main caveat of this approach is the inherent dependence on the sample size: a small number of studies would result in a loss of precision of the estimated between-study variance. In such case, we can resort to a fully Bayesian meta-analysis (Smith et al., 1995; Sutton and Abrams, 2001; Sutton and Higgins, 2008). This approach is based on hierarchical Bayesian models that assume a distribution on the true effect and place suitable priors on its hyperparameters.

The above described methodologies can be used to combine point estimates of multiple independent studies. However, by following the estimation process described in this paper we obtain a posterior distribution of the general causal effect for each analyzed cookie pair. As a result, combining the estimates of the individual pairs is a lot more intuitive.

For example, let \( \tau_{j,t}(w, \tilde{w}) \) be the temporal average causal effect on the \( j \)-th cookie pair and assume we estimated a posterior distribution for each \( j \) as in (18). Then, we can define the summary temporal average effect across all \( j \) pairs and its posterior distribution as,

\[
\bar{\tau}_t(w, \tilde{w}) = \frac{1}{J} \sum_{j=1}^{J} \tau_{j,t}(w, \tilde{w})
\]

(19)

\[
Pr(\bar{\tau}_t(w, \tilde{w}) | Y_{1:t^*}(w), Y_{1:t^*}(\tilde{w})) = \frac{1}{J} \sum_{j=1}^{J} Pr(\tau_{j,t}(w, \tilde{w}) | Y_{1:t^*}(w), Y_{1:t^*}(\tilde{w})).
\]

(20)

In words, to combine the estimated temporal average effect of the individual cookie pairs we can directly average across their posterior distributions.
4 Simulation study

We now describe a simulation study exploring the frequentist properties of our proposed approach for correctly specified models and a misspecified model. As expected, our model performs well under the correct model and minor misspecification; more importantly, we show that posterior predictive checks are a viable approach to test model adequacy.

4.1 Design

We generate simulated data according to the following MBSTS model:

\[
\begin{align*}
Y_t &= \mu_t + \gamma_t + X_t \beta + \epsilon_t \\
\mu_{t+1} &= \mu_t + \eta_{t,\mu} \\
\gamma_{t+1} &= -\sum_{s=0}^{S-2} \gamma_{t-s} + \eta_{t,\gamma} \\
\epsilon_t &\sim N_d(0, H_t \Sigma) \\
\eta_{t,\mu} &\sim N_d(0, c_1 \Sigma) \\
\eta_{t,\gamma} &\sim N_d(0, c_2 \Sigma)
\end{align*}
\] (21)

Where \( Y_t = (Y_1, Y_2) \) is a bivariate time series, \( \mu_t \) is a trend component evolving according a random walk and \( \gamma_t \) is a seasonal component with period \( S = 7 \). We further set \( H_t = 1, c_1 = 3, c_2 = 2 \) and \( \Sigma = \begin{bmatrix} 1 & -0.3 \\ -0.3 & 1 \end{bmatrix} \).

We then assume a regression component formed by two covariates, \( X_1 \sim f(x) \), with \( f(x) = 1 - x + N(0, 0.5) \) and \( X_2 \sim N(2, 0.3) \) while \( \beta \) is sampled from a matrix-normal distribution with mean \( b_0 = 0 \) and \( H = I_P \).

To estimate the causal effect, we use two different models for inference: a correctly specified model with both trend and seasonal components (M1) and a misspecified model with only the seasonal part (M2). For both models we choose the following set of hyperparameters: \( \nu_\epsilon = \nu_\tau = 4; S_x = S_r = 0.2 \begin{bmatrix} s_1^2 & s_1 s_2 \rho \\ s_1 s_2 \rho & s_2^2 \end{bmatrix} \), where \( s_1^2 \) and \( s_2^2 \) are the sample variances of, respectively, \( Y_1 \) and \( Y_2 \) and \( \rho = -0.8 \) is a correlation coefficient reflecting our prior belief of their dependence structure; and Zellner’s g-prior for the variance-covariance matrix of \( \beta \).

To make our simulation close to our empirical application, we generated 1,000 data sets in a fictional time period starting January 1, 2018 and ending June 30, 2019. We model the intervention as taking place on January 2, 2019, and assume a fixed persistent contemporaneous effect; for example, the series goes up by +10% and stays at this level throughout. To study the empirical power and coverage, we tried 5 different impact sizes ranging from +1% to +100% on \( Y_1 \) and from −1% to −90% on \( Y_2 \). After generating the data, we estimated the effects using both M1 and M2, for a total of 10,000 estimated models (one for each data set, impact size and model type), each having 1,000 draws from the resulting posterior distribution. Finally, we predicted the counterfactual series in the absence of intervention for three-time horizons, namely, after 1 month, 3 months, and 6 months from the intervention.

We evaluate the performance of the models in terms of:

1. length of the credible intervals around the temporal average general effect \( \bar{\tau}_t(1,0), (0,0) \);
2. absolute percentage estimation error, computed as
\[
\frac{|\hat{\tau}_t(1,0), (0,0) - \bar{\tau}_t(1,0), (0,0)|}{\bar{\tau}_t(1,0), (0,0)};
\]
3. interval coverage, namely, the proportion of the true pointwise effects covered by the estimated 95% credible intervals.
We focus on the percentage estimation error because without normalizing the bias different effect sizes are not immediately comparable. To see this, consider that a small bias for estimating a substantial effect is better than that same bias when trying to estimate a small effect.

4.2 Results

Tables 1 reports the average interval length under M1 and M2 for all effect sizes and time horizons. As expected, the length of credible intervals estimated under M1 increases with the time horizon. In contrast, for M2, the interval length is stable across time as the model lacks a trend component and assumes a certain level of stability. Figure 2 shows the absolute percentage errors for the first time horizon. We see, unsurprisingly, that it decreases as the effect size increases. This suggests that small effects are more difficult to detect. To confirm this claim, in Figure 3, we report the percentage of times we detect a causal effect over the 1,000 simulated data sets. Under M1 for the two smallest effect sizes—which exhibit the highest estimation errors—we rarely correctly conclude that a causal effect is present. However, when the effect size increases we can detect the presence of a causal effect at a much higher rate. The results under M2 are somewhat counterintuitive as, even though the model is misspecified, smaller effects are more easily detected. This phenomenon occurs primarily because of the smaller credible intervals; that is, for small effect sizes, our results are biased with low variance, which means we often conclude there is an effect.

Finally, Table 2 reports the average interval coverage under M1 and M2. The coverage under M2 ranges from 82.0% to 88.6%, which is lower than the desired 95%. In contrast, the frequentists coverage under M1 is exactly at the nominal 95% for both $Y_1$ and $Y_2$.

Table 1: Length of credible intervals around the temporal average general effect, $\tilde{\tau}_t((1,0),(0,0))$ estimated under M1 and M2 for each effect size and time horizon.

<table>
<thead>
<tr>
<th>$\tilde{\tau}_t((1,0),(0,0))$</th>
<th>1 month</th>
<th>3 months</th>
<th>6 months</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$Y_1$</td>
<td>$Y_2$</td>
<td>$Y_1$</td>
</tr>
<tr>
<td>(1.01, 0.99)</td>
<td>20.93</td>
<td>21.10</td>
<td>27.62</td>
</tr>
<tr>
<td>(1.10, 0.90)</td>
<td>21.34</td>
<td>21.37</td>
<td>28.09</td>
</tr>
<tr>
<td>M1</td>
<td>21.33</td>
<td>21.30</td>
<td>28.18</td>
</tr>
<tr>
<td>(1.25, 0.75)</td>
<td>21.30</td>
<td>21.31</td>
<td>28.11</td>
</tr>
<tr>
<td>(1.50, 0.50)</td>
<td>21.38</td>
<td>21.25</td>
<td>28.24</td>
</tr>
<tr>
<td>(2.00, 0.10)</td>
<td>30.39</td>
<td>30.39</td>
<td>30.40</td>
</tr>
<tr>
<td>(1.01, 0.99)</td>
<td>30.48</td>
<td>30.48</td>
<td>30.50</td>
</tr>
<tr>
<td>M2</td>
<td>30.48</td>
<td>30.46</td>
<td>30.51</td>
</tr>
<tr>
<td>(1.25, 0.75)</td>
<td>30.45</td>
<td>30.43</td>
<td>30.47</td>
</tr>
<tr>
<td>(1.50, 0.50)</td>
<td>30.49</td>
<td>30.49</td>
<td>30.52</td>
</tr>
</tbody>
</table>

Overall, the simulation results suggest that when the model is correctly specified, the proposed approach performs well in estimating the causal effect of an intervention. Conversely, when the model is misspecified, the estimation error increases and the credible intervals do not achieve the required coverage. Although the results are likely to still provide practitioners with useful insights.

