Causal Inference with Panel Data under Temporal and Spatial Interference

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Abstract

Many social events and policy interventions generate treatment effects that persistently spill over into neighboring areas, resulting in a phenomenon statisticians refer to as “interference” both in time and space. In this paper, I put forward a design-based framework to identify and estimate these spillover effects in panel data with a spatial dimension, when temporal and spatial interference intertwine in intricate ways that are unknown to researchers. The framework defines estimands that enable researchers to measure the influence of each type of interference, and I propose estimators that are consistent and asymptotically normal under the assumption of sequential ignorability and mild regularity conditions. I show that fixed effects models in panel data analysis, such as the difference-in-differences (DID) estimator, can lead to significant biases in such scenarios. I test the method’s performance on both simulated datasets and the replication of two empirical studies.

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1 Introduction

The objective of many empirical studies in political science is to evaluate the impact of social events or policy interventions. In instances where experiments are not feasible, researchers often resort to panel data analysis and assess treatment effects using fixed effects models such as the difference-in-differences (DID) estimator or the two-way fixed effects (TWFE) model, which control for unobservable confounders that satisfy a certain form. However, the validity of these methods relies on the stable unit treatment value assumption (SUTVA), which stipulates that the treatment status of any observation can only influence its own outcome (Imai and Kim, 2019). In panel data, SUTVA is violated when the treatment of a unit affects either its future outcome (referred to as temporal interference) or the outcomes of other units (referred to as spatial interference). When interference is present, untreated observations are contaminated by the spillover from treated ones and can no longer serve as a valid benchmark for researchers to identify the causal parameter of interest (Cook, Hays and Franzese, 2023). In fact, the familiar estimands under SUTVA, such as the average treatment effect (ATE) or the average treatment effect on the treated (ATT), are not even well-defined in this context.

Consider, for instance, the study by Stokes (2016) that probes into the impact of building wind turbines in Ontario, Canada. Turbines erected in one precinct may stir discontent not only among local residents, but also among those in nearby areas, motivating them to vote for the opposition party in subsequent elections. If these neighboring precincts are employed as the control group, in line with fixed effects models, the estimate will capture a mixture of both the direct effects from the treatment and the spillover effects. Typically, this is not an informative measure for researchers or policy makers. Moreover, identifying the contaminated units or the impact of the spillover is challenging, especially when the effects are heterogeneous across observations. While such scenarios are common in empirical research, there is no consensus in the literature on how to account for or estimate these causal effects driven by interference accurately. Most existing methods are built upon structural restrictions that are neither realistic nor testable.\footnote{For example, the spillover effects only come from contiguous units and are proportional to the percentage of treated...}
In this article, I propose a design-based framework for causal inference in panel data with a spatial dimension, when both temporal and spatial interference exist and intertwine with each other in complex ways that are unknown to researchers. I introduce a group of novel estimands, the average marginalized effects (AMEs), which remain meaningful and well-defined under treatment effect heterogeneity and interference. These AMEs capture the marginal effect of a unit’s treatment assignment history on its neighbors in any period, and help us disentangle the direct effects from the influences of temporal interference and spatial interference. I demonstrate that the AMEs can be nonparametrically identified under the assumption of sequential ignorability, which posits that the current treatment status of each unit is dictated solely by its observable history (Robins, Hernan and Brumback, 2000; Blackwell, 2013; Blackwell and Glynn, 2018; Imai, Kim and Wang, 2021). When the degree of dependence among the observations is mildly limited, they can be consistently estimated by the inverse probability of treatment weighting (IPTW) estimators. I prove the asymptotic normality of these estimators, and provide methods for calculating their variances and constructing confidence intervals.

I then investigate the performance of the fixed effects models in such scenarios, and reveal that the quantities their estimates converge to only hold substantive interpretations under highly restrictive circumstances, such as when treatment effects are homogeneous. I corroborate the theoretical analyses using simulated data, and then put the proposed method into practice to replicate the findings in Wang and Wong (2021) and Stokes (2016). The results of these replication studies highlight the bias of fixed effects models, provide illustrative examples of how to estimate spillover effects across both time and space, and demonstrate the process of validating the research design using placebo outcomes.

This article introduces a novel approach to address interference in panel data with a spatial dimension, a persistent challenge in political methodology (Stimson, 1985). Unlike existing methods (Franzese and Hays, 2007; Acemoglu, García-Jimeno and Robinson, 2015; Ogburn et al., 2020; Butts, 2021; Cook, Hays and Franzese, 2023), this approach does not require knowledge on the ones among them.
interference structure—how the outcome of each observation is decided by the treatment status of others, hence mitigates potential biases arising from misspecification of the outcome model.\textsuperscript{2}

It is motivated by recent advancements in causal inference that aim to understand interference from the design-based perspective (Hudgens and Halloran, 2008; Sinclair, McConnell and Green, 2012; Aronow and Samii, 2017; Papadogeorgou et al., 2020; Sävje, Aronow and Hudgens, 2021). I generalize the AMEs introduced by Wang et al. (2020) for spatial experiments to panel data, showing that they help researchers distinguish various types of interference and sidestep the widely recognized issue of accounting for confounders from neighboring units in spatial statistics (Papadogeorgou, Choirat and Zigler, 2019; Reich et al., 2021).

The paper also contributes to the growing reflections on the biases of fixed effects models when treatment effects are heterogeneous (Strezhnev, 2018; Imai and Kim, 2019; Goodman-Bacon, 2018; Sun and Abraham, 2020; De Chaisemartin and d’Haultfoeuille, 2020), arguing that these biases become even more pronounced in the presence of interference. In contrast, estimators based on sequential ignorability can effectively handle both temporal and spatial interference, although they are incompatible with the existence of unobservable confounders. These discussions underscore the trade-offs researchers must consider when selecting the identification assumption in panel data under interference.

The rest of the paper is organized as follows: Section 2 describes the basic framework and defines the estimands. Section 3 discusses assumptions we need for identification and introduce the estimators. Section 4 demonstrates statistical properties of the proposed estimators. Section 5 is dedicated to analyze the caveats of using fixed effects models under interference. Section 6 explores possible extensions. Sections 7 and 8 present results from simulation and the replication exercise, respectively. Section 9 concludes.

\textsuperscript{2}Although researchers still need to accurately model the treatment assignment process, this task is typically more manageable and can be validated using available data.
2 The framework

2.1 Set up

Consider \( N \) units located on a geography \( \mathcal{X} \) equipped with a metric (e.g., distance). Each unit \( i \) is observed for \( T \) consecutive periods, with \( T \ll N \). In each period \( t \), we observe the outcome \( Y_{it} \) and the treatment status \( Z_{it} \in \{0, 1\} \) for unit \( i \). Throughout the paper, I use uppercase letters for random variables (e.g., \( Y_{it} \)), lowercase letters for their specific value (e.g., \( y_{it} \)), and boldface ones for vectors and matrices. The subscripts indicate a random variable or its value for a particular observation, unit, or period, while the superscripts indicate its history over several periods. For instance, \( Z^{s:t}_i = (Z_{is}, Z_{i,s+1}, \ldots, Z_{it}) \) represents unit \( i \)'s treatment status between period \( s \) and period \( t \), and \( Z^{s:t} = (Z^{s:t}_1, Z^{s:t}_2, \ldots, Z^{s:t}_N) \) represents the treatment assignment history of all the units between the two periods. Additionally, \( Z^{s:t} \setminus Z^{s':t'}_i \) is used to denote the remaining part in \( Z^{s:t} \) after fixing \( Z \)'s value for unit \( i \) between period \( t' \) and period \( s' \), assuming that \( s' \geq s \) and \( t' \leq t \).

Figure 1: A DAG illustration

Notes: The DAG includes two units \( i, j \) and two periods \( t, t + 1 \). Variables are marked by circles and causal paths by arrows. Black circles represent the treatment variable and white circles represent the outcome variable. Black arrows indicate the direct effect from an observation’s own treatment. Red, blue, and purple arrows represent effects driven by temporal interference, by spatial interference, and by both types of interference, respectively. Gray arrows show other relationships that may affect treatment assignment. Its structure is justified by assumptions introduced in Section 3.1.

This framework allows for general interference. The outcome for unit \( i \) in period \( t \), \( Y_{it} \), could be jointly decided by the history of treatment assignment across all the \( N \) units: \( Y_{it} = Y_{it}(Z^{1:T}) = \)
$Y_{it}(Z_{1:1:T}, Z_{2:1:T}, \ldots, Z_{N:1:T})$, whose functional form is unknown to the researcher. Here, $Z_{1:T}$ is an $N \times T$-dimensional vector, indicating that there are $2^{N \times T}$ different potential outcomes for each observation $(i, t)$. In the absence of interference, $Y_{it} = Y_{it}(Z_{it})$ and SUTVA holds. With only temporal interference, $Y_{it} = Y_{it}(Z_{i:1:T}) = Y_{it}(Z_{i1}, Z_{i2}, \ldots, Z_{iT})$; while with only spatial interference, $Y_{it} = Y_{it}(Z_{it}) = Y_{it}(Z_{i1}, Z_{i2}, \ldots, Z_{iN})$. Figure 1 depicts each type of interference using a directed acyclic graph (DAG) with two units and two periods. Assuming the emphasis is on the effect generated by $Z_{it}$, the arrow from it to $Y_{it}$ denotes the direct treatment effect. The arrows from $Z_{it}$ to $Y_{i,t+1}$, $Y_{jt}$, and $Y_{j,t+1}$ represent the spillover effects caused by temporal interference, spatial interference, and both, respectively.

**An illustration** I provide a concrete example for concepts defined above using a simulated dataset, which comprises 400 units observed over 5 periods. The units are represented by tiles in a $20 \times 20$ raster (left plot of Figure 2). The treatment assignment process is specified to follow the structure of staggered adoption, meaning that once a unit is treated, it remains treated in all subsequent periods, and the treatment does not occur until the third period.\(^3\) Note that this structure is imposed solely for illustrative purposes, and more complex histories are compatible with the framework. From the right plot of Figure 2, we can see that there are four distinct treatment assignment histories from period 3 to period 5: $Z_{i:3:5} \in \{(0,0,0), (0,0,1), (0,1,1), (1,1,1)\}$. Considering the outcome of an arbitrary unit $i$ (e.g., unit 42) in any period (e.g., period 5), without further restrictions, its value is a function of the treatment assignment history of all the 400 units from period 1 to period 5: $Y_{42,5} = Y_{42,5}(Z_{1:1:5}, Z_{2:1:5}, \ldots, Z_{400:1:5})$.

### 2.2 Estimands

This paper focuses on the causal effect generated by switching the treatment assignment history between periods $t - k$ and $t$ from $Z_{(t-k):2}$ to $Z_{(t-k):2}$ (e.g., from $(0,0,0)$ to $(0,1,1)$). For unit $j$ in

\(^3\)Details of the data generating process are described in Section 7.
Figure 2: Simulated dataset and treatment assignment

Notes: The left plot shows the locations of the 400 units in the simulated dataset, where the colors indicate the variation of treatment effect heterogeneity and the red circles show the circles around the spotted unit. The right plot depicts the treatment assignment history for the first 20 units in the sample.

period $t$, define the treatment effect from the change in unit $i$’s treatment assignment history as:

$$\tau_{jt;i} \left( z^{(t-k):t}, \tilde{z}^{(t-k):t}; Z^{1:T} \setminus Z^{(t-k):t} \right) \triangleq Y_{jt} \left( z^{(t-k):t}; Z^{1:T} \setminus Z^{(t-k):t} \right) - Y_{jt} \left( \tilde{z}^{(t-k):t}; Z^{1:T} \setminus Z^{(t-k):t} \right).$$

In each of the two potential outcomes, the value of $Z^{(t-k):t}_i$ is fixed whereas $Z^{1:T} \setminus Z^{(t-k):t}_i$, the remaining part in the treatment assignment history of all the units, is not. Therefore, their difference is still a random variable. As the ATE or the ATT are averages of these quantities, they are also random and no longer well-defined. Following the literature, we take expectation over $Z^{1:T} \setminus Z^{(t-k):t}_i$ in the expression above, and obtain the **marginalized individualistic treatment effect**:

$$\tau_{jt;i} \left( z^{(t-k):t}, \tilde{z}^{(t-k):t} \right) \triangleq E \left[ Y_{jt} \left( z^{(t-k):t}; Z^{1:T} \setminus Z^{(t-k):t}_i \right) - Y_{jt} \left( \tilde{z}^{(t-k):t}; Z^{1:T} \setminus Z^{(t-k):t}_i \right) \right].$$

It captures the marginal effect from unit $i$’s treatment assignment history on $j$’s outcome in period $t$, and is analogous to the individualistic treatment effect when SUTVA holds. Such a quantity can be
defined for every combination of $i$, $j$, and $t$.

Next, for each $i$, let’s aggregate $\tau_{j:i} \left( z^{(t-k):t}, \tilde{z}^{(t-k):t} \right)$ over a pre-specified set of $j$ denoted as $\Omega_d$, where $d \in D$ and $D$ is a set of indexes. This gives:

$$\mu_i \left( \left\{ \tau_{j:i} \left( z^{(t-k):t}, \tilde{z}^{(t-k):t} \right) \right\}_{j \in \mathcal{N} ; \Omega_d} \right) := \frac{\sum_{j=1}^{N} 1\{ j \in \Omega_d \} \tau_{j:i} \left( z^{(t-k):t}, \tilde{z}^{(t-k):t} \right)}{\sum_{j=1}^{N} 1\{ j \in \Omega_d \}}, \quad (1)$$

where $1\{ \cdot \}$ is the indicator function. The form of $\Omega_d$ should be chosen by the researcher based on the purpose of the study. One option is the “circle average” introduced by Wang et al. (2020), where $d$ stands for any distance value. $\Omega_d$ can be a circle, where $\Omega_d := \{ j \in \mathcal{N} : d_{ij} = d \}$, a “donut,” where $\Omega_d := \{ j \in \mathcal{N} : d - \kappa < d_{ij} \leq d \}$ and $\kappa$ is a constant, or a “disk,” where $\Omega_d := \{ j \in \mathcal{N} : d_{ij} \leq d \}$, around each $i$. In this case, the quantity defined in (1) indicates the marginal effect from unit $i$’s treatment assignment history on all the other units that are roughly $d$ away (or within the range of $d$) from it. With different values of $d \in D$, it enables us to see how the marginal effect of $i$ varies over distance. For instance, the red spot in Figure 2 indicates the location of unit $i$ and the red circles represent each $\Omega_d$ around it. Both $d$ and $\Omega_d$ can be adjusted for specific contexts. For example, $d$ may measure the traffic accessibility or even the cultural proximity between two units. If the spillover effects on contiguous units are of interest, $\Omega_d$ can be defined as the $d$th degree neighbors of each unit over the geography. $\Omega_d$ will be represented with $d$ when there is no confusion.

