Distributionally Robust Causal Inference with Observational Data

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Abstract

We consider the estimation of average treatment effects in observational studies and propose a new framework of robust causal inference with unobserved confounders. Our approach is based on distributionally robust optimization and proceeds in two steps. We first specify the maximal degree to which the distribution of unobserved potential outcomes may deviate from that of observed outcomes. We then derive sharp bounds on the average treatment effects under this assumption. Our framework encompasses the popular marginal sensitivity model as a special case, and we demonstrate how the proposed methodology can address a primary challenge of the marginal sensitivity model that it produces uninformative results when unobserved confounders substantially affect treatment and outcome. Specifically, we develop an alternative sensitivity model, called the distributional sensitivity model, under the assumption that heterogeneity of treatment effect due to unobserved variables is relatively small. Unlike the marginal sensitivity model, the distributional sensitivity model allows for potential lack of overlap and often produces informative bounds even when unobserved variables substantially affect both treatment and outcome. Finally, we show how to extend the distributional sensitivity model to difference-in-differences designs and settings with instrumental variables. Through simulation and empirical studies, we demonstrate the applicability of the proposed methodology.

Keywords: average treatment effect, confounding, distributionally robust optimization, observational studies, sensitivity analysis

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1. Introduction

Although the unbiased estimates of causal effects can be relatively easily obtained from experimental data, it is often difficult to randomize treatment assignment for ethical and logistical reasons. As a result, many researchers rely on observational data to ascertain causal effects. The primary challenge of such observational studies is the possible existence of unobserved confounders. While the standard approach assumes that the treatment assignment is unconfounded (e.g., Rosenbaum and Rubin, 1983b; Robins et al., 1994a), such an assumption often lacks credibility in real-world applications.

We propose a new methodological framework for causal inference with observational data under the general observational study settings with the possibility of unobserved confounding. Specifically, we formulate the estimation of average treatment effects in the presence of unobserved confounders as a distributionally robust optimization problem under a particular ambiguity set (see, for example, Bertsimas and Den Hertog (2022) and Rahimian and Mehrotra (2019) for detailed reviews of the literature on distributionally robust optimization). We first specify the maximal degree to which the distribution of unobserved potential outcomes may differ from that of observed outcomes. We then bound the average treatment effects under this assumption.

The proposed framework includes as a special case of the popular marginal sensitivity model of Tan (2006), which is closely related to the sensitivity model of Rosenbaum (2002). This marginal sensitivity model has been recently used by several other researchers (e.g., Zhao et al., 2019; Kallus and Zhou, 2021). We show that the sensitivity analysis based on this model solves the distributionally robust optimization problem with a particular ambiguity set. The construction of this ambiguity set assumes that the distribution of counterfactual outcome among the treated units is equal to the weighted distribution of observed outcome among the control units.

One potential problem with the marginal sensitivity model, however, is that if unobserved confounders substantially affect the treatment and outcome, the distribution of potential outcome may not be comparable between the treated and control units. Partly for this reason, the marginal sensitivity model can produce uninformative results in practice. To address this issue, we develop an alternative sensitivity model under our proposed framework, called the distributional sensitivity model. The model assumes that the distribution of counterfactual outcome among the treated units is similar in its shape (but not in its location) to that of the distribution of the observed outcome among the same set of units. This assumption is satisfied if the heterogeneity of treatment effect is small relative to that of the outcome variable. A related assumption is often invoked when estimating heterogeneous treatment effects to justify the direct modeling of conditional average treatment effect (e.g., Hahn et al., 2020; Nie and Wager, 2021; Kennedy, 2020).

We also show that this distributional sensitivity model can be extended to other study designs, including the difference-in-differences, instrumental variables, and settings with
high-dimensional covariates. Finally, we conduct simulation and empirical analyses to evaluate the performance of the proposed methodology.