In practice, we recommend testing the adequacy of our model before performing substantive analysis by using posterior predictive checks. In Appendix A.2 we provide examples results obtained under M1 (Figures 9 and 10) and posterior predictive checks under both M1 and M2. From the observation of Figures 11 and 12, we can immediately see that M1 yields a better approximation of the empirical density of the simulated data and lower residual autocorrelation than M2.
Figure 2: Average absolute percentage error (± 2 s.e.m) at the first time horizon under M1 (blue) and M2 (orange) for the impact sizes ≥ 10% (Y₁) and ≤ −10% (Y₂).

Figure 3: Average proportion of credible intervals excluding zero (± 2 s.e.m) at the first time horizon under M1 (blue) and M2 (orange) for all impact sizes.

Table 2: Interval coverage under M1 and M2 for each effect size and time horizon.

<table>
<thead>
<tr>
<th>τₜ((1, 0), (0, 0))</th>
<th>1 month</th>
<th>3 months</th>
<th>6 months</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Y₁</td>
<td>Y₂</td>
<td>Y₁</td>
</tr>
<tr>
<td>(1.01, 0.99)</td>
<td>96.0</td>
<td>95.0</td>
<td>96.1</td>
</tr>
<tr>
<td>(1.10, 0.90)</td>
<td>95.9</td>
<td>94.9</td>
<td>96.0</td>
</tr>
<tr>
<td>M1 (1.25, 0.75)</td>
<td>96.0</td>
<td>95.0</td>
<td>96.0</td>
</tr>
<tr>
<td>(1.50, 0.50)</td>
<td>96.1</td>
<td>94.9</td>
<td>96.1</td>
</tr>
<tr>
<td>(2.00, 0.10)</td>
<td>95.9</td>
<td>95.0</td>
<td>96.1</td>
</tr>
<tr>
<td>(1.01, 0.99)</td>
<td>86.8</td>
<td>88.4</td>
<td>85.5</td>
</tr>
<tr>
<td>(1.10, 0.90)</td>
<td>87.0</td>
<td>88.5</td>
<td>85.7</td>
</tr>
<tr>
<td>M2 (1.25, 0.75)</td>
<td>87.0</td>
<td>88.6</td>
<td>85.7</td>
</tr>
<tr>
<td>(1.50, 0.50)</td>
<td>86.9</td>
<td>88.6</td>
<td>85.6</td>
</tr>
<tr>
<td>(2.00, 0.10)</td>
<td>86.9</td>
<td>88.6</td>
<td>85.7</td>
</tr>
</tbody>
</table>
5 Empirical analysis

We now describe the results of our empirical application where we analyze a marketing campaign run by an Italian supermarket chain in its Florence’s stores. The campaign consisted of introducing a permanent price reduction on a selected subset of store brands. The main goal of the policy change was to increase the customer base and sales. The policy change affected 707 products in several categories; below, we provide the details for the “cookies” category.

5.1 Data & methodology

Among the 284 items in the “cookies” category, there are 28 store brands, of which 11 were selected for a permanent price reduction ranging from $-3.5\%$ to $-23.2\%$; in particular, the tenth store brand exhibits the highest price reduction, while the median is $-11.8\%$. For each store brand, the supermarket chain identified a direct competitor brand, thereby defining 11 pairs of cookies. Those in the same pair are almost identical except for their brand name. In contrast, cookies belonging to different pairs differ on several characteristics (e.g., ingredients, market target, and weight). This suggests that the permanent discount on a store brand is likely to impact its direct competitor, but is unlikely to affect the sales of the cookies in different pairs, allowing us to justify the partial temporal no-interference assumption.

Our data consists of daily sales data for all cookies from September 1, 2017, until April 30, 2019. Our outcome variable is the average units sold per hour—computed as the number of units sold daily divided by the number of hours that the stores stay open. We focus on hourly average sales because Italian regulations dictate that the supermarket chain only operates for a limited number of hours on Sundays; this discrepancy leads to a considerable difference in daily sales.

As an example, Figure 4 shows the time series of daily units sold by two store brands, their price, and the autocorrelation function. The plots show a strong weekly seasonal pattern. Figure 5 exhibits the same plots for two competitor brands. The occasional drops in the price series are from temporary promotions run regularly by the supermarket chain. In our data, the competitor brands are subject to several promotions during the analysis period. However, those differ from the permanent price reduction on their temporary nature and the regular frequency. As our goal is to evaluate the effectiveness of the store’s policy change—a permanent price reduction—we will not consider temporary promotions as interventions. There is also considerable visual evidence from the data that the intervention on the store brands has influenced the competitor cookies’ prices policy. Indeed, all competitor brands (with the exception of brand 10) received a temporary promotion matching the time of the intervention, suggesting that competitors may have reacted to the new policy.

Under partial temporal no-interference, we fit an MBSTS model for each pair; we also use covariates to improve the prediction of the counterfactual series. In particular, the set of regressors include: two dummies taking value 1 on Saturday and Sunday, the former being the most profitable day of the week, whereas on the latter stores operate reduced hours; a holiday dummy taking value 1 on the day before and after a national holiday, accounting for consumers’ tendency to shop more before and after a closure day; a set of synthetic controls selected among one category that did not receive active treatment (e.g., wine sales). Including covariates should increase prediction accuracy in the absence of intervention, but suitable covariates must respect two conditions: they should be good predictors of the outcome before the intervention, and they must satisfy Assumption 3. As a result, the unit prices can not be part of our models; nevertheless, they are important drivers of sales, especially during promotions (Neslin et al., 1985; Blattberg et al., 1995; Pauwels et al., 2002). We solved this issue by using the “prior price”, which is equal to the actual price up to the intervention, and

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5Because of missing data on one of the competitor brands, we perform the analysis on 10 pairs.
6The same plots for all the remaining store and competitor brands are provided in Appendix A.2.
7See Figure 8 in Appendix A.2.
Figure 4: Store brands. Starting from the left: time series of the average unit sold per hour; evolution of price per unit; autocorrelation function. The price plot shows the permanent price reduction after the intervention date (indicated by the vertical bar), i.e., the price of the store brands is lowered and stays at this level throughout.

Figure 5: Competitor brands. Starting from the left: time series of the average unit sold per hour; evolution of price per unit; evolution of price relative to the store brand; autocorrelation function. The price plot shows the temporary promotions these brands are subject to, i.e., both before and after the intervention date (indicated by the vertical bar) the price of competitor brands is reduced for a while and then bounces back to the original level.

then it is set equal to the last price before intervention (which is the most reliable estimate of what would have happened in the absence of intervention).

Finally, to speed up computations, the set of synthetic controls is selected in two steps: first, we select the best ten matches among the 260 possible control series in the “wines” category by dynamic time warping,\(^8\) then, we group them with the other predictors and perform multivariate Bayesian variable selection.

Each model is estimated in the period before the intervention; then, as described in Section 3.1.3, we predicted the counterfactual series in the absence of intervention by performing out-of-sample forecasts. Next, we estimate the intervention’s causal effect at three different time horizons: one month, three months, and six months from the treatment day. This allows us to determine whether the effect persists over time or quickly disappears.

\(^8\)Dynamic time warping (DTW) is a technique for finding the optimal alignment between two time series. Instead of minimizing the Euclidean distance between the two sequences, it finds the minimum-distance warping path, i.e., given a matrix of distances between each point of the first series with each point of the second series, contiguous set of matrix elements satisfying some conditions. For further details see Keogh and Ratanamahatana (2005); Salvador and Chan (2007). Implementation of DTW has been done with the R package MarketMatching (Larsen, 2019).
5.2 Results

We now present the results for the best MBSTS model with both a trend and seasonality component. Our posterior predictive checks selected this model, see Appendix A.2 for the details, and Appendix A.3 for a description of the other models tried. Convergence diagnostics are provided in Appendix B.5.