**Remark** Note that the definition of $\Omega_d$ does not require any knowledge of the interference structure, that is, the functional form of $Y_{jt}(Z^{1:T})$. Instead of specifying how the outcome of a unit is affected by the treatment of the others—the “exposure mapping” defined first by Aronow and Samii (2017), this paper focuses on how the treatment of a unit makes a difference in expectation. This is why the framework is design-based. It does not assume that any $\Omega_d$ or their collection captures all the influences generated by a unit. The choice of $\Omega_d$ is flexible and not unique.\(^4\)

Overlapping of $\Omega_d$ across the units is also permissible.

\(^4\)But it does have impacts on statistical inference, as discussed in Section 4.
Table 1: Example of AMEs

| $d$ | $z^{3:5}$ | $(0,0,1)$ | $(1,1,1)$ |
|-----|-----------|-----------|-----------|
| $d = 0$ | $\tau_5 ((0,0,1),(0,0,0);0)$ | $\tau_5 ((1,1,1),(0,0,0);0)$ | direct effect |
| $d > 0$ | $\tau_5 ((0,0,1),(0,0,0);d)$ | $\tau_5 ((1,1,1),(0,0,0);d)$ | spatial interference |
|      |           |           | both types of interference |

Finally, taking the average of $\mu_i \left( \left\{ \tau_{j,t;\hat{d}} \left( z^{(t-k):t}, \tilde{z}^{(t-k):t} \right) \right\}_{j \in \mathcal{N};d} \right)$ across all the units leads to:

$$
\tau_t \left( z^{(t-k):t}, \tilde{z}^{(t-k):t};d \right) := \frac{1}{N} \sum_{i=1}^{N} \mu_i \left( \left\{ \tau_{j,t;\hat{d}} \left( z^{(t-k):t}, \tilde{z}^{(t-k):t} \right) \right\}_{j \in \mathcal{N};d} \right). \tag{2}
$$

This estimand is termed the **average marginalized effect** (AME), following the terminology of Wang et al. (2020).\(^5\) It measures the average marginal treatment effect when the treatment assignment history of a unit switches from from $\tilde{z}^{(t-k):t}$ to $z^{(t-k):t}$ between periods $t - k$ and $t$ on its neighbors belonging to $\Omega_d$. The interpretation resembles that of the average marginal component effect (AMCE) in conjoint experiments (Hainmueller, Hopkins and Yamamoto, 2014), but applies to the neighbors of a unit rather than the unit itself. In addition, if there is an effect function varying over $d$ and the effects from different units are additive, then the AME will be equal to the effect function’s average value at $d$, as shown by Wang et al. (2020). Clearly, the AME’s value depends on the treatment assignment histories and the form of $\Omega_d$. When there is a pure control group in which the units are never treated between periods $t - k$ and $t$, $\tilde{z}^{(t-k):t}$ can be set to be the all-zero vector as it is a natural reference point.

**An illustration (continued)** To understand the role played by each type of interference, we can define a series of AMEs with varying values of $z^{(t-k):t}$ and $d$. For the simulated example,

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\(^5\)Much like the ATE under SUTVA, this quantity cannot be computed in practice due to the fundamental problem of causal inference. However, it can be consistently estimated, as demonstrated in the next section.
let’s set $t = 5$, $k = 2$, and $d \in \{0, 1, \ldots, 7\}$ measured by the Euclidean distance. $\Omega_d$ is defined as a circle, as depicted in the left plot of Figure 2. Table 1 presents four different AMEs. When $d = 0$, $\Omega_d$ only includes the unit itself. Under the history $(0, 0, 1)$, the treatment occurs only in period 5. Therefore, $\tau_5 ((0, 0, 1), (0, 0, 0); 0)$ reflects the average “direct effect” of an observation’s treatment on its own outcome.\(^6\) In contrast, $\tau_5 ((1, 1, 1), (0, 0, 0); 0)$ includes both the direct effect and the persistent effect from the treatments in the previous two periods on the outcome. The difference between the two estimands reflects the strength of temporal interference. When $d > 0$, $\tau_5 ((0, 0, 1), (0, 0, 0); d)$ summarizes the spillover effects in space from the contemporaneous treatment, and $\tau_5 ((1, 1, 1), (0, 0, 0); d)$’s value is driven by both temporal and spatial interference. I discuss other possible estimands in Section 6.

I specify an effect function that emanates from each unit and declines in distance (left plot of Figure 3). Effects received by each unit are additive across its neighbors and amplified by an idiosyncratic constant that represents treatment effect heterogeneity (reflected by varying colors in the plot). The effects in each period persist into the next one with a discount factor of 0.6. The assignment process is repeated for 1,000 times, and each $\tau_{jt;i} ((1, 1, 1), (0, 0, 0))$ is approximated by the average difference between $Y_{jt} \left( (1, 1, 1); Z_{1:T} \setminus Z_i^{(t-k):r} \right)$ and $Y_{jt} \left( (0, 0, 0); Z_{1:T} \setminus Z_i^{(t-k):r} \right)$ over the assignments. Each $\tau_{jt;i} ((0, 0, 1), (0, 0, 0))$ is approximated similarly. The AMEs are then constructed in line with their definition in expression (2). I plot the constructed AMEs against the distance values in the right plot of Figure 3. The bottom curve shows $\tau_5 ((0, 0, 1), (0, 0, 0); d)$, while the top one shows $\tau_5 ((1, 1, 1), (0, 0, 0); d)$. As discussed, when $d = 0$, $\tau_5 ((0, 0, 1), (0, 0, 0); d)$ measures the magnitude of the direct effect, which accurately approximates the effect function,\(^7\) and its difference from $\tau_5 ((1, 1, 1), (0, 0, 0); d)$ reveals the influence of temporal interference. When $d > 0$, $\tau_5 ((0, 0, 1), (0, 0, 0); d)$ reflects the influence of solely spatial interference while $\tau_5 ((1, 1, 1), (0, 0, 0); d)$ captures the interaction between the two types of interference.

\(^6\)Note that this is a marginal effect, as those in Hudgens and Halloran (2008) and Sävje, Aronow and Hudgens (2021).

\(^7\)The AME curve is expected to match the effect function given that the effects are additive. The roughness seen in the curve results from the discontinuity at the boundary between two tiles.
Notes: The left plot shows how the effect function that emanates from each unit varies over distance. The right plot presents the estimands, $\tau_5 ((0,0,1), (0,0,0); d)$ and $\tau_5 ((1,1,1), (0,0,0); d)$, over the same range of distance values.

3 Identification and estimation

3.1 Identification

I now state the assumptions that are needed to identify the AMEs.

Assumption 1 (No anticipation). If $Z_i^{1:t} = \tilde{Z}_i^{1:t}$, then

$$Y_{it}(Z_i^{1:T}) = Y_{it}(\tilde{Z}_i^{1:T}).$$

for any $i$ and $t$.

This assumption requires that the potential outcome of any observation $(i, t)$ is not affected by treatments in the future. It is a fairly weak restriction on the interference structure and common in the literature of panel data analysis. It will be violated if the units anticipate the occurrence of the treatment in advance and adjust their behaviors accordingly. Under Assumption 1, $Y_{it}(Z_i^{1:T})$ can
written as \( Y_{it}(Z_{1:t}) \).

**Assumption 2** (Sequential ignorability).

\[
Z_{it} \perp \{ Y_{jt}(z_t; Z_{1:t}) \}_{j=1}^N | Z_{1:(t-1)}, Y_{1:(t-1)}, X_{1:t},
\]

\[
Z_{i1} \perp \{ Y_{j1}(z_1; Z_1 \setminus \{Z_{i1}\}) \}_{j=1}^N | X_1,
\]

and \( \eta < P(Z_{1:t} = z_{1:t}) < 1 - \eta \), where \( \eta > 0 \), for any \( z_{1:t} = (z_1, z_2, \ldots, z_t) \), \( i, j, \) and \( t \).

This is the most crucial assumption for the method to work. It stipulates that the treatment assignment for unit \( i \) in period \( t \), \( Z_{it} \), is as if randomized conditional on past treatment assignments, past outcomes, and covariates that are not affected by \( Z_{it} \). The requirement of positivity is also imposed, meaning that each treatment assignment history of interest should occur with a probability that falls strictly between 0 and 1. The assumption is a variant of “selection on observables” in cross-sectional studies. It implies that the data are generated by a (hypothetical) dynamic experiment (Blackwell and Glynn, 2018) and the information contained in history dictates the current probability of being treated, the propensity score \( P(Z_{it} = 1|V_{it}) \). Here \( V_{it} \) represents the set of confounders—variables in \( (Z_{1:(t-1)}, Y_{1:(t-1)}, X_{1:t}) \) that affect the outcome and the treatment assignment process in period \( t \). Under the first two assumptions, we can write the propensity score for each treatment assignment history of interest, \( P\left( Z_{i(t-k):t} = z_{(t-k):t} \right) \), as \( \prod_{s=t-k}^{t-1} P(Z_{is} = z_s|V_{is}) \). Unlike the assumption behind the fixed effects models (often known as strict exogeneity), sequential ignorability does not permit the existence of unobservable confounders. I further elaborate the trade-offs when choosing between the two assumptions in Section 5.

To facilitate the estimation of the propensity scores, I impose an extra restriction on \( V_{it} \):

**Assumption 3** (No contagion). For any \( i \) and \( t \), \( P(\left. Z_{i(t-k):t} = z_{(t-k):t} \right) \) is decided only by unit \( i \)’s own history.

By assuming no contagion, the causal paths from \( (Z_{1:(t-1)}, Y_{1:(t-1)}, X_{1:t}) \) to \( Z_{it} \) for \( j \neq i \) is blocked. It reduces the dimension of \( V_{it} \) dramatically. In practice, we may further restrict what
variables are included in $V_{it}$, such as only the history from the previous $f$ periods serves as confounders. Note that neither Assumption 3 nor these restrictions are likely to be realistic in conventional approaches. To correctly model how the outcome of each unit is affected by its neighbors, all the factors that influence the treatment status of the neighbors must be accounted for. Even when the treatment is independently assigned across the units, $V_{it}$ will contain variables from others. We avoid this challenge of handling “spatial confounding” (Papadogeorgou, Choirat and Zigler, 2019; Reich et al., 2021) by taking the design-based perspective and focusing the effect generated by each unit rather than the causes of its outcome. Assumptions 1 - 3 justify the structure of the DAG in Figure 1, which will be more complicated without any of them. For example, there will exist an arrow from $Z_{i,t+1}$ to $Y_{it}$ or an arrow from $Z_{jt}$ to $Z_{i,t+1}$. I explore consequences of lifting Assumption 1 or 3 in Section 6.

Under Assumptions 1 - 3, the AME is identifiable from data:

$$ \tau_t \left( z^{(t-k):t}, \tilde{z}^{(t-k):t} ; d \right) = \text{E} \left[ \frac{1}{N} \sum_{i=1}^{N} \mathbf{1} \left\{ Z^{(t-k):t}_i = z^{(t-k):t} \right\} \mu_i \left( \left\{ Y_{jt} \right\}_{j \in N} ; d \right) \right] - \text{E} \left[ \frac{1}{N} \sum_{i=1}^{N} \mathbf{1} \left\{ Z^{(t-k):t}_i = \tilde{z}^{(t-k):t} \right\} \mu_i \left( \left\{ Y_{jt} \right\}_{j \in N} ; d \right) \right], $$

where $\mu_i \left( \left\{ Y_{jt} \right\}_{j \in N} ; d \right) = \frac{\sum_{j=1}^{N} 1 \left\{ j \in \Omega_d \right\} Y_{jt}}{\sum_{j=1}^{N} 1 \left\{ j \in \Omega_d \right\}}$ is the average outcome across $i$’s neighbors that belong to $\Omega_d$ and denoted as the transformed outcome of unit $i$. The result indicates that each AME equals the marginal or expected difference between two sample averages over the transformed outcome. Intuitively, after re-weighed by $\mathbf{1} \left\{ Z^{(t-k):t}_i = \tilde{z}^{(t-k):t} \right\} / P \left( Z^{(t-k):t}_i = z^{(t-k):t} \right)$, each observed outcome $Y_{jt}$ is an unbiased estimate of the marginalized outcome $\text{E} \left[ Y_{jt} \left( z^{(t-k):t} ; Z^{(t-k):t} \setminus Z^{(t-k):t}_i \right) \right]$, then the equality holds due to the linearity of $\mu_i(\cdot)$. The full proof can be found in the appendix.

In practice, the validity of Assumptions 1 - 3 hinges on researchers’ ability to identify the correct set of confounders. To validate their choice, researchers may attempt to find a placebo outcome.
that should not be affected by the treatment. For instance, if there are two pre-treatment periods, 1
and 2, and we believe that the treatment assignment is only influenced by the outcome from the
previous period, then the same DAG in Figure 1 would apply to observations from these two periods,
excluding arrows from any $Z$ to any $Y$ (since the treatment has not occurred). We can then use the
methods outlined in the following section to estimate the AME on the outcome from period 2. The
presence of any significant estimate would suggest that the outcome is being influenced by variables
not specified in the DAG, which violates the identification assumptions.