Related literature. Much of the previous work on sensitivity analysis builds upon either the sensitivity model of Rosenbaum (2002) (see e.g., Tan, 2006; Hasegawa and Small, 2017; Zhao et al., 2019; Kallus and Zhou, 2021; Yadlowsky et al., 2018; Fogarty, 2020; Dorn et al., 2021; Tan, 2022) or regression models with unobserved variables (see e.g., Rosenbaum and Rubin, 1983a; Scharfstein et al., 1999; Imbens, 2003; Imai et al., 2010; Cinelli and Hazlett, 2020; Chernozhukov et al., 2022).

In contrast, the proposed framework provides an alternative approach based on the method of distributionally robust optimization. It includes the marginal sensitivity model of Tan (2006) as a special case, but can be further extended to other study designs, including difference-in-differences, and instrumental variable designs, under the same distributionally robust optimization framework. The proposed methodology also exploits the commonly used assumption that

Another related literature is the one about partial identification (e.g., Balke and Pearl, 1997; Manski, 2007). The proposed methodology shows how a wide range of partial identification results can be obtained using the distributionally robust optimization approach. In fact, we contribute to the growing literature that utilizes robust optimization for various causal inference problems with partial identification, mostly in the context of individualized policy learning (e.g., Ben-Michael et al., 2021; Cui, 2021; Kallus and Zhou, 2021; Pu and Zhang, 2021; Zhang et al., 2022).

Organization of the paper. The rest of the paper is organized as follows. In Section 2, we introduce the proposed methodological framework. We show that this framework incorporates the marginal sensitivity model as a special case. We then develop an alternative sensitivity model, called the distributional sensitivity model, based on the assumption that the heterogeneity of treatment effect is small relative to the variation in the outcome across the treated and control units. We also extend this framework to the difference-in-differences as well as the settings with instrumental variables and high-dimensional covariates. In Section 3, we conduct simulation studies to evaluate the performance of the proposed methodology. Finally, in Section 4 we apply the proposed methodology to three empirical data sets and compare its performance with some alternative methods.

2. Distributionally Robust Causal Inference

In this section, we describe the proposed methodology. We begin by presenting our distributionally robust causal inference framework and then show how it can be applied to various models and settings.
2.1 Setup

Suppose we have a random sample of \( n \) units from a target population \( \mathcal{P} \). We observe the binary treatment \( T_i \in \{0,1\} \), a \( J \)-dimensional vector of pre-treatment covariates \( \mathbf{X}_i \), and the outcome variable of interest, \( Y_i \in \mathbb{R} \). We also have a vector of unobserved pre-treatment covariates \( \mathbf{U}_i \). All together, we assume the tuple \( \{\mathbf{X}_i, \mathbf{U}_i, T_i, Y_i(1), Y_i(0)\} \) is independently and identically distributed where \( Y_i(t) \) represents the potential outcome under the treatment assignment \( T_i = t \) with \( t \in \{0,1\} \). The relation between the observed and potential outcomes is given by

\[
Y_i = T_i Y_i(1) + (1 - T_i) Y_i(0)
\]

where we make the standard assumption of no interference between units (Rubin, 1990).

We consider a general observational study setting, in which both observed and unobserved pre-treatment covariates, i.e., \( \mathbf{X} \) and \( \mathbf{U} \), respectively, are potential confounders. Formally, we assume that the probability density function (PDF) of the potential outcomes \( Y_i(t) \) and the treatment \( T_i \) exist, and can be described by the following nonparametric models,

\[
\begin{align*}
\psi_y(t, \mathbf{X}_i, \mathbf{U}_i) & = f_{Y_i(t)|\mathbf{X}_i, \mathbf{U}_i}(y), \\
\pi(\mathbf{X}_i, \mathbf{U}_i) = P(T_i = 1 | \mathbf{X}_i, \mathbf{U}_i)
\end{align*}
\]

for all \( y \in \mathbb{R} \).

This model is quite general and makes no functional form assumption for \( \psi \) and \( \pi \). In particular, while all the confounders are assumed to be included in \( \mathbf{X}_i \) and \( \mathbf{U}_i \), both \( \mathbf{X}_i \) and \( \mathbf{U}_i \) may also contain variables that only affect either the treatment or outcome variable, but not both. We further note that all results in the paper would apply if we only assume the existence of the cumulative distribution function rather than the PDF, at the expense of increased notation to define the quantities of interest in the paper.