The estimates of the temporal average general effect are reported in Table 3, which reveals the presence of three significant causal effects — where the 95% credible intervals do not include 0 — on the store brands belonging to pairs 4, 7 and 10 at the first time horizon. Interestingly, we do not find a significant effect on the competitor brands in the same pairs. This can be explained by the fact that, during the intervention period, competitor brands were subject to multiple temporary promotions that might have reduced the negative impact of the permanent discount on store brands. Furthermore, in Italy store brands products are rather newer than their competitors; so, despite the price reduction on store brand cookies, some consumers may still prefer the competitor cookie because of subjective factors, such as brand loyalty. Another important result is that after the initial surge in sales, we cannot detect a significant effect for longer time horizons. Figure 6 plots the pointwise general effect $\hat{\tau}(1,0),(0,0))$ for the fourth pair at each time horizon, that is, the difference between the observed series and the predicted counterfactual computed at every time point. See Appendix A.2 for additional plots.

Overall, these results suggest that the policy change had a minor impact on the store brands’ sales. Furthermore, since we do not detect an effect after the first month, it seems that this intervention failed to significantly and permanently impact sales. Of course, as we showed in the simulation study, there could have been a small effect that our model was unable to detect. However, since the company needed a significant boost in sales to make up for the loss in profits due to the price reduction, we can conclude that this policy was not effective. This result is robust to different prior assumptions, see Appendix B.4 for a detailed sensitivity analysis.

As discussed in the introduction, we could have analyzed the data by aggregating the sales of store and competitor brands and treating each aggregate as a univariate time series. This procedure, however, leads to a loss of information, providing misleading results that could drive the analyst to make the wrong decision. To show that, we estimated the causal effect using the univariate BSTS models on a range of different aggregated sales. We report the results for three: the average sales of the brands in the same pair, the average sales of all store brands, and the average sales of all store and competitor brands. The average is computed as the total number of units sold daily by all products in the aggregate divided by the opening hours. Notice that we did not consider the aggregate of the competitor brands alone. This is because it would have required the prediction of the counterfactual series under treatment.

Like the multivariate analysis, for each aggregate, we used a model that contained a trend and seasonality component as well as a set of covariates. The covariates included the three dummies (described earlier), aggregate sales of all wines, and the prior price—computed by averaging the prior prices of all cookies in each aggregate. Table 4 shows the results of the univariate analysis. We find evidence of a positive effect on the tenth pair at the first and second-time horizons and a positive effect on the eighth pair at the first horizon. In addition, the estimated effects on the store brands aggregate and the store-competitor aggregate are both positive and significant for the first time horizon. To provide a comparison with these last two aggregates, Table 4 reports the summary temporal average effect on all cookie pairs obtained by combining the individual estimates with a meta-analysis, as described in Section 3.2.1. The summary effect on the store brands is positive and significant at the first time horizon and, interestingly, it is in line with the estimated effect on the store brands aggregate from the univariate analysis. However, with a univariate analysis we are not able to isolate the effect on the competitor brands and we would have erroneously concluded that the new policy had a positive impact on the store-competitor aggregate, whereas the meta-analysis shows that the effect on competitor brands is not significant. Overall, despite a similar result for the tenth pair, however, we would have reached wrong conclusions for pairs 4, 7 and 8, and we would have reported the misleading finding of an overall positive impact on the sales.
of store-competitor aggregate.

To further illustrate all the different types of effects that it is possible to estimate in a multivariate setting, we estimated the mean marginal effect and the conditional effect \( \hat{\tau}_t((1,1),(0,1)) \). Ultimately, we found three significant marginal effect on the sales of store brands but there is no evidence that the new price policy has had an effect in a scenario where both cookies in pairs are treated compared to the scenario where only the competitor brand is treated. The results are given in Table 5 and Table 6 in Appendix A.1.

**Table 3:** Temporal average general causal effects of the new price policy on the ten store (s) - competitor (c) pairs computed at three time horizon. In this table, \( \hat{\tau}_t \) stands for the general effect \( \hat{\tau}_t((1,0),(0,0)) \).

<table>
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<th>6 months</th>
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<td>97.5%</td>
<td>( \hat{\tau}_t )</td>
<td>2.5%</td>
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</tr>
<tr>
<td></td>
<td>c</td>
<td>0.04</td>
<td>-9.36</td>
<td>9.79</td>
<td>1.92</td>
</tr>
</tbody>
</table>

**Figure 6:** Pointwise causal effect of the permanent price reduction on the fourth store-competitor pair at 1 month, 3 months and 6 months after the intervention.
Table 4: Univariate temporal average causal effect ($\hat{\tau}_t$) at three time horizons of the new price policy on: i) aggregated sales (pairs 1-10); ii) the store brands aggregate (SA); iii) the store - competitor aggregate (SCA). The last two lines show, separately for the store brands (META-S) and the competitor brands (META-C), the summary temporal average effect combined with a meta-analysis.

<table>
<thead>
<tr>
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<td>$\hat{\tau}_t$</td>
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</tr>
<tr>
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<td>121.54</td>
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6 Conclusion

This paper formalized a potential outcomes framework to estimate causal effects in panel settings with interference and multiple treated units. Our motivating example was the introduction of a new price policy (permanent price reduction) by a supermarket chain on a selected subset of store brands. Having observed the sales of store and competitor brands before and after the policy change, we were interested in estimating the causal effect of the permanent price reduction on both brands.

We first addressed the issue of interference between units by relying on the partial temporal no-interference assumption. Then, we introduced three classes of estimands focusing on the heterogeneous causal effect and proposed to estimate them by using multivariate Bayesian structural time series to forecast the group outcome in the absence of intervention. Finally, we tested our approach on a simulation study, and then we applied it to our motivating example.

We believe that our approach brings several contributions to the nascent stream of literature on synthetic control methods in panel settings with interference. First, we derived a wide class of new causal estimands. Second, MBSTS allows us to model the interference between units in the same group by explicitly modeling their dependence structure and, simultaneously, ensuring a transparent way to deal with the surrounding uncertainty. Finally, the approach is flexible, and the underlying distributional assumptions can be tested in a very natural way by posterior inference.
References


Appendices
A

A.1 Additional tables

We report below the results for the mean marginal effect as defined in (7) and the conditional effect $\hat{\tau}_t((1, 1), (0, 1))$.

Table 5: Temporal average mean marginal causal effect of the new price policy on the ten store brands computed at three time horizons.

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Table 6: Temporal average conditional causal effect of the new price policy on the ten store (s) - competitor (c) pairs computed at three time horizons. In this table, $\hat{\tau}_t$ stands for the conditional effect $\hat{\tau}_t((1, 1), (0, 1)).$

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<td>0.84</td>
<td>-0.31</td>
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<td>(7)</td>
<td>s 0.20</td>
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<td>1.60</td>
<td>0.21</td>
<td>-1.14</td>
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<td>6.93</td>
<td>0.07</td>
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<td>10.90</td>
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A.2 Additional plots

Figure 7: Store brands. Starting from the left: time series of the average unit sold per hour; evolution of price per unit; autocorrelation function. The vertical bar indicates the intervention date.
Figure 8: Competitor brands. Starting from the left: time series of the average unit sold per hour; evolution of price per unit; evolution of price relative to the store brand (in here, referred to as TP, treated product in the pair); autocorrelation function. The vertical bar indicates the intervention date.
Figure 9: For one of the simulated data sets at 6-month horizon, the figure plots: (a) simulated time series assuming an effect size of +50% (orange) vs true counterfactual series generated under model (21) (blue); (b) true counterfactual vs predicted counterfactual series under M1; (c) true effect (black dashed line) vs the inferred effect under M1.

Figure 10: For one of the simulated data sets at 6-month horizon, the figure plots: (a) simulated time series assuming an effect size of −50% (orange) vs true counterfactual series generated under model (21) (blue); (b) true counterfactual vs predicted counterfactual series under M1; (c) true effect (black dashed line) vs the inferred effect under M1.
Figure 11: Posterior predictive checks under M1 for $Y_1$ (first row) and $Y_2$ (second row) for one of the simulated data sets. Starting from the left: i) density of observed data (black) plotted against the posterior predictive mean (blue); ii) observed maximum compared to the distribution of the maximum from the posterior draws; iii) Normal QQ-Plot of standardized residuals; iv) autocorrelation function of standardized residuals.