### 3.2 Estimators

The result on identification in the previous subsection leads to a natural estimator for the AME—the
difference in the quantities under the expectation sign:

$$
\hat{\tau}_t(z^{(t-k):t}, \tilde{z}^{(t-k):t}; d) = \frac{1}{N} \sum_{i=1}^{N} \mathbb{1}\left\{Z_i^{(t-k):t} = z^{(t-k):t}\right\} \mu_i \left(\left\{Y_{jt}\right\}_{j \in \mathcal{N}}; d\right)
- \frac{1}{N} \sum_{i=1}^{N} \mathbb{1}\left\{Z_i^{(t-k):t} = \tilde{z}^{(t-k):t}\right\} \mu_i \left(\left\{Y_{jt}\right\}_{j \in \mathcal{N}}; d\right).
$$

It takes the form of the Horvitz-Thompson estimator, one of the IPTW estimators. Note that
the propensity scores are unknown, requiring estimates to be incorporated into the expression.
Researchers may rely on a simple logistic model or more complex techniques such as the sieve
estimator (Hirano, Imbens and Ridder, 2003) or the method of covariate balancing propensity score
(Imai and Ratkovic, 2015) for the estimation of the propensity scores. The impact of this choice is
further discussed in the appendix.

The Horvitz-Thompson estimator may exhibit a large variance in practice, especially when the
estimated propensity scores contain values that are close to 0 or 1. The estimates can be made more
stable by utilizing the Hajek estimator (Hájek, 1964), which replaces the denominator $N$ with the
weighted sum of the treatment indicator: \[ \hat{\tau}_{t,HA}(z^{(t-k):t}, \tilde{z}^{(t-k):t}, d) = \frac{\sum_{i=1}^{N} 1 \{ Z_i^{(t-k):t} = z^{(t-k):t} \} \mu_i \left( \{ Y_{jt} \}_{j \in \mathcal{A}} ; d \right) / \hat{P} \left( Z_i^{(t-k):t} = z^{(t-k):t} \right)}{\sum_{i=1}^{N} 1 \{ Z_i^{(t-k):t} = \tilde{z}^{(t-k):t} \} / \hat{P} \left( Z_i^{(t-k):t} = \tilde{z}^{(t-k):t} \right)} - \frac{\sum_{i=1}^{N} 1 \{ Z_i^{(t-k):t} = \tilde{z}^{(t-k):t} \} \mu_i \left( \{ Y_{jt} \}_{j \in \mathcal{A}} ; d \right) / \hat{P} \left( Z_i^{(t-k):t} = \tilde{z}^{(t-k):t} \right)}{\sum_{i=1}^{N} 1 \{ Z_i^{(t-k):t} = \tilde{z}^{(t-k):t} \} / \hat{P} \left( Z_i^{(t-k):t} = \tilde{z}^{(t-k):t} \right)}. \]

In the appendix, I show that both estimators are asymptotically unbiased and converge to normal distributions. However, the Hajek estimator is always more efficient. Another benefit of the Hajek estimator is that it admits a regression representation (Samii and Aronow, 2012), which simplifies implementation and hypothesis testing. In the appendix, I deduce the expression of the Hajek estimator’s variance and demonstrate that it can be estimated using the spatial heteroscedasticity and auto-correlation consistent (HAC) variance estimator (Conley, 1999) in regression analysis. The variance estimator requires researchers to calculate the distance between any pair of units and specify a cutoff value \( \tilde{d} \). For two units whose distance is beyond \( \tilde{d} \), there will be no dependence between their transformed outcomes. In practice, researchers can decide the value of \( \tilde{d} \) using substantive knowledge, or first estimate \( \tau_t(z^{(t-k):t}, \tilde{z}^{(t-k):t}, d) \) for a range of distance values and set \( \tilde{d} \) to be two times the distance value where the effect becomes negligible. I provide theoretical justifications for this choice in the next section.

I summarize the implementation of the proposed method in Algorithm 1 below. Step 4 in the algorithm is inspired by the regression representation of the Hajek estimator. The regression coefficient \( \hat{\tau}_t(d) \) will be numerically equivalent to \( \hat{\tau}_{t,HA}(z^{(t-k):t}, \tilde{z}^{(t-k):t}, d) \). From the outcome-based perspective, the regression equation is evidently misspecified as it does not control for the effects from other units. However, as shown in the subsequent section, properly weighting the units ensures that the regression coefficient converges to the estimand \( \tau_t(z^{(t-k):t}, \tilde{z}^{(t-k):t}, d) \). In the appendix, I demonstrate that the IPTW estimators can be augmented by employing outcome models to more accurately approximate the transformed outcome (Glynn and Quinn, 2010). As
such, outcome-based approaches (Acemoglu, García-Jimeno and Robinson, 2015; Cook, Hays and Franzese, 2023) can be incorporated into the proposed framework. Steps 5 to 7 are justified in the next section. Step 8 is based on previous discussions that the identification assumptions can be validated by applying the estimators to a placebo outcome.

Algorithm 1: Implementation of the method

1. For any period $t$, choose the length of the history $k$, the metric $d$, the form of $\Omega_d$, and the set of confounders $V_{it}$, then obtain the distance matrix $\{d_{ij}\}_{N \times N}$ between all units $i$ and $j$.

2. For each unit $i$, construct the transformed outcome $\mu_i \left( \{Y_{jt}\}_{j \in \mathcal{N}_i} ; d \right) = \frac{\sum_{j=1}^{N} 1\{j \in \Omega_d \} Y_{jt}}{\sum_{j=1}^{N} 1\{j \in \Omega_d \}}$.

3. Select a propensity score model, $P(Z_{it} = 1|V_{it})$, fit it on the sample, and predict the probability for any history $\mathbf{z}^{(t-k):t}$ to occur:

$$\hat{P} \left( Z_{it}^{(t-k):t} = \mathbf{z}^{(t-k):t} \right) = \prod_{s=t-k}^{t} \hat{P}(Z_{is} = z_s | V_{is}).$$

4. Regress $\mu_i \left( \{Y_{jt}\}_{j \in \mathcal{N}_i} ; d \right)$ on $\{Z_{it}^{(t-k):t} = \tilde{\mathbf{z}}^{(t-k):t}\}$ and $\{Z_{it}^{(t-k):t} = \mathbf{z}^{(t-k):t}\}$ without the intercept, using $W_i = \frac{1\{Z_{it}^{(t-k):t} = \mathbf{z}^{(t-k):t}\}}{\hat{P}(Z_{it}^{(t-k):t} = \mathbf{z}^{(t-k):t})} + \frac{1\{Z_{it}^{(t-k):t} = \tilde{\mathbf{z}}^{(t-k):t}\}}{\hat{P}(Z_{it}^{(t-k):t} = \tilde{\mathbf{z}}^{(t-k):t})}$ as the regression weight.

5. Use the regression coefficient for $\{Z_{it}^{(t-k):t} = \mathbf{z}^{(t-k):t}\}$, denoted as $\hat{\tau}_i(d)$, as the estimate of $\tau_i(\mathbf{z}^{(t-k):t}, \tilde{\mathbf{z}}^{(t-k):t}, d)$.

6. Calculate the standard error of $\hat{\tau}_i(d)$, denoted as $\hat{\sigma}_i(d)$, using the spatial HAC variance estimator with the distance matrix and the cutoff value $\tilde{d}$.

7. Construct the confidence interval $[\hat{\tau}_i(d) - z_{1 - \frac{\alpha}{2}} \hat{\sigma}_i(d), \hat{\tau}_i(d) + z_{1 - \frac{\alpha}{2}} \hat{\sigma}_i(d)]$ for the significance level $\alpha$, where $z_{1 - \frac{\alpha}{2}}$ is the $1 - \frac{\alpha}{2}$ quantile of the standard normal distribution.

8. (Optional) Validate the identification assumptions by applying the method to a placebo outcome.

4 Statistical theory

To describe the behavior of the estimators in large samples, let’s consider a sequence of collections of units $\mathcal{M}_N$ with an increasing sample size $N$. The number of periods $T$ is fixed. For the proposed estimators to behave well asymptotically, it is essential that interdependence among the observations
caused by interference (known as interference dependence) is not too strong. Suppose each observation interferes with all the other observations, then the effective sample size remains small even when \( N \) grows to infinity.

I measure interference dependence in the sample using the dependency graph (Baldi and Rinott, 1989; Sävje, Aronow and Hudgens, 2021). Given the choice of \( \Omega_d \) and the treatment assignment histories of interest, the graph in period \( t \) is represented by a \( N \times N \) matrix \( G_{(t,d,k)} = \{ g_{ij; (t,d,k)} \}_{N \times N} \). Each unit is considered as a vertex in the graph and \( g_{ij; (t,d,k)} = 1 \) if and only if unit \( j \) depends on unit \( i \). We say unit \( i \) interferes with unit \( j \) if the latter’s transformed outcome is influenced by the former’s treatment history. This is documented with a dummy variable \( I_{ij; (t,d,k)} \) for each pair of units, therefore

\[
I_{ij; (t,d,k)} = \begin{cases} 
1, & \text{if } i = j; \\
1, & \text{if } \mu_j \left( \left\{ Y_n(Z_{1:t}^1) \right\}_{n \in \mathcal{N}} ; d \right) \neq \mu_j \left( \left\{ Y_n(Z_{1:t}^1) \right\}_{n \in \mathcal{N}} ; d \right) \\
& \text{for } Z_{1:t} \setminus Z_{i}^{(t-k):t} = \tilde{Z}_{1:t} \setminus Z_{i}^{(t-k):t} ; \\
0, & \text{otherwise.}
\end{cases}
\]

Then, \( g_{ij; (t,d,k)} = 1 \) if and only if \( I_{ij; (t,d,k)} = 1 \) or \( I_{il; (t,d,k)} I_{lj; (t,d,k)} = 1 \) for a third unit \( l \). In other words, unit \( j \) depends on unit \( i \) if the latter interferes with the former or another unit interferes with both of them. I denote the set of units that depend on unit \( i \) in \( G_{(t,d,k)} \) as \( \mathcal{B}_{i; (t,d,k)} \) and its cardinality as \( b_{i; (t,d,k)} \).

Assumption 4 (Finite degree of interference dependence).

\[ b_{i; (t,d,k)} \leq \bar{b}. \]

\(^8\)The graph’s structure is unknown to researchers, hence cannot be used to construct exposure mappings as in Aronow and Samii (2017).
for all units i.

Assumption 4 says that for any unit i, the number of units that depend on it, $b_i; (t,d,k)$, is always bounded by a fixed number $\tilde{b}$ regardless of the sample size $N$. As the number of units increases, we anticipate the dependency graph to expand in size rather than density, hence yielding more information. This assumption holds true when the effect from any unit dissipates to zero beyond a fixed range or when units are sufficiently distanced from each other geographically. It can be further relaxed to accommodate growth in the size of the neighborhood (as further discussed in the appendix). Since the structure of $G_{(t,d,k)}$ varies across the values of $t$, $d$, and $k$, the validity of Assumption 4 must be verified for each combination of the three parameters. Satisfying this assumption generally becomes more challenging when $\Omega_d$ encompasses more units. In practical settings, researchers might focus on an $\Omega_d$ that is localized around each unit, such as circles within a certain distance range.

The final assumption we need is the boundedness of the potential outcomes, which ensures the existence of all the moments of the transformed outcome.

**Assumption 5** (Bounded potential outcomes). There exist a constant $\tilde{y}$ such that

$$|Y_{it}(z^{1:t})| \leq \tilde{y}$$

for all units i and any $z^{1:t}$.

Assuming the aforementioned assumptions are met, it can be shown that the variances of the proposed estimators converge to zero as $N \to \infty$, provided that the propensity score model is correctly specified. Consistency of these estimators follows from Markov’s inequality. Furthermore, their asymptotic normality can be established using the central limit theorem from Ogburn et al. (2020). For the Hajek estimator, the result is as follows:

**Theorem 1.** Under Assumptions 1-5, when the estimates of the propensity scores converge to their true values at the rate of $O_P \left( \frac{1}{\sqrt{N}} \right)$, estimates from the Hajek estimator are consistent and
asymptotically normal:

$$\sqrt{N} \left( \hat{\tau}_{t,HA}(z^{(t-k):t};d) - \tau_t(z^{(t-k):t};d) \right) \to N \left( 0, V_{HA;(t,d,k)} \right),$$

where $N \left( 0, V_{HA;(t,d,k)} \right)$ is a normal distribution with mean zero and the variance $V_{HA;(t,d,k)} = \text{Var}[\hat{\tau}_{t,HA}(z^{(t-k):t};d)].$

Similar results hold for the other estimators, and are presented in the appendix. The asymptotic variance of the estimators comprises three parts: the traditional Neyman variance, the variance induced by interference, and the variance from estimating the propensity scores. The second part accounts for the dependence among the units. It is assumed that the third part converges to zero no slower than the first two parts. The explicit expression of each part is presented in the appendix, where I also demonstrate that the spatial HAC variance estimator (Conley, 1999) is asymptotically valid for $V_{HA;(t,d,k)}$ under an additional assumption that Wang et al. (2020) refer to as homophily in treatment effects. The assumption requires that units which generate larger-than-average effects reside close to each other and is satisfied in many practical applications. The cutoff value $\tilde{d}$ is decided by the maximal degree of interference dependence in the dependency graph. Suppose the effect becomes negligible beyond $d$, then two units will not interfere each other if their distance is larger than $d$, and not depend on each other if their distance is larger than $2 \times d$, which justifies the proposal to set $\tilde{d} = 2 \times d$ in the previous section.

5 Caveats of fixed effects models

Fixed effects models are commonly used in panel data analysis to adjust for unobservable confounders when SUTVA holds. For these models to identify causal effects, two conditions must be fulfilled. The first is strict exogeneity, meaning that conditional on both observable and unobservable confounders, the treatment assignment is independent to the distribution of potential outcomes in any period.\(^9\) The second condition is that the influence of the unobservables on the outcome should

\(^9\)In the context of the DID design, the assumption is equivalent to “parallel trends.”
conform to a certain form. In the DID estimator or TWFE model, for example, the unobservables are represented by the sum of the unit fixed effect and the period fixed effect. By adjusting the outcome, such as subtracting the unit- and period-specific averages from each $Y_t$, we can eliminate these fixed effects and attribute the remaining difference between treated and untreated observations to the treatment. Essentially, the chosen model enables researchers to account for the unobservables and impute the counterfactual for the treated observations to estimate the ATT. The idea is referred to as the counterfactual estimation framework (Liu, Wang and Xu, 2019) in the literature.