Figure 12: Posterior predictive checks under M2 for $Y_1$ (first row) and $Y_2$ (second row) for one of the simulated data sets. Starting from the left: i) density of observed data plotted against the posterior predictive mean; ii) observed maximum compared to the distribution of the maximum from the posterior draws; iii) Normal QQ-Plot of standardized residuals; iv) autocorrelation function of standardized residuals.
Figure 13: Pointwise causal effect of the permanent price reduction on each store-competitor pair at 1 month, 3 months and 6 months after the intervention.
Figure 14: For each store-competitor pair, observed outcome (in black) plotted against the counterfactual outcome in the absence of intervention (in blue) after 1 month, 3 months and 6 months from the intervention, indicated by the red vertical line.
Figure 15: Posterior predictive checks for each pair. Starting from the left: i) density of observed data (black) plotted against the posterior predictive mean (blue); ii) observed maximum compared to the distribution of the maximum from the posterior draws; iii) Normal QQ-Plot of standardized residuals; iv) autocorrelation function of standardized residuals.
Figure 16: Inclusion probabilities above the 0.5 threshold of the regressors included in the model.
A.3 Posterior predictive checks of alternative models

Figure 17: Posterior predictive checks for a seasonal MBSTS model. Starting from the left: i) density of observed data (black) vs posterior predictive mean (blue); ii) observed maximum vs distribution of the maximum from the posterior draws; iii) Normal QQ-Plot of standardized residuals; iv) autocorrelation function of standardized residuals.
Figure 18: Posterior predictive checks for a trend MBSTS model. Starting from the left: i) density of observed data (black) plotted against the posterior predictive mean (blue); ii) observed maximum compared to the distribution of the maximum from the posterior draws; iii) Normal QQ-Plot of standardized residuals; iv) autocorrelation function of standardized residuals.
Figure 19: Posterior predictive checks for a trend and seasonal MBSTS model estimated on the daily units sold. Starting from the left: i) density of observed data (black) plotted against the posterior predictive mean (blue); ii) observed maximum compared to the distribution of the maximum from the posterior draws; iii) Normal QQ-Plot of standardized residuals; iv) autocorrelation function of standardized residuals.
B

B.1 Posterior inference

We provide below the proof of relations (11), (12) and (13).

$\beta$ has prior density function given by

$$
\Pr(\beta | \Sigma, \varrho, \theta) = (2\pi)^{-p_{\theta}/2} \det(\Sigma)^{-p_{\theta}/2} \exp \left\{ \frac{-1}{2} \text{tr} \left[ \Sigma^{-1} \beta \Sigma^{-1} \beta' \right] \right\} 
$$

$$
= (2\pi)^{-p_{\theta}/2} \det(\Sigma)^{-p_{\theta}/2} \exp \left\{ \frac{-1}{2} \text{tr} \left[ \beta' H^{-1}_{\theta} \beta \Sigma^{-1} \right] \right\}
$$

Where $p_{\theta}$ is the number of selected regressors. Similarly, the density function $\Pr(\tilde{Y}_{1:t^*})$ can be written as,

$$
\Pr(\tilde{Y}_{1:t^*} | \beta_{\theta}, \Sigma_{\epsilon}, \varrho, \theta) = (2\pi)^{-dt^*/2} \det(\Sigma_{\epsilon})^{-t^*/2} \exp \left\{ -\frac{1}{2} \sum_{t=1}^{t^*} (\tilde{Y}_{1:t^*} - X_{\theta} \beta) \Sigma_{\epsilon}^{-1} (\tilde{Y}_{1:t^*} - X_{\theta} \beta)' \right\}
$$

$$
= (2\pi)^{-dt^*/2} \det(\Sigma_{\epsilon})^{-t^*/2} \exp \left\{ -\frac{1}{2} \text{tr} \left[ (\tilde{Y}_{1:t^*} - X_{\theta} \beta)' (\tilde{Y}_{1:t^*} - X_{\theta} \beta) \Sigma_{\epsilon}^{-1} \right] \right\}
$$

Now we can derive the posterior distribution for the regression coefficients as follows,

$$
\Pr(\beta_{\theta} | \tilde{Y}_{1:t^*}, \Sigma_{\epsilon}, \varrho, \theta) \propto \Pr(\tilde{Y}_{1:t^*} | \beta_{\theta}, \Sigma_{\epsilon}, \varrho, \theta) \Pr(\beta_{\theta} | \Sigma_{\epsilon}, \varrho, \theta)
$$

$$
\propto \exp \left\{ -\frac{1}{2} \text{tr} \left[ (\tilde{Y}_{1:t^*} - X_{\theta} \beta)' (\tilde{Y}_{1:t^*} - X_{\theta} \beta) \Sigma_{\epsilon}^{-1} \right] \right\}
\exp \left\{ -\frac{1}{2} \text{tr} \left[ \beta_{\theta}' H^{-1}_{\theta} \beta_{\theta} \Sigma_{\epsilon}^{-1} \right] \right\}
$$

$$
\propto \exp \left\{ -\frac{1}{2} \text{tr} \left[ \beta_{\theta}' (X_{\theta}' X_{\theta} + H^{-1}_{\theta}) \beta_{\theta} \Sigma_{\epsilon}^{-1} - 2 \beta_{\theta}' X_{\theta} \tilde{Y}_{1:t^*} \Sigma_{\epsilon}^{-1} - 2 \beta_{\theta}' X_{\theta} \Sigma_{\epsilon}^{-1} - 2 \beta_{\theta}' X_{\theta} \tilde{Y}_{1:t^*} \Sigma_{\epsilon}^{-1} \right] \right\}
$$

Which is the kernel of a matrix-normal distribution $\mathcal{N}(M, W, \Sigma_{\epsilon})$, with $W = (X_{\theta}' X_{\theta} + H^{-1}_{\theta})^{-1}$ and $M = (X_{\theta}' X_{\theta} + H^{-1}_{\theta})^{-1} X_{\theta}' \tilde{Y}_{1:t^*}$.

Integration of the above quantity is necessary to derive the posterior distribution of $\Sigma_{\epsilon}$ and yields the inverse of the normalization constant, which is $\kappa = (2\pi)^{p_{\theta}d/2} \det(W)^{d/2} \det(\Sigma_{\epsilon})^{p_{\theta}/2}$. However, $\kappa$ simplifies with the constants singled out from the integral, which are

$(2\pi)^{-p_{\theta}d/2} \det(\Sigma_{\epsilon})^{-p_{\theta}/2} \det(H_{\theta})^{-d/2}$ and $(2\pi)^{-dt^*/2} \det(\Sigma_{\epsilon})^{-t^*/2}$, leaving

$\det(H_{\theta})^{-d/2} \det(W)^{d/2}(2\pi)^{-dt^*/2} \det(\Sigma_{\epsilon})^{-t^*/2}$. 

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Pr(\(\Sigma_c|\tilde{Y}_{1:t^*}, \varrho, \theta\)) \propto Pr(\tilde{Y}_{1:t^*}|\Sigma_c, \varrho, \theta) Pr(\Sigma_c|\varrho, \theta) \\
\propto Pr(\Sigma_c|\varrho, \theta) \int Pr(\tilde{Y}_{1:t^*}|\beta_{\varrho}, \Sigma_c, \varrho, \theta) Pr(\beta_{\varrho}|\Sigma_c, \varrho, \theta) d\beta \\
\propto \det(\Sigma_c)^{-d/(\nu_c + t^* + 1)/2} \exp \left\{ -\frac{1}{2} \text{tr} (S_c \Sigma_c^{-1}) \right\} \exp \left\{ -\frac{1}{2} \text{tr} \left[ (\tilde{Y}_{1:t^*} - M'W^{-1}M)\Sigma_c^{-1} \right] \right\} \\
\propto \det(\Sigma_c)^{-d/(\nu_c + t^* + 1)/2} \exp \left\{ -\frac{1}{2} \text{tr} \left[ (S_c + \tilde{Y}_{1:t^*} - M'W^{-1}M)\Sigma_c^{-1} \right] \right\}