However, when interference is present, such an approach loses its feasibility as the untreated observations are now contaminated by the spillover from the treated ones. Consequently, the ATT is no longer well defined, and it becomes impossible to remove the influences of the unobservables or interference via any adjustment of the outcome without further restrictions on the spillover effects. I elaborate this point using the following example.

The two-period DID case  Assume that there are two periods and the $N$ units are from two groups. The treatment group receives the treatment only in period 2 ($Z_i^2 = 1$) and the control group remains untreated in both periods. The propensity score for each unit $i$ is denoted as $p_i$. The following DGP is adopted:

$$Y_{i1} = \delta + \alpha_i + \xi_1 + \epsilon_{i1},$$

$$Y_{i2} = \delta + g_i(Z_2) + \alpha_i + \xi_2 + \epsilon_{i2},$$

where $\delta$ is the intercept; $\alpha_i$ and $\xi_t$ represent unit and period fixed effects, respectively; $\epsilon_{it}$ is the idiosyncratic error term; and $g_i(.)$ stands for the treatment effect on unit $i$ in period 2. To be consistent with the literature, I assume that the $N$ units are drawn from a larger population and $E[\epsilon_{it}|\alpha_i, \xi_t, Z_{1:2}^t] = 0$, where the expectation $E$ is taken over sampling. This is the assumption of strict exogeneity and implies “parallel trends” in this DID setting:

$$E[Y_{i2}(0^{1:2}) - Y_{i1}(0^{1:2})|Z_i^2 = 1] = E[Y_{i2}(0^{1:2}) - Y_{i1}(0^{1:2})|Z_i^2 = 0].$$
The classic DID estimator takes the form:

\[ \hat{\tau}_{\text{DID}} = \frac{1}{N_1} \sum_{i=1}^{N} Z_{i2} (Y_{i2} - Y_{i1}) - \frac{1}{N_0} \sum_{i=1}^{N} (1 - Z_{i2}) (Y_{i2} - Y_{i1}) \]

\[ \rightarrow \frac{\mathbb{E}[p_{i} g_{1i}]}{\mathbb{E} p_i} - \frac{\mathbb{E}[(1 - p_{i}) g_{0i}]}{\mathbb{E}(1 - p_{i})}, \text{ as } N \to \infty, \]

where \( g_{1i} = \mathbb{E}_{Z_{1:2}|Z_{2}} [g_{i}(1, Z_{1:2}\setminus Z_{2})] \) and \( g_{0i} = \mathbb{E}_{Z_{1:2}|Z_{2}} [g_{i}(0, Z_{1:2}\setminus Z_{2})] \) are the marginalized treatment effects on unit \( i \) from \( Z_{i} \) when it equals 1 and 0, respectively.

The last expression indicates that under interference, the DID estimator converges to the difference between two weighted averages of the marginalized treatment effects. This quantity does not equal an average effect on any population unless when the marginalized treatment effects are homogeneous \( (g_{1i} = g_{1} \text{ and } g_{0i} = g_{0}) \) or when the propensity scores are the same across units \( (p_{i} = p) \). If both a unit’s propensity score and its marginalized effects are positively correlated with its unit fixed effect, the AME at \( d = 0 \) will generally be overestimated by the DID estimator, and vice versa. In cases where there are multiple periods and the two-way fixed effects model is employed, Strezhnev (2018) and De Chaisemartin and d’Haultfoeuille (2020) have shown that the estimate equals a weighted average across a series of DID estimates, hence the problem persists. The argument also applies to fixed effects models with more complicated forms, such as the interactive fixed effects models (Xu, 2017; Athey et al., 2018).

Thus, when interference exists in panel data, researchers are faced with a trade-off about how they want to remain agnostic. If we have a clear understanding of the treatment assignment process but limited knowledge about how the effects caused by interference vary, the proposed method presents a preferable choice. Conversely, if we are confident in our comprehension of how the effects spread from one observation to another but uncertain about the set of confounders, sticking with the fixed effects models and striving to accurately control the spillover effects may be a more fitting decision.

The cost of dropping unit fixed effects may not be as high as many would expect in the spatial
setting. Firstly, when the units are part of larger divisions such as states or provinces, it is possible to maintain good control over the influence of unobservable confounders by incorporating division fixed effects into the propensity score model. Secondly, for smaller units that are contiguous, such as towns, polling stations, or pixels on maps, it is reasonable to assume that their fixed effects vary continuously over the geography \( \mathcal{X} \). Hence, their influence can be well approximated by a smooth function of the geographic coordinates.\(^{10}\) In Section 7, I provide simulation evidence that adding a polynomial of the geographic coordinates into the propensity score model effectively eliminates the bias caused by continuous unobservable confounders.

6 Extensions

**Weaker assumptions.** Both Assumption 1 and Assumption 3 can be relaxed without changing the main conclusions. Even when they fail, the AME can still be identified and estimated as before. But the set of confounders may differ, as well as the structure of the dependency graph in period \( t \). Then, the validity of Theorem 1 for given \( d \) and \( k \) should be re-examined. If Assumption 1 is no longer satisfied, treatment assignments and outcomes from future periods will become confounders. Assumption 3 is likely to be violated if the treatment is contagious.\(^ {11} \) Now, \( Z_{it} \) is affected by the histories of other units, and they should be incorporated into the model of the propensity score. Various options are available for this scenario, including semi-parametric methods in spatial statistics (Thaden and Kneib, 2018; Dupont, Wood and Augustin, 2022), and spatial auto-regression models that are popular in social science (Simmons and Elkins, 2004; Egami, 2018; Cook, Hays and Franzese, 2023). Researchers may select the appropriate model based on their contextual understanding of the treatment.

**Other estimands.** For any treatment assignment history with length \( k \), the AMEs can be aggregated over periods between \( T_1 \) and \( T_2 \) (\( T_1 \geq t - k \) and \( T_2 \leq t \)) to capture the average effect it

\(^{10}\)Such an assumption is common in spatial statistics Refer to Thaden and Kneib (2018) and Dupont, Wood and Augustin (2022) for more details.

\(^{11}\)For example, states may copy each other’s policy innovations.
generates:
\[
\tau(z(t-k):t, zhat(t-k):t; d) = \frac{1}{T_2 - T_1 + 1} \sum_{t=T_1}^{T_2} \tau_t(z(t-k):t, zhat(t-k):t; d)
\]

It is more challenging to disentangle the influence of different types of interference if treatment assignment in the data does not follow the structure of staggered adoption. In this case, one option is to evaluate the effect generated by a summary statistic of various treatment assignment histories, such as the total number of periods under treatment, \( g_{tk} = \sum_{s=t-k}^{t} z_s \). The estimand \( \tau(g, 0; d) \) can be interpreted as the marginal effect generated by all the treatment assignment histories that have \( g \) treated periods between periods \( t - k \) and \( t \). It is similar to a marginal structural model (Robins, Hernan and Brumback, 2000) in biostatistics but does not require the model to be correctly specified. Both types of estimands can be estimated using the proposed method. Whether these quantities are substantively meaningful depends on the purpose of the study and the research questions of interest.

**Application in networks.** The proposed framework can be applied to settings where the units are embedded in a network. A natural choice of the metric \( d \) is the length of the shortest path between any pair of units. The entire framework remains intact except for the interpretation of the estimands. Nevertheless, Assumption 4 is less likely to hold in networks due to the well-known “small world” phenomenon. When there exists a short path between any pair of units, and the true effect declines slowly along it, the assumption is violated. Kojevnikov, Marmer and Song (2021) have illustrated the distinction between space and network in their topological structure, highlighting the need for a clear understanding of the network formation process before applying the method to networks. One possible approach to addressing this issue is to restrict the effect’s declining rate over \( d \), as in Kojevnikov, Marmer and Song (2021) and Leung (2022). I discuss this approach in the appendix.

### 7 Simulation

In this section, I test the performance of the proposed method using the simulated dataset examined in Section 2, which consists of 400 units and 5 periods. The untreated potential outcome, \( Y_d(0^{1:t}) \),
is generated from the following process:

\[ Y_{it}(0^{1:t}) = 5 + 0.3 \times X_{1,it} + 0.5 \times X_{2,it} + \alpha_i + \xi_t + \varepsilon_{it}, \]

where \( \alpha_i \) and \( \xi_t \) represent unit and period fixed effects, respectively; \( X_{1,it} \) and \( X_{2,it} \) are two covariates; \( \varepsilon_{it} \) is the idiosyncratic shock. \( \xi_t, X_{2,it}, \) and \( \varepsilon_{it} \) are independently drawn from the standard normal distribution. \( \alpha_i \) is interpolated by fitting a kriging model on 16 randomly selected points in the raster. The colors in Figure 2 show the distribution of \( \alpha_i \). \( X_{2,it} \) obeys a standard normal distribution centered around \( \alpha_i \). Remember that the effect function \( g(d) \) emanating from each unit is monotonically decreasing over the distance \( d \) to the unit, as shown in Figure 3.\(^{12}\) The effect received by each unit \( i \) equals the sum of effects from all the other units in the sample, multiplied by its fixed effect \( \alpha_i \). Effects in period \( t-1 \) persist into period \( t \) with a discount factor of 0.6. Therefore, for any observation \((i,t)\), the treatment effect on it is:

\[ \tau_{it}(Z^{1:t}) = \alpha_i \sum_{j=1}^{N} Z_{ij} g(d_{ij}) + 0.6 \times 1\{t > 1\} \times \tau_{i,t-1}(Z^{1:t-1}), \]

which incorporates both temporal and spatial interference. The observed outcome \( Y_{it}(Z^{1:t}) \) equals \( Y_{it}(0^{1:t}) + \tau_{it}(Z^{1:t}). \)

Recall that treatment occurs from the third period and follows a staggered adoption design. The propensity score of unit \( i \) in period \( t \) \((t \geq 3)\) is decided by the \( i \)'s outcome and treatment status in the previous period, as well as the two covariates, but not the fixed effects. Specifically, the true propensity score for each observation is:

\[ P(Z_{it} = 1) = \text{Logit}(-3 + 0.05 \times X_{1,it} + 0.1 \times X_{2,it} + 0.05 \times Y_{i,t-1} + 0.4 \times Z_{i,t-1} + v_{it}), \]

where \( v_{it} \sim N(0,1) \). On average, the number of treated units increases from 85 in period 3 to 210 in period 5.

\(^{12}\)Results based on other effect functions are shown in the appendix.
The transformed outcomes at each \( d \in \{0, 1, \ldots, 7\} \) are constructed as circle means around the units. The propensity scores are estimated via a pooled logistic regression. In each of the 1,000 assignments, \( \hat{\tau}_5 ((0, 0, 1), (0, 0, 0); d) \), \( \hat{\tau}_5 ((0, 1, 1), (0, 0, 0); d) \), and \( \hat{\tau}_5 ((1, 1, 1), (0, 0, 0); d) \) are obtained for each \( d \) from the Hajek estimator and plotted against the true AME curves in the top row of Figure 4. Despite the moderate sample size (400 units), the bias of the Hajek estimator is negligible, confirming the asymptotic unbiasedness of the IPTW estimators.

Next, I modify the DGP for the propensity score to include the unit fixed effect \( \alpha_i \). Now, the assumption of sequential ignorability is violated. Since \( \tau_{it}(Z_{1:t}) \) is positively correlated with \( \alpha_i \), the analysis in Section 5 suggests that estimates from the DID estimator will be upward biased. The simulation results, shown in the middle row of Figure 4, confirm this prediction across all distance values.\(^{13}\) In this setting, ignoring the unit fixed effects when estimating the propensity scores also results in biased IPTW estimates. However, since \( \alpha_i \) varies smoothly over the geography, we can control its influence by adding a second-order polynomial of the geographic coordinates to the logistic regression model, as discussed in Section 5. The estimates from this approach are presented in the bottom row of Figure 4, where the bias becomes negligible again. Therefore, under the structural restriction of smooth unit fixed effects, the proposed estimators outperform the DID estimator.

The online appendix contains additional simulation results. They suggest that: 1. the IPTW estimators perform well when the effect function is non-monotonic; 2. the bias of the DID estimator disappears when the effect is homogeneous; and 3. the augmented estimators improve the efficiency of estimation. These findings are consistent with the theoretical predictions and provide further evidence of the effectiveness of the proposed methods.

\(^{13}\)For \( d > 0 \), I construct the circle averages as before and apply the DID estimator to them. Covariates are controlled linearly in estimation.
Figure 4: Bias of different estimators

Notes: The black curves in these plots represent the three true AME curves. The top row displays the estimates obtained from the Hajek estimator for all 1,000 assignments under sequential ignorability. Estimates shown in the middle and bottom rows are obtained from the DID estimator and the Hajek estimator, respectively, when unit fixed effects influence the treatment assignment process. A quadratic polynomial of the geographic coordinates of the units is incorporated into the propensity score model when generating estimates for the bottom row. The bias of the estimators is illustrated as the difference between the average of gray curves and the black curve in each plot. The effect function is monotonic.
8 Applications

I now present replication results of two empirical studies. The first study, involving only two periods, serves to illustrate the bias of the DID estimator in the presence of interference. The second study, with three periods, demonstrates how we can decompose the treatment effects caused by various types of interference.

8.1 Protest and public support for the opposition

Wang and Wong (2021) explored the impact of Hong Kong’s Umbrella Movement in 2014 on public support for the pro-democracy opposition that organized the protest. During the movement, protesters occupied several streets across the city for 79 days. The outcome of interest is the opposition’s vote shares in the 2016 parliamentary election across constituencies in Hong Kong. The treatment is defined as a dichotomous variable which equals 1 in 2016 if a street in a constituency was occupied by protesters and 0 otherwise.\footnote{Wang and Wong (2021) defined the treatment based on the distance between each constituency and the nearest protest site. I have altered the coding rule for illustrative purposes.} There were 19 treated constituencies and 380 untreated ones.\footnote{Locations of the treated versus untreated constituencies are displayed in the appendix.}

The top-left plot in Figure 5 presents results from the DID estimator. I use the election year preceding the protest, 2012, as the pre-treatment period. Among the treated constituencies, the opposition’s vote share fell from 58.8\% in 2012 to 52.1\% in 2016. In contrast, the change among the untreated constituencies was from 55.5\% to 54.5\%. I also plot the opposition’s vote shares from the 2008 election, which suggests that the assumption of parallel trends is likely valid. Under the assumption, the opposition’s vote share would have been 57.9\% if the protest had not occurred, thus the DID estimate equals $-5.8\%$.

However, Hong Kong is a densely populated city, so it is conceivable that the protest’s impact could spill over and cause interference across constituencies. To assess this possibility, I divide all the untreated constituencies into two groups, the remote and the nearby, based on whether their
nearest distance to the treated ones exceeds or falls short of 6 km.\textsuperscript{16} It is reasonable to expect the nearby group to be more susceptible to the influence of spatial interference. As displayed in the top-right and bottom-left plots in Figure 5, the DID estimate equals \(-9.0\%\) when the remote constituencies serve as the control group, but it shrinks significantly to \(-2.7\%\) if the nearby ones play the role. This variation aligns with the presence of spatial interference, leading to the conclusion that none of the DID estimates are entirely credible. Additionally, the parallel trends assumption seems to hold in both instances, which rules out the likelihood that the divergence is due to unobserved confounders.

The bottom-right plot in Figure 5 shows the results from the proposed method. I used a logistic regression model to predict the propensity score for each constituency. The model controls the opposition’s vote share in 2012, three different covariates,\textsuperscript{17}, a second-order polynomial of geographic coordinates for each constituency, and dummy variables indicating which of the 18 administrative districts the constituency falls within. I construct the transformed outcome as donuts with a width of 2 km centered around each constituency. The distance values range from 0 km, where the transformed outcome equals the constituency’s outcome, to 10 km. I set the cutoff value of the spatial HAC variance estimator to be 12 km as the effects become near-zero after 6 km.\textsuperscript{18} The estimates indicate that the protest did generate a significantly negative impact on the opposition’s vote share within the range of 4 km.\textsuperscript{19} When \(d = 0\), the estimated AME \((-6.0\%)\) is close in magnitude to the DID estimate. But the former holds an interpretation as the marginal effect of the protest, whereas the latter does not have a meaningful one.

\textsuperscript{16}6 km is the median distance between an untreated constituency to the nearest treated one in the sample.
\textsuperscript{17}They are the percentages of mandarin speakers, college students, and residents whose monthly income is above $7,740.
\textsuperscript{18}The Bell-McCaffrey correction (Bell and McCaffrey, 2002) is applied to the variance’s degree of freedom as the number of treated units is small, although it does not change the main conclusions.
\textsuperscript{19}Results of the placebo test based on the 2008 election outcomes are presented in the appendix, which support the validity of the identification assumption.
Figure 5: Replication of Wang and Wong (2021)

Notes: In the top-left, top-right, and bottom-right plots, triangles along the black and gray lines respectively display the average vote shares for the opposition among treated and untreated constituencies over the 2008, 2012, and 2016 elections. The black dashed lines represent the predicted counterfactual under the assumption of parallel trends. In the bottom-right plot, the black dots indicate the AME estimates at each distance $d$, from 0 km to 8 – 10 km. The black segments represent the 95% confidence intervals, calculated using the spatial HAC variance estimator.
8.2 Political consequences of building wind turbines

As previously mentioned in the introduction, Stokes (2016) studied the political consequences of building wind turbines in Ontario, Canada, where laws were passed by the Liberal Party government since 2003 to support these projects. The study collected election data from 6,186 precincts in Ontario over three elections: 2003, 2007, and 2011, and combined the data with the location of each operational or proposed turbine. Precinct \( i \) is considered as treated in year \( t \) if a turbine project within its boundary has been proposed before the election.\(^{20}\) The data exhibit the structure of staggered adoption, with 53 precincts treated in 2007 and 131 in 2011.

In the original analysis, the author used the two-way fixed effects model, controlling for precinct and election year fixed effects. She found that turbines significantly increased the turnout rate (+2.4%) and decreased the Liberal Party’s vote share (−4.2%) in the treated precincts. She then investigated spatial spillovers by constructing donuts with a radius of 1 km around each turbine. The distance values range from 1 km to 5 km, with significant effects detected at distances up to 3 km.

I modify the author’s approach by assuming sequential ignorability instead of strict exogeneity.\(^{21}\) The propensity scores are estimated by a logistic regression model, which controls for turnout rate and the Liberal Party’s vote share in the previous election, a quadratic polynomial of the longitude and latitude for each precinct, and a period dummy.\(^ {22}\) I construct donuts with a radius of 2 km around each treated and untreated precinct,\(^ {23}\) and estimate the AMEs in the distance range of 0 km to 16 km. I focus on two treatment assignment histories between 2007 and 2011, \((1,1)\) and \((0,1)\), and estimate their effects in 2011 relative to the history \((0,0)\). Cutoff values of the spatial HAC variance estimator are set at 16 km and 12 km for the turnout rate and the Liberal Party’s vote share, respectively.

\(^{20}\)I focus on proposed turbines since few turbines were operational over the period.
\(^{21}\)In this example, sequential ignorability is plausible as the treatment status of each observation is decided by the turbine builders after examining its characteristics in the past.
\(^{22}\)In 2011, only precincts that have not been treated are used for estimating the propensity scores. The precincts that received treatment in 2007 inherently possess a propensity score of 1.
\(^{23}\)The digital map used by the author is no longer available. I can only construct the donuts based on the distance between the centroids of any two precincts, which is less precise.
Figure 6: Replication of Stokes (2016)

Notes: The figures on the left display the estimates for the turnout rate, while the ones on the right show the estimates for the Liberal Party’s vote share in election year $t$. The results are based on the Hajek estimator, with distance values ranging from 0 km to 14 – 16 km. The black dots in figures on the top represent $\hat{\tau}_{2011}((1,1), (0,0); d)$, while the gray dots represent $\hat{\tau}_{2011}((0,1), (0,0); d)$. The black and gray segments represent their respective 95% confidence intervals, calculated using the spatial HAC variance estimator. Gray dots and segments in figures on the bottom represent $\hat{\tau}_{2007}((1,1), (0,0); d)$ and their 95% confidence intervals.
The replication results are presented in Figure 6. They provide evidence for the presence of both temporal and spatial interference. Being treated in both 2007 and 2011 would increase the turnout rate by 7.7% and decrease the Liberal Party’s vote share by 6.4% in a precinct. The effects are also observed among its neighbors that are 2-4 km away, where the turnout rate would increase by 5.0%, and the Liberal Party’s vote share would decrease by 6.0%. In precincts treated only in 2011, treatment assignment history caused a rise of 3.1% in the turnout rate and a decline of 7.0% in the Liberal Party’s vote share. The indirect effects on its neighbors that are 2-4 km away are of similar magnitude. The effects on the outcome variables become close to zero (and statistically insignificant) among precincts that are 6 and 8 km away from the treatment, respectively, which justifies the choice of the cutoff values. In summary, spatial interference seems to pervade both outcome variables, while temporal interference predominantly sways the turnout rate. This asymmetry may be worth further investigations by empirical researchers.

The bottom plots of Figure 6 present results using the outcomes in 2007 as placebo outcomes. For precincts where turbine projects were proposed between the 2007 and 2011 elections (i.e., treated only in 2011), their outcomes should be statistically indistinguishable from those of the untreated precincts in 2007, once all confounders are properly accounted for. The results affirm this expectation. For both outcomes, the estimates are statistically insignificant and hover around zero, thereby supporting the identification assumption. In the appendix, I present more results to demonstrate the robustness of the findings, including estimates from the augmented estimator and from using a propensity score model that allows the treatment to be contagious.

9 Conclusion

This paper addresses the challenge of causal inference with panel data, when both temporal and spatial interference are present. I illustrate that interference undermines the conventional approaches that rely on strict exogeneity, such as the DID estimator and the TWFE model, making them unsuitable for delivering meaningful or consistent estimates of causal effects. In contrast, under
sequential ignorability, researchers can rely on the proposed IPTW estimators to obtain valid estimates of the AMEs generated by a unit’s treatment assignment history on itself or its neighbors, even when the interference structure is unknown. The estimators can be implemented via weighted regression with a transformed outcome, while statistical inference can be conducted by combining the spatial HAC variance estimator with normal approximation.

As demonstrated by both simulation and the replication studies, this method provides researchers with straightforward tools to measure spillover effects in time and space, under assumptions that are easy to verify in practice. I thus anticipate it to have broad applications in political science as well as in other disciplines. I have developed an R-package, SpatialEffect, to facilitate the implementation. Future research may investigate ways to generalize the method to larger geographic spaces or social networks with dense ties. It is also worthwhile to explore how to adapt the method to long panel or time-series cross-sectional (TSCS) data, where estimating the impacts of unobservable confounders on the treatment assignment process becomes feasible (Arkhangelsky and Imbens, 2019; Feng, 2020; Arkhangelsky et al., 2021; Blackwell and Yamauchi, 2021).

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Causal Inference with Panel Data under Temporal and Spatial Interference
(Supplementary Information)
Ye Wang*

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A. Proofs

A1. Identification under sequential ignorability

First note that for any $z(l-k):t$, we have

$$\int \left[ P \left( Z^{(l-k)}:t = z^{(l-k)}:t \right) \right] \prod_{i \in N} \left[ \sum_{d} \mu_{l} \left( \{ Y_{jt} \}_{j \in S} : d \right) \right]$$

$$= \int \left[ P \left( Z^{(l-k+1)}:t = z^{(l-k+1)}:t \right) \right] \prod_{i \in N} \left[ \sum_{d} \mu_{l} \left( \{ Y_{jt} \}_{j \in S} : d \right) \right]$$

$$= \int \left[ P \left( Z^{(l-k+1)}:t = z^{(l-k+1)}:t \right) \right] \prod_{i \in N} \left[ \sum_{d} \mu_{l} \left( \{ Y_{jt} \}_{j \in S} : d \right) \right]$$

$$= \int \left[ P \left( Z^{(l-k+1)}:t = z^{(l-k+1)}:t \right) \right] \prod_{i \in N} \left[ \sum_{d} \mu_{l} \left( \{ Y_{jt} \}_{j \in S} : d \right) \right]$$

$$= \ldots$$

$$= \int \sum_{j \in \Omega_{l}} \left[ Y_{jt} \left( \{ z^{(l-k)}:t \} : d \right) \right]$$

$$= \sum_{j \in \Omega_{l}} \left\{ Y_{jt} \left( \{ z^{(l-k)}:t \} : d \right) \right\}$$

The above part iterates the same step from period $t-k+1$ to $t$. The third equality uses Assumption 2 and the law of iterated expectation. Then,

$$\tau_{l} \left( z^{(l-k)}:t, z^{(l-k)}:t \right) = \frac{1}{N} \sum_{i=1}^{N} \mu_{i} \left( \{ Y_{jt} \}_{j \in S} : d \right)$$

$$= \frac{1}{N} \sum_{i=1}^{N} \left[ \sum_{j \in \Omega_{l}} \left( Y_{jt} \left( \{ z^{(l-k)}:t \} : d \right) \right) \right]$$

$$= \frac{1}{N} \sum_{i=1}^{N} \left[ \sum_{j \in \Omega_{l}} \left( Y_{jt} \left( \{ z^{(l-k)}:t \} : d \right) \right) \right]$$

We have proved the claim in the main draft that the AME can be identified from data.

A2. Variance of the estimators

I first derive the variance of the Horvitz-Thompson estimators when the propensity scores are known. The extra uncertainty induced by estimating the nuisance parameters is discussed in Section A6. The result is stated in the following lemma:

**Lemma 1.** Under Assumptions 1-3 in the main text, we have the following bound for the variance of the Horvitz-Thompson estimator $\tau_{l} \left( z^{(l-k)}:t, z^{(l-k)}:t \right)$ when the propensity scores are known:

$$\text{Var} \left( \tilde{\tau}_{l} \left( z^{(l-k)}:t, z^{(l-k)}:t \right) \right)$$

$$\leq \frac{1}{N^{2}} \sum_{i=1}^{N} \left[ \sum_{j \in \Omega_{l}} \left( Y_{jt} \left( \{ z^{(l-k)}:t \} : d \right) \right) \right]$$

$$+ \frac{1}{N^{2}} \sum_{i=1}^{N} \left[ \sum_{j \in \Omega_{l}} \left( Y_{jt} \left( \{ z^{(l-k)}:t \} : d \right) \right) \right]$$

$$- \frac{1}{N^{2}} \sum_{i=1}^{N} \left[ \sum_{j \in \Omega_{l}} \left( Y_{jt} \left( \{ z^{(l-k)}:t \} : d \right) \right) \right]$$

The result is stated in the following lemma:
Proof. Using the expression of the Horvitz-Thompson estimator, we have:

\[
\text{Var} \left( \hat{\tau}_t (z(t-k):t, \tilde{z}(t-k):t; d) \right) = \frac{1}{N^2} \text{Var} \left[ \sum_{i=1}^{N} \left( \frac{1}{P(Z_{i}^{(t-k)} = z(t-k))} - \frac{1}{P(Z_{i}^{(t-k)} = \tilde{z}(t-k))} \right) \mu_i \left( \{Y_j\}_{j \in A}; d \right) \right]
\]

\[
= \frac{1}{N^2} \sum_{i=1}^{N} \text{Var} \left[ \left( \frac{1}{P(Z_{i}^{(t-k)} = z(t-k))} - \frac{1}{P(Z_{i}^{(t-k)} = \tilde{z}(t-k))} \right) \mu_i \left( \{Y_j\}_{j \in A}; d \right) \right]
\]

\[
+ \frac{1}{N^2} \sum_{i=1}^{N} \sum_{j \in A} \text{Cov} \left[ \left( \frac{1}{P(Z_{i}^{(t-k)} = z(t-k))} - \frac{1}{P(Z_{i}^{(t-k)} = \tilde{z}(t-k))} \right) \mu_i \left( \{Y_j\}_{j \in A}; d \right), \mu_j \left( \{Y_j\}_{j \in A}; d \right) \right]
\]

\[
\leq \frac{1}{N^2} \sum_{i=1}^{N} E \left[ \left( \frac{1}{P(Z_{i}^{(t-k)} = z(t-k))} - \frac{1}{P(Z_{i}^{(t-k)} = \tilde{z}(t-k))} \right) \right]^2 \mu_i \left( \{Y_j\}_{j \in A}; d \right)
\]