This is the kernel of an Inverse-Wishart distribution with \(\nu = \nu_c + t^*\) degrees of freedom and scale matrix \(SS_c = (S_c + \tilde{Y}_{1:t^*} - M'W^{-1}M)\). We can also derive the posterior of the latent vector \(\varrho\),

\[
Pr(\varrho|\tilde{Y}_{1:t^*}, \theta) = \frac{Pr(\tilde{Y}_{1:t^*}|\varrho, \theta) Pr(\varrho)}{\sum_{\varrho} Pr(\tilde{Y}_{1:t^*}|\varrho, \theta) Pr(\varrho)}
\]

where,

\[
Pr(\tilde{Y}_{1:t^*}|\varrho, \theta) = \int \left( \int Pr(\tilde{Y}_{1:t^*}|\beta_{\varrho}, \Sigma_c, \varrho, \theta) Pr(\beta_{\varrho}|\Sigma_c, \varrho, \theta) Pr(\Sigma_c|\varrho, \theta) d\beta d\Sigma_c \right) \\
= \int \left( \int \left( \int Pr(\tilde{Y}_{1:t^*}|\beta_{\varrho}, \Sigma_c, \varrho, \theta) Pr(\beta_{\varrho}|\Sigma_c, \varrho, \theta) d\beta \right) Pr(\Sigma_c|\varrho, \theta) d\Sigma_c \right) \\
= \frac{\det(\Sigma_c)^{\nu_c/2}}{2^{\nu_c/2}2^{\nu_c/2}} \det(\Sigma_c)^{-(\nu_c + d + 1)/2} \exp \left\{ -\frac{1}{2} \text{tr} \left[ (\tilde{Y}_{1:t^*} - M'W^{-1}M)\Sigma_c^{-1} \right] \right\} \\
= \frac{\det(\Sigma_c)^{\nu_c/2}}{2^{\nu_c/2}2^{\nu_c/2}} \det(\Sigma_c)^{-(\nu_c + d + 1)/2} \exp \left\{ -\frac{1}{2} \text{tr} \left[ (S_c + \tilde{Y}_{1:t^*} - M'W^{-1}M)\Sigma_c^{-1} \right] \right\} \\
= \frac{\det(\Sigma_c)^{\nu_c/2}}{2^{\nu_c/2}2^{\nu_c/2}} \det(\Sigma_c)^{-(\nu_c + d + 1)/2} \exp \left\{ -\frac{1}{2} \text{tr} \left[ (S_c + \tilde{Y}_{1:t^*} - M'W^{-1}M)\Sigma_c^{-1} \right] \right\} \\
= \frac{\det(\Sigma_c)^{\nu_c/2}}{2^{\nu_c/2}2^{\nu_c/2}} \det(\Sigma_c)^{-(\nu_c + d + 1)/2} \exp \left\{ -\frac{1}{2} \text{tr} \left[ (S_c + \tilde{Y}_{1:t^*} - M'W^{-1}M)\Sigma_c^{-1} \right] \right\}
\]

Notice that if we set \(H_{\varrho} = (X'_pX_p)^{-1}\), the above expressions simplify to \(W = \frac{1}{2}(X'_pX_p)^{-1}\), \(M = \frac{1}{2}(X'_pX_p)^{-1}X'_p\tilde{Y}_{1:t^*}\), and \(SS_c = S_c + \tilde{Y}_{1:t^*} - \frac{1}{2}Y'_1Y_{1:t^*}(X'_pX_p)^{-1}X'_p\tilde{Y}_{1:t^*}\).

In order to evaluate the posterior distribution \(Pr(\varrho|\tilde{Y}_{1:t^*}, \theta)\) we can resort to the odds and update the elements of the selection vector one component at a time, while the others are held fixed. This ensures that at each step only the most likely model is retained, either the one with \(X_p\) in it or the one without. More formally, let \(\varrho_p = 1\) and indicate with \(\varrho_{-p}\) the vector of all the elements in \(\varrho\) except \(\varrho_p\). The full conditional of \(\varrho_p\) is given by,
To sample from the joint posterior distribution of the states and model parameters we can employ the Gibbs sampler to draw from the joint posterior distribution of the states and model parameters. Algorithm 1

\begin{align*}
\text{Require: } & \Sigma^{(0)}, \Sigma^{(0)}_R, \theta, H, \text{niter} \\
1: & \text{for } s \text{ in } 1 : \text{niter do} \\
2: & \text{draw } \alpha^{(s)}_t \text{ from } \Pr(\alpha_t | \mathbf{Y}_{1:t^*}, \Sigma^{(s-1)}_\epsilon, \Sigma^{(s-1)}_R, \theta) \text{ using the simulation smoothing by Durbin and Koopman (2002)} \\
3: & \text{draw } \Sigma^{(s)}_R \text{ from } \Pr(\Sigma_R | \eta^{(s)}_{1:t^*}, \theta) \text{ according to equation (13)} \\
4: & \text{compute } \mathbf{Y}^{(s)}_{1:t^*} \text{ and draw } \varrho^{(s)}_p \text{ from } \Pr(\varrho_p | \mathbf{Y}^{(s)}_{1:t^*}, \mathbf{q}^{(s)}_p, \theta) \text{ by changing } \varrho_p \text{ one component at a time and computing its posterior probability (this ensures that every time a component } \varrho_p \text{ is changed, the most likely model is retained, i.e. either the one with } X_p \text{ in or the one without } X_p) \\
5: & \text{draw } \Sigma^{(s)}_\epsilon \text{ from } \Pr(\Sigma_\epsilon | \mathbf{Y}^{(s)}_{1:t^*}, \mathbf{q}^{(s)}_p, \theta) \text{ according to equation (12)} \\
6: & \text{draw } \beta^{(s)}_\theta \text{ from } \Pr(\beta_\theta | \mathbf{Y}^{(s)}_{1:t^*}, \Sigma^{(s)}_\epsilon, \mathbf{q}^{(s)}_p, \theta) \text{ according to equation (11)} \\
7: & \text{end for}
\end{align*}
B.2 Unbiased causal effects

**Theorem 1** For a positive integer \( k \), define \( \hat{Y}_{t+k}(\mathbf{w}) = E[\Pr(Y_{t+k}(\mathbf{w})|Y_{t}(\mathbf{w}))] \) and \( \hat{Y}_{t+k}(\tilde{\mathbf{w}}) = E[\Pr(Y_{t+k}(\tilde{\mathbf{w}})|Y_{t}(\mathbf{w}))] \); under model (9), \( \hat{Y}_{t+k}(\mathbf{w}) \) and \( \hat{Y}_{t+k}(\tilde{\mathbf{w}}) \) are the k-step ahead forecast of \( Y_{t+k}(\mathbf{w}) \) and \( Y_{t+k}(\tilde{\mathbf{w}}) \) given the information set up to time \( t^* \), \( \mathcal{I}_{t^*} = \{ Y_{1:t^*}, X_{1:t^*} \} \). Then, \( \tau_{t+k}(\mathbf{w}, \tilde{\mathbf{w}}) = Y_{t+k}(\mathbf{w}) - Y_{t+k}(\tilde{\mathbf{w}}) \) is the point estimator of the general causal effect and, conditionally on \( \mathcal{I}_{t^*} \) we have,

\[
\begin{align*}
\tau_{t+k}(\mathbf{w}, \tilde{\mathbf{w}}) - \hat{\tau}_{t+k}(\mathbf{w}, \tilde{\mathbf{w}}) &\sim N(0, \Sigma_{\mathbf{w}} + \Sigma_{\tilde{\mathbf{w}}}) \\
\Delta_{t+k} - \hat{\Delta}_{t+k} &\sim N(0, \Sigma_{D(\mathbf{w})} + \Sigma_{D(\tilde{\mathbf{w}})}) \\
\hat{\tau}_{t+k}(\mathbf{w}, \tilde{\mathbf{w}}) - \hat{\tau}_{t+k}(\mathbf{w}, \tilde{\mathbf{w}}) &\sim N\left(0, \frac{1}{k^2}(\Sigma_{D(\mathbf{w})} + \Sigma_{D(\tilde{\mathbf{w}})}) \right)
\end{align*}
\]

where, \( \Sigma_{\mathbf{w}} = \text{Var}\left[Y_{t+k}(\mathbf{w}) - \hat{Y}_{t+k}(\mathbf{w}) | \mathcal{I}_{t^*}\right] \), \( \Sigma_{D(\mathbf{w})} = \text{Var}\left[\sum_k (Y_{t+k}(\mathbf{w}) - \hat{Y}_{t+k}(\mathbf{w})) | \mathcal{I}_{t^*}\right] \) with \( \mathbf{w} \in \{ \mathbf{w}, \tilde{\mathbf{w}} \} \) are defined as follows.