The first two terms in the above expression can be further expanded as:

\[
= \frac{1}{N^2} \sum_{i=1}^{N} E \left[ \left( \frac{1}{P(Z_{i}^{(t-k)} = z(t-k))} - \frac{1}{P(Z_{i}^{(t-k)} = \tilde{z}(t-k))} \right) \mu_i \left( \{Y_j\}_{j \in A}; d \right) \right]^2
\]

\[
- \frac{1}{N^2} \sum_{i=1}^{N} E^2 \left[ \left( \frac{1}{P(Z_{i}^{(t-k)} = z(t-k))} - \frac{1}{P(Z_{i}^{(t-k)} = \tilde{z}(t-k))} \right) \mu_i \left( \{Y_j\}_{j \in A}; d \right) \right]
\]

\[
= \frac{1}{N^2} \sum_{i=1}^{N} E \left[ \left( \frac{1}{P(Z_{i}^{(t-k)} = z(t-k))} - \frac{1}{P(Z_{i}^{(t-k)} = \tilde{z}(t-k))} \right) \mu_i \left( \{Y_j\}_{j \in A}; d \right) \right]^2 + \frac{1}{N^2} \sum_{i=1}^{N} E \left[ \frac{1}{P(Z_{i}^{(t-k)} = z(t-k))} \mu_i^2 \left( Y_i | Z(t-k)| ; d \right) \right]^2
\]

\[
- \frac{1}{N^2} \sum_{i=1}^{N} E^2 \left[ \left( \frac{1}{P(Z_{i}^{(t-k)} = z(t-k))} - \frac{1}{P(Z_{i}^{(t-k)} = \tilde{z}(t-k))} \right) \mu_i \left( \{Y_j\}_{j \in A}; d \right) \right]
\]

\[
= \frac{1}{N^2} \sum_{i=1}^{N} E \left[ \left( \frac{1}{P(Z_{i}^{(t-k)} = z(t-k))} - \frac{1}{P(Z_{i}^{(t-k)} = \tilde{z}(t-k))} \right) \mu_i \left( \{Y_j\}_{j \in A}; d \right) \right]^2 + \frac{1}{N^2} \sum_{i=1}^{N} E \left[ \frac{1}{P(Z_{i}^{(t-k)} = z(t-k))} \mu_i^2 \left( Y_i | Z(t-k)| ; d \right) \right]^2
\]

\[
- \frac{1}{N^2} \sum_{i=1}^{N} E^2 \left[ \left( \frac{1}{P(Z_{i}^{(t-k)} = z(t-k))} - \frac{1}{P(Z_{i}^{(t-k)} = \tilde{z}(t-k))} \right) \mu_i \left( \{Y_j\}_{j \in A}; d \right) \right]
\]

\[
\leq \frac{1}{N^2} \sum_{i=1}^{N} E \left[ \frac{1}{P(Z_{i}^{(t-k)} = z(t-k))} \mu_i^2 \left( Y_i | Z(t-k)| ; d \right) \right]^2 + \frac{1}{N^2} \sum_{i=1}^{N} E \left[ \frac{1}{P(Z_{i}^{(t-k)} = \tilde{z}(t-k))} \mu_i^2 \left( Y_i | Z(t-k)| ; d \right) \right]^2.
\]
And the first covariance term equals:

\[
\frac{1}{N^2} \sum_{i=1}^{N} \sum_{l \neq i} \text{Cov} \left( \frac{1}{P(Z_{i}(t-k) = y(t-k)^{r})} \mu_i \left( \{ Y_{y} \}_{j \in S} ; d \right) , \frac{1}{P(Z_{l}(t-k) = y(t-k)^{r})} \mu_l \left( \{ Y_{y} \}_{j \in S} ; d \right) \right)
\]

\[
= \frac{1}{N^2} \sum_{i=1}^{N} \sum_{l \neq i} E \left[ \frac{1}{P(Z_{i}(t-k) = y(t-k)^{r})} \frac{1}{P(Z_{l}(t-k) = y(t-k)^{r})} \mu_i \left( \{ Y_{y} \}_{j \in S} ; d \right) \mu_l \left( \{ Y_{y} \}_{j \in S} ; d \right) \right]
\]

\[
- \frac{1}{N^2} \sum_{i=1}^{N} \sum_{l \neq i} E \left[ \frac{1}{P(Z_{i}(t-k) = y(t-k)^{r})} \mu_i \left( \{ Y_{y} \}_{j \in S} ; d \right) E \left[ \frac{1}{P(Z_{l}(t-k) = y(t-k)^{r})} \mu_l \left( \{ Y_{y} \}_{j \in S} ; d \right) \right] \right]
\]

\[
= \frac{1}{N^2} \sum_{i=1}^{N} \sum_{l \neq i} E \left[ \mu_i \left( \{ Y_{y} \}_{j \in S} ; Z_{i} \cup Z_{l} \right) \right] E \left[ \mu_l \left( \{ Y_{y} \}_{j \in S} ; Z_{i} \cup Z_{l} \right) \right]
\]

The last equality uses the fact that for \( l \notin \mathcal{B}_{t}(d,k) \), the expectation of the product equals the product of expectations. Other covariance terms have similar forms. The final result is obtained by combining these terms together.

Next, I present a bound for the asymptotic variance of the Hajek estimator with known propensity scores:

**Lemma 2.** Define

\[
\tilde{V}_{t,k,d} = \frac{1}{N^2} \sum_{i=1}^{N} \sum_{l \neq i} \frac{E \left[ \mu_i \left( \{ Y_{y} \}_{j \in S} ; Z_{i} \cup Z_{l} \right) \right] - \mu_{d,k} \left( Z_{i} \cup Z_{l} \right) \left( \mu \left( \mu_{d,k} \left( Z_{i} \cup Z_{l} \right) \right) \right) }{P \left( Z_{i}(t-k) = y(t-k)^{r}) \right)}
\]

\[
+ \frac{1}{N^2} \sum_{i=1}^{N} \sum_{l \neq i} \sum_{a \neq b} (-1)^{1(a-b)} \left( \mu \left( \mu_{d,k} \left( Z_{i} \cup Z_{l} \right) \right) \right) \left( \mu \left( \mu_{d,k} \left( Z_{i} \cup Z_{l} \right) \right) \right) \left( \mu \left( \mu_{d,k} \left( Z_{i} \cup Z_{l} \right) \right) \right) \left( \mu \left( \mu_{d,k} \left( Z_{i} \cup Z_{l} \right) \right) \right)
\]

\[
- \frac{1}{N^2} \sum_{i=1}^{N} \sum_{l \neq i} \sum_{a \neq b} (-1)^{1(a-b)} \left( \mu \left( \mu_{d,k} \left( Z_{i} \cup Z_{l} \right) \right) \right) \left( \mu \left( \mu_{d,k} \left( Z_{i} \cup Z_{l} \right) \right) \right) \left( \mu \left( \mu_{d,k} \left( Z_{i} \cup Z_{l} \right) \right) \right) \left( \mu \left( \mu_{d,k} \left( Z_{i} \cup Z_{l} \right) \right) \right)
\]

where \( \mu_{d,k} \) is a constant that satisfies \( 1 \leq \hat{h}_{t,k,d} \leq \max_{t \in \{1,2, ..., N\}} \hat{h}_{t,k,d} \).

The proof is similar to that in Wang et al. (2020) hence omitted. Assumption 4 in the main text indicates that \( \hat{h}_{t,k,d} \) is constant. When \( \hat{h}_{t,k,d} \) increases with \( N \), the difference between the Horvitz-Thompson estimator and the Hajek estimator diminishes asymptotically. But in any finite sample, the Hajek estimator is still more efficient, as suggested by the difference between the two variance expressions in Lemma 1 and Lemma 2. The variance expression can be further simplified under an extra assumption:

**Assumption (Homophily in treatment effects).** For given \( t,d,k, \) define

\[
\frac{1}{N} \sum_{i=1}^{N} \left[ \mu_i \left( \{ Y_{y} \}_{j \in S} ; d \right) - \bar{\mu}_{t,k,d} \left( \{ Y_{y} \}_{j \in S} ; d \right) \right] \left[ \sum_{l \neq i} \left( \frac{1}{P(Z_{i}(t-k) = y(t-k)^{r})} \mu_i \left( \{ Y_{y} \}_{j \in S} ; d \right) \right) \right] \geq 0.
\]

The assumption indicates that the expected treatment effect generated by unit \( i \)’s history at distance \( d \) is positively correlated with those effects generated by its neighbors over the period from \( t-k \) to \( t \). There is “homophily” in treatment effects over the geography \( \mathcal{B} \): units that generate larger-than-average effects reside close to each other. It is often the case in reality. But researchers need to justify the assumption using their substantive knowledge.
Lemma 3. Under Assumptions 1-5 and the extra assumption, the variance bound defined in lemma 2 can be replaced by:

\[ V_{e,t,k,d} = \frac{1}{N^2} \sum_{i=1}^{N} \mathbb{E} \left[ \left( \frac{Y_i}{P(Z_i^{(t,k)} = 1)} - \frac{Z_i^{(t,k)}}{P(Z_i^{(t,k)} = 1)} \right)^2 \right] + \frac{1}{N^2} \sum_{j=1}^{N} \mathbb{E} \left[ \left( \frac{Y_j}{P(Z_j^{(t,k)} = 1)} - \frac{Z_j^{(t,k)}}{P(Z_j^{(t,k)} = 1)} \right)^2 \right] \]

where \( \tilde{b}(t,d,k) \) is the same constant as in Lemma 2 and \( \mathbb{V}_{HA}(t,d,k) = O_P(1) \).

Proof. Note that by definition,

\[ \mathbb{V}_{HA}(t,d,k) = \mathbb{E} \left[ \left( \frac{Y_i}{P(Z_i^{(t,k)} = 1)} - \frac{Z_i^{(t,k)}}{P(Z_i^{(t,k)} = 1)} \right)^2 \right] \]

Hence, the last term of the variance bound in lemma 2 can be simplified as

\[ -\frac{1}{N^2} \sum_{i=1}^{N} \sum_{j=1}^{N} \mathbb{E} \left[ \mu_i \left( \frac{Y_i}{P(Z_i^{(t,k)} = 1)} - \frac{Z_i^{(t,k)}}{P(Z_i^{(t,k)} = 1)} \right) \right] \]

Lemma 4. (Ogburn et al. (2020), Lemma 1 and 2) Consider a set of \( N \) units. Let \( U_1, \ldots, U_N \) be bounded mean-zero random variables with finite fourth moments and dependency neighborhoods \( \mathcal{B}_{l,d,k} \). If \( \sum_{i=1}^{N} \mathbb{E}\left[ U_i^2 \right] \rightarrow 0 \) for all \( i \), then

\[ \frac{\sum_{i=1}^{N} U_i}{\sqrt{\text{Var}(\sum_{i=1}^{N} U_i)}} \rightarrow N(0,1). \]

Next, I prove Theorem 1 in the main text.

Proof. Define \( U_i \) as

\[ U_i = \frac{\sum_{i=1}^{N} \mathbb{E}\left[ U_i^2 \right]}{\sqrt{\text{Var}(\sum_{i=1}^{N} U_i)}} \]

where \( \mathcal{B}_{l,d,k} \) is the same constant as in Lemma 2 and \( \sqrt{\sum_{i=1}^{N} \mathbb{V}_{HA}(l,d,k)} = O_P(1) \).
Then, $\sum_{i=1}^{N} U_i = \sqrt{\frac{N}{N_{t,d,k}}} \left( \hat{\tau}(z^{(t+k)}; \hat{z}^{(t+k)}; :d) - \tau(z^{(t+k)}; z^{(t+k)}; :d) \right)$ and $E[U_i] = 0$. We know that $U_i$ has finite fourth moments as all the outcomes are bounded, and $\text{Var}(\sum_{i=1}^{N} U_i) = \frac{N}{N_{t,d,k}} \text{Var} \left( \frac{\hat{\tau}(z^{(t+k)}; \hat{z}^{(t+k)}; :d)}{\sqrt{\text{Var}(\hat{\tau}(z^{(t+k)}; \hat{z}^{(t+k)}; :d))}} \right)$ is also finite. From Lemma 4, it can be seen that
\[
\frac{\hat{\tau}(z^{(t+k)}; \hat{z}^{(t+k)}; :d)}{\sqrt{\text{Var}(\hat{\tau}(z^{(t+k)}; \hat{z}^{(t+k)}; :d))}} \rightarrow N(0,1) \quad \text{as long as } \max_{i \in \{1, \ldots, N\}} b_{t,i(d,k)} = o_P \left( N^{1/2} \right).
\]
Theorem 1 is a special case of the result when $\max_{i \in \{1, \ldots, N\}} b_{t,i(d,k)} = O_P(1)$. The asymptotic distribution of the Hajek estimator can be obtained via the Delta method.

The requirement that $\max_{i \in \{1, \ldots, N\}} b_{t,i(d,k)} = o_P \left( N^{1/2} \right)$ is unlikely to be satisfied in networks as the size of the neighborhood may grow fast. Kojevnikov, Marmer and Song (2021) and Leung (2022) suggest that it is necessary to control the decline rate of the spillover effect in this case for any estimator to behave well in large samples. Leung (2022) introduces a condition termed "approximate neighborhood independence" for this purpose. Let's denote unit $i$'s $s$-neighborhood in $\mathcal{G}(t,d,k)$ as $\mathcal{B}_{i,s}(t,d,k)$, the condition can be restated as follows:

**Assumption (Approximate neighborhood independence):** There exist uniformly bounded constants $\{\theta_{N,s}\}$ such that (a) $\sup_{N} \theta_{N,s} \rightarrow 0$ as $s \rightarrow \infty$, and (b) for any $N$, $i \in \mathcal{N}_N$, and $s \geq 0$,

$$\max_{i \in \mathcal{N}_N} \mathbb{E} \left[ \mu \left( \left\{ Y_{i,j}(Z^{(t)}) \right\}_{j \in \mathcal{N}_N} :d \right) - \mu \left( \left\{ Y_{i,j}(\hat{Z}^{(t)}) \right\}_{j \in \mathcal{N}_N} :d \right) \right] \leq \theta_{N,s},$$

where $Y_{i,j}(Z^{(t)}) = Y_{i,j} \left( \left\{ Z_{i,j}^{(t)} \right\}_{j \in \mathcal{N}_j} \left\{ Z_{i,j}^{(t)} \right\}_{j \in \mathcal{N}_N} \right)$. The condition implies that the influence on $\mu \left( \left\{ Y_{i,j}(Z^{(t)}) \right\}_{j \in \mathcal{N}_N} :d \right)$ from units other than $i$ declines as the distance to $i$ rises in $\mathcal{G}(t,d,k)$. It enables us to apply the central limit theorem in Kojevnikov, Marmer and Song (2021). See Leung (2022) for more details.