\[
\Sigma_{\mathbf{w}} = Z_t P_t Z'_t + \Sigma_{\epsilon}
\]

\[
\Sigma_{D(\mathbf{w})} = \left(D_{t+1}P_{t+1}D_{t+1}' + \sum_k (D_{t+k}R_{t+k-1}C_{t+k-1}R_{t+k-1}'D_{t+k}' \right) + KH_t
\]

and

\[
\begin{align*}
D_{t+k} &= Z_{t+k} + D_{t+k-1}T_{t+k-1} + k = 1, \ldots, K - 1 \\
D_{t+k} &= Z_{t+k}
\end{align*}
\]

**Proof.**

The difference between the general causal effect and its estimator can be written as,

\[
\begin{align*}
\tau_{t+k}(\mathbf{w}, \tilde{\mathbf{w}}) - \hat{\tau}_{t+k}(\mathbf{w}, \tilde{\mathbf{w}}) &= Y_{t+k}(\mathbf{w}) - Y_{t+k}(\tilde{\mathbf{w}}) - \left[\hat{Y}_{t+k}(\mathbf{w}) - \hat{Y}_{t+k}(\tilde{\mathbf{w}})\right] \\
&= \underbrace{Y_{t+k}(\mathbf{w}) - \hat{Y}_{t+k}(\mathbf{w})}_{A} - \underbrace{Y_{t+k}(\tilde{\mathbf{w}}) - \hat{Y}_{t+k}(\tilde{\mathbf{w}})}_{B}
\end{align*}
\]

Let’s focus our attention on \( A \) and define \( \mathbf{a}_{t+k} = E[\mathbf{a}_{t+k} | \mathcal{I}_{t^*}] \) and \( \mathbf{P}_{t+k} = \text{Var}[\mathbf{a}_{t+k} | \mathcal{I}_{t^*}] \). Under model (9) we have,

\[
\begin{align*}
Y_{t+k}(\mathbf{w}) - \hat{Y}_{t+k}(\mathbf{w}) &= Z_{t+k}\mathbf{a}_{t+k} + X_{t+k}\mathbf{\beta} + \varepsilon_{t+k} - E[Y_{t+k}(\mathbf{w}) | \mathcal{I}_{t^*}] \\
&= Z_{t+k}\mathbf{a}_{t+k} + X_{t+k}\mathbf{\beta} + \varepsilon_{t+k} - Z_{t+k}\mathbf{a}_{t+k} - X_{t+k}\mathbf{\beta} \\
&= Z_{t+k}\mathbf{a}_{t+k} - Z_{t+k}\mathbf{a}_{t+k} + \varepsilon_{t+k}
\end{align*}
\]
Then,

\[
E[Y_{t^*+k}(w) - \hat{Y}_{t^*+k}(w)|I_{t^*}] = 0
\]

\[
\text{Var}[Y_{t^*+k}(w) - \hat{Y}_{t^*+k}(w)|I_{t^*}] = Z_{t^*+k}P_{t^*+k}Z'_{t^*+k} + \Sigma_e = \Sigma_w
\]

Following the exact same steps for B we can show that \( Y_{t^*+k}(\tilde{w}) - \hat{Y}_{t^*+k}(\tilde{w}) \sim N(0, \Sigma_{\tilde{w}}) \). Since the potential paths are independent of each other, relation (23) follows from the properties of the difference of two independent multivariate Normal random variables.

Based on the above result, we can easily show that the expectation of the difference between the cumulative effect and its estimator is zero. In what follows we derive the proof for \( t' = t^* + K \) but it could be shown for every \( k = 1, \ldots, K \).

\[
E \left[ \Delta_{t^*+K} - \hat{\Delta}_{t^*+K} \bigg| I_{t^*} \right] = E \left[ \sum_{k=1}^{K} (\tau_{t^*+k}(w, \tilde{w}) - \hat{\tau}_{t^*+k}(w, \tilde{w})) \bigg| I_{t^*} \right] = 0
\]

The derivation of the variance may be somewhat more cumbersome, because the time dependency also come into play. So we have three dependence structures to take into account: the one between the \( d \) series, the one between times and the one between the states. To address this issue it is useful to re-define \( \varepsilon_t \sim N(0, H_t, \Sigma) \); in this way, \( \varepsilon_t \) can be seen as a single-row matrix following a matrix Normal distribution, which is in line with the definition provided in Section 3. Thus, we have

\[
\text{Var} \left[ \Delta_{t^*+K} - \hat{\Delta}_{t^*+K} \bigg| I_{t^*} \right] = \text{Var} \left[ \sum_{k=1}^{K} (Y_{t^*+k}(w) - \hat{Y}_{t^*+k}(w)) \bigg| I_{t^*} \right] = \text{Var} \left[ \sum_{k=1}^{K} (Y_{t^*+k}(w) - \hat{Y}_{t^*+k}(w)) \bigg| I_{t^*} \right] + KH_t
\]

Focusing on the first term,

\[
\text{Var} \left[ \sum_{k=1}^{K} (Y_{t^*+k}(w) - \hat{Y}_{t^*+k}(w)) \bigg| I_{t^*} \right] = \text{Var} \left[ \sum_{k=1}^{K} Z_{t^*+k}a_{t^*+k} + \sum_{k=1}^{K} \varepsilon_{t^*+k} \bigg| I_{t^*} \right] = \text{Var} \left[ \sum_{k=1}^{K} Z_{t^*+k}a_{t^*+k} \bigg| I_{t^*} \right] + KH_t
\]

where,
Finally, applying the usual properties of variance we obtain relation (25) for the temporal average causal effect, where the variance of the intervention.

This yields to the final result in equation (27). Repeating these steps for the second term we obtain equation (24). Finally, applying the usual properties of variance we obtain relation (25) for the temporal average causal effect,

\[ \text{Var} \left[ \sum_{k=1}^{K} Z_{t^*+k} \alpha_{t^*+k} \right] = \text{Var} \left[ Z_{t^*+1} \alpha_{t^*+1} + Z_{t^*+2} \alpha_{t^*+2} + \cdots + Z_{t^*+K} \alpha_{t^*+K} \right] \]

Thus, in general we have

\[ D_{t^*+k} = Z_{t^*+k} + D_{t^*+k+1} T_{t^*+1} \]

and

\[ D_{t^*+K} = Z_{t^*+K} \]

This yields to the final result in equation (27). Repeating these steps for the second term we obtain equation (24). Finally, applying the usual properties of variance we obtain relation (25) for the temporal average causal effect,

\[ \text{Var} \left[ \hat{\tau}_{t^*+K}(w, \tilde{w}) - \hat{\tau}_{t^*+K}(w, \tilde{w}) \right] = \text{Var} \left[ \frac{1}{K} \sum_{k=1}^{K} \tau_{t^*+k}(w, \tilde{w}) - \frac{1}{K} \sum_{k=1}^{K} \tilde{\tau}_{t^*+k}(w, \tilde{w}) \right] \]

Theorem 1, states that the point estimator of the general causal effect and, by extension, the marginal and the conditional causal effect estimators are unbiased. From equation (27) we can infer that the variance of the difference between the cumulative effect and its estimator increases with the variance of both \( \varepsilon_t \) and \( \eta_t \). Furthermore, the variance is an increasing function of \( D_t \), therefore, our uncertainty increases with time, reflecting our intuition that we have less information about potential outcomes that are further from the time of the intervention.
B.3 Posterior predictive checks

To produce reliable causal effect estimates from the model-based predictions, the assumed model has to adequately describe the data. One way to check the quality of the model fit within a Bayesian framework is to use posterior predictive checks (Rubin, 1981, 1984; Gelman et al., 2013). Intuitively, this entails generating synthetic data sets from the fitted model and comparing them to the observed data.