### A4. Variance estimation

I first present the expression of the spatial heteroscedasticity and auto-correlation consistent (HAC) variance estimator (Conley, 1999). Let's denote the diagonal weighting matrix

$$\left\{ \left( \alpha^{(t+k)}_{-\hat{z}^{(t+k)}} \right)_{Z_{i,j}^{(t+k)}} \left( \left( \alpha^{(t+k)}_{-\hat{z}^{(t+k)}} \right)_{Z_{i,j}^{(t+k)}} \right) \right\}_{i,j \in \mathcal{N}_N}$$

as $\mathbf{\hat{M}} = \{ \hat{M}_{i,j} \}_{i,j \in \mathcal{N}_N}$. Define $\hat{\mathbf{X}}$,

\[
\begin{pmatrix}
1 \left( Z_{i,j}^{(t+k)} = \hat{z}_{(t+k)}^{(t+k)} \right) \\
1 \left( Z_{i,j}^{(t+k)} \neq \hat{z}_{(t+k)}^{(t+k)} \right)
\end{pmatrix}
\begin{pmatrix}
\left\{ Z_{i,j}^{(t+k)} \right\}_{j \in \mathcal{N}_N} \\
\left\{ \hat{Z}_{i,j}^{(t+k)} \right\}_{j \in \mathcal{N}_N}
\end{pmatrix}
\] and $\hat{\mathbf{Y}}_R$ as

\[
\begin{pmatrix}
\mu_1 \left( Y_{i,j} \right)_{j \in \mathcal{N}_N} :d \\
\mu_2 \left( Y_{i,j} \right)_{j \in \mathcal{N}_N} :d \\
\vdots \\
\mu_N \left( Y_{i,j} \right)_{j \in \mathcal{N}_N} :d
\end{pmatrix}
\].

Then the regression representation of the Hajek estimator has

\[
\hat{\tau}(d) = \left( \hat{\mathbf{X}}_{OLS} \left( \hat{\mathbf{Z}}^{(t+k)}; \hat{\mathbf{Z}}^{(t+k)}; :d \right) \right) = \left( \hat{\mathbf{X}}_{OLS} \left( \hat{\mathbf{Z}}^{(t+k)}; \hat{\mathbf{Z}}^{(t+k)}; :d \right) \right)^{-1} \hat{\mathbf{X}}_{OLS} \left( \hat{\mathbf{Z}}^{(t+k)}; \hat{\mathbf{Z}}^{(t+k)}; :d \right) \hat{\mathbf{Y}}_R.
\]

Using simple algebra, it can be shown that $\hat{\tau}(d) = \frac{\sum_{i=1}^{N} \left( \alpha^{(t+k)}_{-\hat{z}^{(t+k)}} \right)_{Z_{i,j}^{(t+k)}} \mu \left( Y_{i,j} \right)_{j \in \mathcal{N}_N} :d}{\sum_{i=1}^{N} \left( \alpha^{(t+k)}_{-\hat{z}^{(t+k)}} \right)_{Z_{i,j}^{(t+k)}} \mu \left( Y_{i,j} \right)_{j \in \mathcal{N}_N} :d}$ and $\hat{\tau}_{HAC}(d)$.

Let's denote the residual for each observation as $\hat{r}_i$ and the variance-covariance matrix of $\{ \hat{r}_i \}_{i \in \mathcal{N}_N}$ as $\Sigma_R$. It is worth noting that the covariance of $\hat{r}_i$ and $\hat{r}_j$ is non-zero if and only if $j \in \mathcal{B}_{i,(t,d,k)}$. Furthermore, $\sum_{i=1}^{N} \left( \alpha^{(t+k)}_{-\hat{z}^{(t+k)}} \right)_{Z_{i,j}^{(t+k)}}$ is denoted as $K_1$ and $\sum_{i=1}^{N} \left( \alpha^{(t+k)}_{-\hat{z}^{(t+k)}} \right)_{Z_{i,j}^{(t+k)}}$ as $K_0$. We know the spatial HAC variance of $\left( \hat{\tau}_{OLS}(d), \hat{\tau}_{HAC}(d) \right)$ can be expressed as:

$$\text{Var} \left( \hat{\tau}_{OLS}(d), \hat{\tau}_{HAC}(d) \right) = \left( K_0 0 \right)^{-1} \left( \begin{array}{c}
\text{Var}(\hat{\tau}_{OLS}(d)) \\
\text{Var}(\hat{\tau}_{HAC}(d))
\end{array} \right) \left( K_0 0 \right)^{-1}.$$

We are interested in entry (2,2) of the above expression. Rearranging the observations such that those with $Z_{i,j}^{(t+k)} = \hat{z}_{(t+k)}^{(t+k)}$ rank before those with...
\( Z_{i}^{(l)} = \tilde{Z}_{i}^{(l)} \), the quantity of interest can be simplified as:

\[
\begin{align*}
\hat{\text{Var}}(\hat{\tau}_{\text{OLS}}(Z_{i}^{(l)}, \tilde{Z}_{i}^{(l)}; d)) &= \frac{1}{K_{1}} \sum_{i=1}^{N} \frac{1}{P(\hat{Z}_{i}^{(l)} = Z_{i}^{(l)}; d)} \hat{\nu}_{i}^{2} + \frac{1}{K_{2}} \sum_{i=1}^{N} \sum_{j \in N(i)} \frac{1}{P(\hat{Z}_{i}^{(l)} = \tilde{Z}_{i}^{(l)}; d)} \hat{\nu}_{i} \hat{\nu}_{j}
\end{align*}
\]

The last step is to show that the variance estimate \( \hat{\text{Var}}(\hat{\tau}_{\text{OLS}}(Z_{i}^{(l)}, \tilde{Z}_{i}^{(l)}; d)) \) is consistent for the Hajek estimator’s asymptotic variance under the assumption of homophily in treatment effects. The proof is similar to that of Lemma A.6 in Wang et al. (2020) hence I omit the details to save space.

A5. The augmented estimator

As mentioned in the main text, the Horvitz-Thompson estimators can also be augmented by transforming the outcome, \( \mu \left( \{ \hat{Y}_{i} \} \}_{i \in A}; d \right) \), more precisely with outcome-based diffusion models. We estimate an outcome model for \( E[Y_{i}|Z_{i}^{(l)}, Y_{i}^{(l)}; X_{i}^{(l)}, Z_{i}^{(l)}] \), acquiring the fitted value \( \hat{Y}_{i} \) and its conditional expectation \( E[\hat{Y}_{i}|\tilde{Z}_{i}^{(l)} \neq \tilde{Z}_{j}^{(l)}] \). Then, the augmented estimator has the form

\[
\begin{align*}
\hat{\tau}_{\text{aug}}(\tilde{Z}_{i}^{(l)}; \tilde{Z}_{j}^{(l)}; d)
&= \frac{1}{N} \sum_{i=1}^{N} \left[ \frac{1}{\hat{P}(\tilde{Z}_{i}^{(l)} = \tilde{Z}_{j}^{(l)}; d)} \mu_{i} \left( \{ \hat{Y}_{i} \} \right) \chi_{j \in A}; d \right] - \frac{1}{N} \sum_{i=1}^{N} \left[ \frac{1}{\hat{P}(\tilde{Z}_{i}^{(l)} = \tilde{Z}_{j}^{(l)}; d)} \mu_{i} \left( \{ \hat{Y}_{i} \} \right) \chi_{j \in A}; d \right]
+ \frac{1}{N} \sum_{i=1}^{N} \mu_{i} \left( E \left[ \hat{Y}_{i}|\tilde{Z}_{i}^{(l)} \neq \tilde{Z}_{j}^{(l)} \right] \right) \chi_{j \in A}; d
\end{align*}
\]

For example, researchers may believe the true effect function is monotonic in distance from each unit, as the one shown in Section 7. Furthermore, for any unit, the effects from its neighbors are homogeneous, additive, and do not persist over time. If so, the following model can be fitted to predict \( Y_{i} \):

\[
Y_{i} = \sum_{d \in D} \beta_{d} Z_{i}^{(l)} \mathbb{1}(d_{i} = d) + h(Z_{i}^{(l)}; Y_{i}^{(l)}, X_{i}^{(l)}) + \epsilon_{i},
\]

where \( h \) is some known function. If the model is close enough to the true DGP, then \( \beta_{d} \) equals the AME at \( d \). In this sense, the augmented estimator is doubly robust: it is unbiased when either the propensity score model or the additive diffusion model for the response surface is correctly specified. Nevertheless, when the model includes interaction terms of the treatments, it is still necessary to estimate the propensity scores correctly to obtain a precise approximation of the outcome’s marginal expectation, \( E \left[ \hat{Y}_{i}|\tilde{Z}_{i}^{(l)} \neq \tilde{Z}_{j}^{(l)} \right] \). The estimator is thus less robust than the doubly robust estimators when SUTVA holds. It can be understood from the perspective of residual balancing (Liu et al., 2019; Athey, Imbens and Wager, 2018): we use the diffusion model to reduce noises and then re-weigh its residuals to balance the remaining influences of the confounders. But as highlighted by Kang and Schafer (2007), achieving balance for all residuals can be a formidable task in finite samples, especially when the outcome model significantly diverges from the actual data generating process.

The statement is formally proved below:

\textbf{Proof.} Consider the scenario where the propensity scores are correctly specified while the diffusion model is not. Then,

\[
E \left[ \hat{\tau}_{\text{aug}}(\tilde{Z}_{i}^{(l)}; \tilde{Z}_{j}^{(l)}; d) \right] = E \left[ \hat{\tau}_{\text{aug}}(\tilde{Z}_{i}^{(l)}; \tilde{Z}_{j}^{(l)}; d) \right]
\]

\[
= \frac{1}{N} \sum_{i=1}^{N} \frac{1}{\hat{P}(\tilde{Z}_{i}^{(l)} = \tilde{Z}_{j}^{(l)}; d)} \mu_{i} \left( \{ \hat{Y}_{i} \} \right) \chi_{j \in A}; d
- \frac{1}{N} \sum_{i=1}^{N} \frac{1}{\hat{P}(\tilde{Z}_{i}^{(l)} = \tilde{Z}_{j}^{(l)}; d)} \mu_{i} \left( \{ \hat{Y}_{i} \} \right) \chi_{j \in A}; d
+ \frac{1}{N} \sum_{i=1}^{N} \mu_{i} \left( E \left[ \hat{Y}_{i}|\tilde{Z}_{i}^{(l)} \neq \tilde{Z}_{j}^{(l)} \right] \right) \chi_{j \in A}; d
= 0.
\]
The second equality uses the linearity of $\mu_i \left( \left\{ \hat{Y}_{jt} \right\}_{j \in N} : d \right)$. Note that the result holds even when the effects are not additive. Next, suppose the diffusion model is accurate but the propensity scores are not. Then, $Y_{jt} - \hat{Y}_{jt} = \bar{e}_i$ and $\operatorname{E}[\bar{e}_i] = 0$. We have

$$E \left[ \tilde{v}_{\text{aug}}(x^{(i-k)}_t, z^{(i-k)}_t : d) \right]$$

$$= \frac{1}{N} \sum_{i = 1}^{N} \left[ \frac{1}{N} \sum_{j = 1}^{N} \tilde{p} \left( Z_i^{(i-k)} = z^{(i-k)}_t \right) \left\{ \left[ \hat{Y}_{jt} - \hat{Y}_{jt} \right] \right\}_{j \in N} : d \right] - \frac{1}{N} \sum_{i = 1}^{N} \left[ \frac{1}{N} \sum_{j = 1}^{N} \tilde{p} \left( Z_i^{(i-k)} = z^{(i-k)}_t \right) \left\{ \left[ \hat{Y}_{jt} - \hat{Y}_{jt} \right] \right\}_{j \in N} : d \right]$$

$$+ E \left[ \frac{1}{N} \sum_{i = 1}^{N} \tilde{p} \left( Z_i^{(i-k)} = z^{(i-k)}_t \right) \left\{ \left[ E \left[ \hat{Y}_{jt} \left( Z_i^{(i-k)} = z^{(i-k)}_t \right) \right] \right\}_{j \in N} : d \right] - \frac{1}{N} \sum_{i = 1}^{N} \tilde{p} \left( Z_i^{(i-k)} = z^{(i-k)}_t \right) \left\{ \left[ E \left[ \hat{Y}_{jt} \left( Z_i^{(i-k)} = z^{(i-k)}_t \right) \right] \right\}_{j \in N} : d \right]$$

$$= \frac{1}{N} \sum_{i = 1}^{N} \left[ E \left[ \hat{Y}_{jt} \left( Z_i^{(i-k)} = z^{(i-k)}_t \right) \right] \right\}_{j \in N} : d$$

$$- \frac{1}{N} \sum_{i = 1}^{N} \tilde{p} \left( Z_i^{(i-k)} = z^{(i-k)}_t \right) \left\{ \left[ E \left[ \hat{Y}_{jt} \left( Z_i^{(i-k)} = z^{(i-k)}_t \right) \right] \right\}_{j \in N} : d \right]$$

$$= \tau(z^{(i-k)}_t, \tilde{Z}_i^{(i-k)} : d).$$

The last equality requires the additivity of the effects. Otherwise, the propensity scores have to be accurate for us to calculate $E \left[ \hat{Y}_{jt} \left( Z_i^{(i-k)} = z^{(i-k)}_t \right) : d \right]$. Denoting the variance of the augmented estimator as $V_{\text{aug}(i,k,d)}$, we have

$$\operatorname{Pr} \left( \frac{N}{\hat{v}_{i,k,d}} V_{\text{aug}(i,d,k)} \leq \frac{N}{\hat{v}_{i,k,d}} \hat{v}_{i,k,d} \right) \rightarrow 1,$$

where

$$\hat{v}_{i,k,d} = \frac{1}{N} \sum_{i = 1}^{N} \left[ \frac{1}{N} \sum_{j = 1}^{N} \tilde{p} \left( Z_i^{(i-k)} = z^{(i-k)}_t \right) \left\{ \left[ E \left[ \hat{Y}_{jt} \left( Z_i^{(i-k)} = z^{(i-k)}_t \right) \right] \right\}_{j \in N} : d \right] - \tilde{p} \left( Z_i^{(i-k)} = z^{(i-k)}_t \right) \left\{ \left[ E \left[ \hat{Y}_{jt} \left( Z_i^{(i-k)} = z^{(i-k)}_t \right) \right] \right\}_{j \in N} : d \right] \right]$$

$$+ \frac{1}{N} \sum_{i = 1}^{N} \tilde{p} \left( Z_i^{(i-k)} = z^{(i-k)}_t \right) \left\{ \left[ E \left[ \hat{Y}_{jt} \left( Z_i^{(i-k)} = z^{(i-k)}_t \right) \right] \right\}_{j \in N} : d \right] - \tilde{p} \left( Z_i^{(i-k)} = z^{(i-k)}_t \right) \left\{ \left[ E \left[ \hat{Y}_{jt} \left( Z_i^{(i-k)} = z^{(i-k)}_t \right) \right] \right\}_{j \in N} : d \right] \right]$$

$$= \frac{1}{N} \sum_{i = 1}^{N} \left[ E \left[ \hat{Y}_{jt} \left( Z_i^{(i-k)} = z^{(i-k)}_t \right) \right] \right\}_{j \in N} : d$$