Typically, we generate replicated data by drawing multiple times from the posterior predictive distribution; then, we compare these draws with the observed data using both numerical and graphical checks (Gelman et al., 2013). More specifically, let $T(Y_{1:t^*}, \theta)$ be a test quantity that depends on the data and the unknown model parameters and denote with $Y_{1:t^*}^{\text{new}}$ a new vector of observations sampled from the posterior predictive distribution, as outlined in equation (14). To describe the degree of the discrepancy, we use the Bayesian $p$-value, which is the probability of observing a test quantity at least as extreme as the observed data, $T(Y_{1:t^*}^{\text{new}}, \theta)$, we denote this by

$$p_B = \Pr(T(Y_{1:t^*}^{\text{new}}, \theta) \geq T(Y_{1:t^*}, \theta) | Y_{1:t^*}).$$  

Unlike in frequentist statistics where a $p$-value near 0 indicates that the corresponding null hypothesis can be rejected, an extreme Bayesian $p$-value denotes that the specific feature of the data captured by the test quantity is inconsistent with the assumed model. For example, if we suspect that our model may not be able to reproduce the large values observed in the data, a suitable test quantity could be the observations’ maximum. In this case, a $p$-value near 0 indicates that, under the assumed model, it is unlikely to encounter a value larger than the observed maximum; so, if the replicated data were generated under a Normal model, a heavy tail distribution may actually be more appropriate. A Bayesian $p$-value can be estimated by computing the proportion of replicated data sets satisfying (28).

We can also provide a graphical representation by plotting the distribution of the test quantity against the observed test quantity; as in a classical setting, the Bayesian $p$-value is the right tail-area probability. Another graphical check consists of computing the posterior predictive mean (i.e., the mean of the posterior predictive distribution) and then plotting it against the distribution of the observed data. Generally, graphical model checks are useful for highlighting the systematic discrepancies between the observed and the simulated data.

Finally, for both linear and non-linear regression models, we can also assess the goodness of fit using residual plots. We can think of Bayesian model residuals as a generalization of classical residuals that accounts for the uncertainty in the model parameters.

In Section 5, we extensively used posterior predictive checks to select and validate the model used for our empirical analysis.
B.4 Sensitivity analysis

Model validation performed through posterior predictive checks shows that the structural time series model with a trend and seasonal component adequately describe the data (see A.2 and A.3 for the details). Nonetheless, posterior inference might still be affected by prior assumptions. Thus, to strengthen our confidence in the assumed model, we performed a sensitivity analysis in order to evaluate to what extent our inferred causal effect changes to different values of the prior hyperparameters.

As described in Section 3.1.1, for the unknown scale matrices of the Inverse-Wishart distributions we chose the following variance-covariance matrix,

\[
S_\varepsilon = S_r = \begin{bmatrix}
hs_1^2 & \sqrt{hk}s_1s_2\rho \\
\sqrt{hk}s_1s_2\rho & ks_2^2
\end{bmatrix},
\]

where, \( s_1^2, s_2^2 \) are the sample variances, which can be scaled by some positive values \( h \) and \( k \), and \( \rho \) is the correlation coefficient. Linking the scale matrix to the sample variances is in line with an objective Bayesian approach and can ensure a reasonable scale for the prior (Brodersen et al., 2015). In our empirical analysis, we set \( h = k = 1 \) but we could have used different values. For example, since the sample variance of the competitor brands is, on average, ten times higher than the sample variance of the store brands, another reasonable scaling can be obtained by setting \( h = 0.1, k = 1 \). Table 7 presents the estimated causal effects under different assumptions for the scaling factors.

Another parameter that can influence our posterior inference is the linear correlation coefficient. We set \( \rho = -0.8 \) based on our prior belief that the two products in the pair are perfect substitutes, but the correlation might be smaller than what assumed or even positive. Table 8 shows the estimated causal effects under different combinations of the correlation and the scaling factors.

Finally, we assumed \( S_\varepsilon = S_r \) but we can also allow the state disturbances to vary more (less) freely than the observation disturbances. The estimated effects under different assumptions for \( S_r \) are reported in Table 9.

Overall, our estimates seem to be robust to different prior assumptions: even if in some instances we find only one or two significant effects, this still supports our general conclusion that the new price policy was not effective.
Table 7: Temporal average general effect estimates at one month horizon under different prior assumptions for the scaling factors \( h \) and \( k \).

<table>
<thead>
<tr>
<th></th>
<th>( h = 1, k = 0.01 )</th>
<th>( h = 1, k = 0.1 )</th>
<th>( h = 1, k = 1 )</th>
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<tbody>
<tr>
<td>( \hat{\tau}_t )</td>
<td>2.5%</td>
<td>97.5%</td>
<td>2.5%</td>
</tr>
<tr>
<td>(1) s</td>
<td>7.24</td>
<td>-23.37</td>
<td>36.89</td>
</tr>
<tr>
<td>(2) c</td>
<td>23.55</td>
<td>-126.97</td>
<td>178.70</td>
</tr>
<tr>
<td>(3) s</td>
<td>7.46</td>
<td>-15.24</td>
<td>29.66</td>
</tr>
<tr>
<td>(4) c</td>
<td>13.57</td>
<td>-76.17</td>
<td>100.46</td>
</tr>
<tr>
<td>(5) s</td>
<td>47.19</td>
<td>0.25</td>
<td>94.40</td>
</tr>
<tr>
<td>(6) c</td>
<td>23.55</td>
<td>-126.97</td>
<td>178.70</td>
</tr>
<tr>
<td>(7) s</td>
<td>7.46</td>
<td>-15.24</td>
<td>29.66</td>
</tr>
<tr>
<td>(8) c</td>
<td>13.57</td>
<td>-76.17</td>
<td>100.46</td>
</tr>
<tr>
<td>(9) s</td>
<td>47.19</td>
<td>0.25</td>
<td>94.40</td>
</tr>
<tr>
<td>(10) c</td>
<td>23.55</td>
<td>-126.97</td>
<td>178.70</td>
</tr>
</tbody>
</table>

\( h = 0.1, k = 0.01 \)  |  \( h = 0.1, k = 0.1 \)  |  \( h = 0.1, k = 1 \)  |
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<thead>
<tr>
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</thead>
<tbody>
<tr>
<td>( \hat{\tau}_t )</td>
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<td>97.5%</td>
</tr>
<tr>
<td>(1) s</td>
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<td>-19.49</td>
</tr>
<tr>
<td>(2) c</td>
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<td>-122.41</td>
</tr>
<tr>
<td>(3) s</td>
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<td>-11.29</td>
</tr>
<tr>
<td>(4) c</td>
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<td>-16.30</td>
</tr>
<tr>
<td>(5) s</td>
<td>40.54</td>
<td>-9.93</td>
</tr>
<tr>
<td>(6) c</td>
<td>15.91</td>
<td>-31.49</td>
</tr>
<tr>
<td>(7) s</td>
<td>12.39</td>
<td>0.81</td>
</tr>
<tr>
<td>(8) c</td>
<td>0.06</td>
<td>-9.02</td>
</tr>
<tr>
<td>(9) s</td>
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<td>2.69</td>
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<tr>
<td>(10) c</td>
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<td>0.81</td>
</tr>
<tr>
<td>(18) c</td>
<td>0.06</td>
<td>-9.02</td>
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Table 8: Temporal average general effect estimates at one month horizon under different prior assumptions for the scaling factors $h$, $k$ and the linear correlation coefficient $\rho$.

<table>
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<tr>
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<th></th>
<th>$h = 1$, $k = 1$, $\rho = -0.3$</th>
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<th>$h = 0.1$, $k = 1$, $\rho = +0.3$</th>
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<td></td>
<td>$\hat{\tau}_t$ 2.5% 97.5%</td>
<td></td>
<td>$\hat{\tau}_t$ 2.5% 97.5%</td>
</tr>
<tr>
<td></td>
<td>c 23.73 -116.98 167.21</td>
<td></td>
<td>c 24.14 -99.19 152.88</td>
<td></td>
<td>20.89 -101.96 144.23</td>
</tr>
<tr>
<td></td>
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<td></td>
<td>c 14.61 -66.66 93.83</td>
<td></td>
<td>15.61 -66.66 93.83</td>
</tr>
<tr>
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<td>s 7.70 -15.19 30.47</td>
<td></td>
<td>s 7.94 -13.96 32.43</td>
<td></td>
<td>8.03 -12.18 30.54</td>
</tr>
<tr>
<td></td>
<td>c 14.52 -69.13 93.31</td>
<td></td>
<td>c 15.36 -60.10 91.26</td>
<td></td>
<td>15.07 -60.56 90.46</td>
</tr>
<tr>
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<td>48.20 3.77 96.39</td>
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<tr>
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<td></td>
<td>24.14 -99.19 152.88</td>
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<td>20.89 -101.96 144.23</td>
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<tr>
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<td>c 14.61 -66.66 93.83</td>
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<td>15.61 -66.66 93.83</td>
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<tr>
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<td>s 7.94 -13.96 32.43</td>
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<td>8.03 -12.18 30.54</td>
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<td>15.07 -60.56 90.46</td>
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<tr>
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<td>s 9.55 -12.56 31.58</td>
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<td>9.38 -13.30 32.70</td>
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<td></td>
<td>c 24.86 -54.29 155.36</td>
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<td>47.98 -52.36 154.19</td>
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<tr>
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<td>48.05 4.46 97.81</td>
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<td>24.14 -99.19 152.88</td>
<td></td>
<td>20.89 -101.96 144.23</td>
</tr>
</tbody>
</table>

Table 9: Temporal average general effect estimates at one month horizon for $h = k = 1$, $\rho = 1$ and under different prior assumptions for $S_r$.

<table>
<thead>
<tr>
<th></th>
<th>$S_r = 0.5S_x$</th>
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<th>$S_r = 10S_x$</th>
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<td></td>
<td>$\hat{\tau}_t$ 2.5% 97.5%</td>
<td></td>
<td>$\hat{\tau}_t$ 2.5% 97.5%</td>
</tr>
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<td>s 6.90 -23.91 37.77</td>
<td></td>
<td>7.10 -26.11 39.83</td>
<td></td>
<td>7.09 -34.28 46.37</td>
</tr>
<tr>
<td></td>
<td>c 23.86 -102.42 152.62</td>
<td></td>
<td>24.79 -101.46 154.68</td>
<td></td>
<td>25.29 -115.70 171.43</td>
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<td></td>
<td>7.06 -16.70 31.65</td>
<td></td>
<td>7.26 -24.02 37.26</td>
</tr>
<tr>
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<td></td>
<td>14.81 -65.48 97.76</td>
<td></td>
<td>15.80 -72.62 110.79</td>
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<td>7.94 -15.49 34.35</td>
<td></td>
<td>8.05 -22.00 41.74</td>
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<tr>
<td></td>
<td>c 15.09 -63.38 91.06</td>
<td></td>
<td>15.65 -62.26 91.39</td>
<td></td>
<td>15.68 -73.12 103.23</td>
</tr>
<tr>
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<td>47.62 2.24 98.14</td>
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<tr>
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<td></td>
<td>29.77 -78.38 136.75</td>
<td></td>
<td>29.36 -90.11 152.06</td>
</tr>
<tr>
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<td></td>
<td>3.32 -47.07 55.74</td>
<td></td>
<td>5.33 -52.97 67.21</td>
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<tr>
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<td>49.28 -58.20 163.80</td>
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<td>46.12 -81.66 170.30</td>
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<tr>
<td></td>
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<td>25.84 -38.52 94.63</td>
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<td>26.15 -47.89 102.64</td>
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<tr>
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<td></td>
<td>78.39 -0.28 156.90</td>
<td></td>
<td>82.02 -9.49 180.16</td>
</tr>
<tr>
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<td></td>
<td>181.14 -233.10 602.97</td>
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<td>169.11 -307.08 619.20</td>
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<td>25.79 -30.32 81.06</td>
<td></td>
<td>26.76 -37.56 90.71</td>
</tr>
<tr>
<td></td>
<td>c 15.90 -14.53 47.33</td>
<td></td>
<td>16.08 -15.65 49.14</td>
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<td>16.38 -21.24 56.59</td>
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<tr>
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<td>16.84 -36.00 69.79</td>
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<td>17.26 -45.31 79.88</td>
</tr>
<tr>
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<td>12.61 1.34 24.15</td>
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<td>12.71 -0.72 25.90</td>
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<tr>
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<td>-0.09 -9.77 9.99</td>
<td></td>
<td>0.05 -9.42 9.85</td>
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</table>
B.5 Convergence diagnostics

To make inference with Markov Chain Monte Carlo (MCMC) methods we need to verify that our Markov chain has converged to the stationary distribution. Geweke’s diagnostic test (Geweke, 1992) compares the sample means of two non-overlapping quantiles of the chain (for example, the first 10% and the last 50% of the draws). If the draws are sampled from the same stationary distribution, the sample means are equal and the test statistic is asymptotically Normal.

Table 10 shows the resulting p-value for the two-sided test for every parameter of the bivariate models estimated on the 10 store-competitor pairs. The Geweke diagnostic fails to detect non-convergence of the chains to the stationary distribution (at the 5% level, the test fails to reject the null hypothesis of the equality of means in 81 cases out of 90).

Finally, for a visual inspection of the chain convergence, we also include the trace plots for the parameters of the first two models (Figures 20 and 21).

Table 10: Geweke’s diagnostics at the lower 10% and upper 50% quantiles. In this table, $\sigma_i^2$, $i \in \{1,2\}$ and $\sigma_{1,2}$ indicate, respectively, the variances and the covariance of the observation disturbances; $\sigma_{\mu_i}^2$ and $\sigma_{\mu_{1,2}}$ the variances and the covariance of the trend disturbances; $\sigma_{\gamma_i}^2$ and $\sigma_{\gamma_{1,2}}$ the variances and the covariance of the disturbances of the seasonal component.

<table>
<thead>
<tr>
<th></th>
<th>$\sigma_1^2$</th>
<th>$\sigma_2^2$</th>
<th>$\sigma_{1,2}$</th>
<th>$\sigma_{\mu_1}^2$</th>
<th>$\sigma_{\mu_2}^2$</th>
<th>$\sigma_{\mu_{1,2}}$</th>
<th>$\sigma_{\gamma_1}^2$</th>
<th>$\sigma_{\gamma_2}^2$</th>
<th>$\sigma_{\gamma_{1,2}}$</th>
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<td>0.75</td>
<td>0.59</td>
<td>0.32</td>
<td>0.71</td>
<td>0.95</td>
<td>0.03</td>
<td>0.03</td>
</tr>
<tr>
<td>2</td>
<td>0.94</td>
<td>0.75</td>
<td>0.25</td>
<td>0.71</td>
<td>0.19</td>
<td>0.55</td>
<td>0.88</td>
<td>0.00</td>
<td>0.01</td>
</tr>
<tr>
<td>3</td>
<td>0.62</td>
<td>0.99</td>
<td>0.64</td>
<td>0.88</td>
<td>0.52</td>
<td>0.80</td>
<td>0.98</td>
<td>0.01</td>
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</tr>
<tr>
<td>4</td>
<td>0.55</td>
<td>0.83</td>
<td>0.96</td>
<td>0.71</td>
<td>0.16</td>
<td>0.67</td>
<td>0.65</td>
<td>0.02</td>
<td>0.07</td>
</tr>
<tr>
<td>5</td>
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<td>0.98</td>
<td>0.65</td>
<td>0.97</td>
<td>0.27</td>
<td>0.73</td>
<td>0.88</td>
<td>0.20</td>
<td>0.30</td>
</tr>
<tr>
<td>6</td>
<td>0.76</td>
<td>0.83</td>
<td>0.81</td>
<td>0.39</td>
<td>0.17</td>
<td>0.98</td>
<td>0.90</td>
<td>0.17</td>
<td>0.28</td>
</tr>
<tr>
<td>7</td>
<td>0.81</td>
<td>0.54</td>
<td>0.16</td>
<td>0.53</td>
<td>0.47</td>
<td>0.04</td>
<td>0.89</td>
<td>0.18</td>
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</tr>
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<td>0.29</td>
<td>0.78</td>
<td>0.47</td>
<td>0.40</td>
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<td>0.83</td>
<td>0.30</td>
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<td>0.79</td>
<td>0.72</td>
<td>0.35</td>
<td>0.04</td>
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</table>
Figure 20: Trace plots of the variance-covariance matrices of the model estimated on the first store-competitor pair.

Figure 21: Trace plots of the variance-covariance matrices of the model estimated on the second store-competitor pair.