This is a straightforward extension of the classic variance formula for doubly robust estimators (see, e.g., Lunceford and Davidian (2004)). It implies that the augmented estimator is more efficient than the Horvitz-Thompson estimator or the Hajek estimator when both the propensity score model and the diffusion model are correct. I leave the question of whether the augmented estimator achieves semiparametric efficiency under interference to future research. Consistency of the estimator comes from the variance expression. We can also see that the difference between the Horvitz-Thompson and the augmented estimator is a sample average. Hence, it is also asymptotically normal.

A6. Estimation of the nuisance parameters

So far, it has been assumed that the nuisance parameters, either the propensity scores or the diffusion model, are known to the researchers. In practice, researchers must estimate them from data, which impacts the variances of the proposed estimators. When the nuisance parameters are estimated using parametric models, the variances can be obtained from the standard theory of M-estimation (Lunceford and Davidian, 2004). It is known that ignoring uncertainties from estimating the nuisance parameters leads to more conservative variance estimates. Furthermore, when the convergence rate of our estimators is lower than $\sqrt{N}$, the uncertainties stemming from estimating the nuisance parameters become negligible in large samples. Researchers may also consider non-parametric estimators for the nuisance parameters, such sieve estimators (Hirano, Imbens and Ridder, 2003), covariate balancing propensity score (Imai and Ratkovic, 2015), and highly adaptive lasso (Ertefaie, Hejazi and van der Laan, 2020). The main results won’t be affected as long as the estimates converge to their true values at a sufficiently fast rate. When sieve estimators are adopted, the variances can be estimated following the proposal in Ackerberg, Chen and Hahn (2012). In other contexts, the variance estimates need to be discussed on a case-by-case basis.

A7. Bias of the DID estimator

Under the DGP described in Section 5 of the main text, we can derive the asymptotic bias of the DID estimator:

$$\text{Bias}_{\text{DID}} = \lim_{N \rightarrow \infty} \hat{\tau}_{\text{DID}} - \tau((0,1),(0,0);0)$$

$$= \text{Cov} \left[ p_1, \delta_1 \right] \left( E \left[ p_1 \right] + E \left[ 1 - p_1 \right] \right)$$

where $g_{1i} = E \left[ Z_i^{(1)} : Z_0 \right] \left[ g_i(1, Z_i^{(1)} : Z_0) \right]$ and $g_{0i} = E \left[ Z_i^{(0)} : Z_0 \right] \left[ g_i(0, Z_i^{(0)} : Z_0) \right]$. Its direction hinges on the correlation between the unobservable propensity score $p_1$ and the magnitude of the marginal treatment effects. When both of them are positively correlated with $\alpha$, $\tau((0,1),(0,0);0)$ will be overestimated by the DID estimator, and vice versa. If the effects are additive across units, it is straightforward to show that the bias of the estimator relative to the ATT equals the difference between the average spillover effect on the treated units and that on the untreated units.

When interference is absent, $g_{0i} = 0$ and $\delta_{1i} = g_i$. The expression reduces to $\frac{g_i(1, Z_i^{(1)} : Z_0)}{E \left[ p_1 \right]}$, the population ATT. The analysis can be generalized to the AMEs where $d > 0$ by replacing $Y_t$ with the transformed outcome. Note that $p_1$ cannot be consistently estimated as $T$ is fixed. This is the well-known incidental parameter problem (Neyman and Scott, 1948) in panel data analysis. As a result, we cannot infer the magnitude of the bias from data or correct it with any model.
B. Extra results from simulation and applications

B1. Bias of the IPTW estimators

We first examine the bias of both the Horvitz-Thompson estimator and the Hajek estimator when sequential ignorability holds and the effect function is non-monotonic. The effect function and the AMEs are displayed in Figure 1. Figure 2 presents the estimation results. We can see that both estimators are unbiased.

Figure 1: Effect function and the AMEs

Notes: The left plot shows how the effect function that emanates from each unit varies over distance. The right plot presents the estimands $\tau_5 ((0,0,1),(0,0,0); d)$ and $\tau_5 ((1,1,1),(0,0,0); d)$ over the same range of distance values.
Figure 2: Bias of the IPTW estimators

Notes: The black curves in these plots represent the three true AME curves. The top and bottom rows respectively display the estimates obtained from the Horvitz-Thompson and the Hajek estimator for all 1,000 assignments under sequential ignorability. The effect function is non-monotonic.
B2. Bias of the DID estimator under homogeneous treatment effect

Figure 3 shows the bias of the DID estimator when sequential ignorability does not hold and unit fixed effects become confounders. The DGP is the same as the one in the main text. The only difference is that the effects are homogeneous across the units and do not accumulate over periods. As predicted by our discussion in Section 5, the DID estimator is now unbiased for the AMEs.

Figure 3: Bias of the DID estimator under homogeneous treatment effect

Notes: The black curves in these plots represent the three true AME curves. The plots display the estimates obtained from the DID estimator for all 1,000 assignments, when unit fixed effects influence the treatment assignment process. The effect function is monotonic and the effects are homogeneous across the units.
B3. Bias of the augmented estimator

To test the performance of the augmented estimator, I adopt the same data generating process in the main text. I employ the following model to predict the value of each $Y_d$:

$$Y_d = \sum_{d \in \mathcal{D}} \alpha_d \sum_{j=1}^{N} I(d_j = d) + \sum_{d \in \mathcal{D}} \beta_d \sum_{j=1}^{N} Z_d I(d_j = d) + X_d \psi + \epsilon_d$$

The specification is similar to that in traditional spatial regression, such as the spatiotemporal autoregressive distributed lag (STADL) model in Cook, Hays and Franzese (2023), where $\{I(d_j = d)\}_{N \times N}$ can be seen as a spatial weighting matrix. But neither the average outcome of other units nor lagged variables are controlled, since the focus is the effect of the treatment assignment history rather than of the treatment from the previous period.

The model can be estimated via OLS. Then, we have the predicted value for each $Y_d$:

$$\hat{Y}_d = \sum_{d \in \mathcal{D}} \hat{\alpha}_d \sum_{j=1}^{N} I(d_j = d) + \sum_{d \in \mathcal{D}} \hat{\beta}_d \sum_{j=1}^{N} Z_d I(d_j = d) + X_d \hat{\psi}.$$ 

The marginalized outcomes can be estimated by:

$$E \left[ Y_d (Z^{(j-k)}_1, Z^{(j-k)}_2) \right] = \sum_{d \in \mathcal{D}} \hat{\alpha}_d \sum_{j=1}^{N} I(d_j = d) + \sum_{d \in \mathcal{D}} \hat{\beta}_d \left( z_d I(d_k = d) + \sum_{j=1}^{N} P(Z_d = 1) I(d_j = d) \right) + X_d \hat{\psi}.$$ 

The model has ignored the effects from treatments in the previous period as well as the interaction among the effects. In the top row of Figure 4, I compare the estimated coefficients $\hat{\beta}_d$ with the AMEs. They are obviously biased. The bottom row of Figure 4 shows that the bias disappears once we augment the diffusion model with the propensity scores. Estimates from the augmented estimator have smaller variances than those from either the Horvitz-Thomson estimator or the Hajek estimator. Across all the distance values, the variance declines by at least 40%.

Figure 4: Bias of the diffusion model

Notes: The black curves in these plots represent the three true AME curves. The top rows respectively display the estimates obtained from only using the outcome model, while the bottom rows show the estimates obtained from the augmented estimator for all 1,000 assignments under sequential ignorability. The effect function is monotonic.
B4. Consistency of the IPTW estimators

Figure 5 illustrates how the bias of the Hajek estimator varies over sample sizes. We can see that when the number of units grows, the average bias, the mean squared error (MSE), and the supreme of bias all decline to zero across distance values, suggesting that the Hajek estimator is consistent. The ruggedness around $d = 2$ is caused by the boundary of the tiles.

**Notes:** Figures from left to right show the average bias, the average MSE, and the supreme of bias obtained from the Hajek estimator for all 1,000 assignments. Different colors indicate varying sample sizes.
B5. Coverage rate of the proposed confidence interval

Figure 6 presents how the coverage rate of the proposed 95% confidence interval, based on the spatial HAC variance estimator varies over sample sizes. The dashed line represents the nominal level of 95%. We can see that as $N$ grows, the coverage rate approaches the nominal level across distance values. The ruggedness around $d = 2$ is caused by the boundary of the tiles.

Figure 6: Consistency of the Hajek estimator

Notes: The figure shows the average coverage rate of the proposed 95% confidence interval across all 1,000 assignments for each of the distance values. Different colors indicate varying sample sizes.
B6. Map on protest locations in Hong Kong

Notes: The figure shows the treatment status (whether there was a protest during the Umbrella Movement) of constituencies in Hong Kong based on Wang and Wong (2021). The red circles indicate a donut around one of the treated constituencies.

B7. Placebo tests for the impacts of the Umbrella Movement

Notes: The outcome variable is the opposition’s vote share in the 2008 election. The black dots indicate the AME estimates at each distance $d$, from 0 km to 10 km. The black segments represent the 95% confidence intervals, calculated using the spatial HAC variance estimator.
B8. Locations of wind turbines from Stokes (2016)

Notes: The figure is taken from Stokes (2016). It shows the locations of the precincts and the proposed wind turbines in Ontario, Canada, in 2011.
B9. Replication of Stokes (2016) using the augmented estimator

We rely on the following diffusion model to predict the value of each $Y_{it}$:

$$
Y_{it} = \sum_{d \in D} a_d \sum_{j=1}^{N} 1\{d_{ij} = d\} + \sum_{d \in D} \beta_d \sum_{j=1}^{N} Z_{j} 1\{d_{ij} = d\} + \sum_{d \in D} \gamma_d \sum_{j=1}^{N} Z_{j-1} 1\{d_{ij} = d\} + \lambda Y_{i,t-1} + X_{i}^{\prime} \psi + \epsilon_{it}
$$

Estimates from the augmented estimator are presented in Figure 7. We can see that the main patterns are similar to what we have detected in the main text using the Hajek estimator. But the magnitude of the estimates becomes smaller for both outcomes, as do their standard error estimates. The results confirm the efficiency gains from using the augmented estimator. The difference in estimates could be driven by the bias of the outcome model under a relatively moderate sample size.

Figure 7: Replication of Stokes (2016) using the augmented estimator

Notes: The figure on the left displays the estimates for the turnout rate, while the one on the right shows the estimates for the Liberal Party’s vote share in election year $t$. The results are based on the augmented estimator, with distance values ranging from 0 – 2 km to 18 – 20 km. The black dots represent $\hat{\tau}_{2011}((0,1), (0,0); d)$, while the gray dots represent $\hat{\tau}_{2011}((1,1), (0,0); d)$. The black and gray segments represent their respective 95% confidence intervals, calculated using the spatial HAC variance estimator.
B10. Replication of Stokes (2016) when the treatment is contagious

The assumption of no contagion can be lifted by assuming that the propensity score is a function of the outcome history and treatment assignment history of nearby units. I estimate the propensity scores using the following model:

\[ P(Z_i = 1) = \text{Logit}(\delta + \alpha_1 Y_{i,t-1} + \alpha_2 \sum_{j=1}^{N} W_{ij} Y_{j,t-1} + \beta_1 Z_{i,t-1} + \beta_2 \sum_{j=1}^{N} W_{ij} Z_{j,t-1} + X_i \gamma + \nu_i), \]

where \( W_{ij} = \begin{cases} 1 & \text{if } d_{ij} \leq 5 \text{ km} \\ 0 & \text{otherwise} \end{cases} \) and \( X_i \) include confounders mentioned in the main text, such as the quadratic polynomial of the geographic coordinates.

The model assumes that only neighbors within the range of 5 km affect unit \( i \)'s treatment status. It is similar to the spatiotemporal autoregressive distributed lag model in Cook, Hays and Franzese (2023) without contemporaneous variables. Figure 8 suggests that the results do not change much when the treatment is contagious.

Figure 8: Replication of Stokes (2016) when the treatment is contagious

Notes: The figure on the left displays the estimates for the turnout rate, while the one on the right shows the estimates for the Liberal Party’s vote share in election year \( t \). The results are based on the augmented estimator, with distance values ranging from 0 km to 18 km. The black dots represent \( \hat{\tau}_{2011}((1,1), (0,0); d) \) (with 95% CI), while the gray dots represent \( \hat{\tau}_{2011}((0,1), (0,0); d) \) (with 95% CI). The black and gray segments represent their respective 95% confidence intervals, calculated using the spatial HAC variance estimator.
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