Under this setup, the average treatment effect on the treated subjects (ATT), which is our quantity of interest throughout this paper, can be written as,

\[
\tau = \frac{\int \int y \pi(\mathbf{X}_i, \mathbf{U}_i) \{\psi_y(1, \mathbf{X}_i, \mathbf{U}_i) - \psi_y(0, \mathbf{X}_i, \mathbf{U}_i)\} dy \ dF(\mathbf{U}_i) \ dF(\mathbf{X}_i)}{\int \int \pi(\mathbf{X}_i, \mathbf{U}_i) dF(\mathbf{U}_i) \ dF(\mathbf{X}_i)}.
\]

2.2 The Proposed Framework

For simplicity, we begin by assuming that the observed pre-treatment covariates are categorical that take a small number of possible values. Under this setting, we can condition on each value of \( \mathbf{X}_i \), conduct the same analysis, and aggregate the results over the values of the covariates, allowing us to ignore the existence of the observed pre-treatment covariates \( \mathbf{X}_i \). Later, we extend the proposed methodology to the cases where \( \mathbf{X}_i \) is high-dimensional and may include continuous covariates (see Section 2.7).

Under this setting, for any given value of the observed pre-treatment covariates \( \mathbf{X}_i = \mathbf{x} \), we can define the following four potential outcome distributions (two distributions defined
separately for the treatment and control groups),
\[
f_{st}(y) = \mathbb{P}(Y_i(s) = y \mid T_i = t) = \frac{\int \psi_y(s, U_i) \{((1-t) + (2t-1)\pi(U_i))dF(U_i)}{(1-t) + (2t-1)\int \pi(U_i)dF(U_i)},
\]
for \(s, t \in \{0, 1\}\) where we simplify the notation by suppressing the fact that we condition on the observed pre-treatment covariates \(X_i = x\).

For illustration, we focus on the average treatment effect for the treated (ATT), which is one of the most common causal quantity of interests in observational studies. The ATT can be rewritten as,
\[
\tau(p_1, f_{11}, f_{01}) = \left[ \int y f_{11}(y)dy - \int y f_{01}(y)dy \right],
\]
where the general definition of \(p_t\) for \(t = 0, 1\) is given by:
\[
p_t = \mathbb{P}(T_i = t) = (1-t) + (2t-1)\int \pi(U_i)dF(U_i).
\]
In typical observational studies, the identifiable distributions are \(p_1, f_{00}(y)\) and \(f_{11}(y)\). Observational data, however, provide no information about the distributions of the counterfactual outcomes, \(f_{01}(y)\) and \(f_{10}(y)\). Researchers, therefore, invoke additional assumptions to point-identify the causal effects of interest. For example, the standard assumption of unconfoundedness implies,
\[
f_{01}(y) = f_{00}(y) \quad \text{and} \quad f_{10}(y) = f_{11}(y)
\]
for all \(y \in \mathbb{R}\) (Rosenbaum and Rubin, 1983b). Unfortunately, these assumptions are not directly testable and hence are often not credible in practice.

Instead, we consider an alternative approach that imposes restrictions on the relations between the unidentifiable distributions, \((f_{10}(y), f_{01}(y))\), and the identifiable ones, \((f_{11}(y), f_{00}(y))\). There are many such restrictions, and we show how they can be used to bound the causal effects under the distributionally robust optimization framework.

Returning to the ATT example, suppose that the degree of confoundedness due to the unobserved variables \(U_i\) is assumed to be sufficiently small. This assumption can be expressed as the closeness between the unidentifiable distribution \(f_{01}(x)\) and the identifiable one \(f_{11}(y)\) instead of assuming their distributional equality as done in Equation (6). Specifically, using total variation distance \(\delta(\cdot, \cdot)\), we can write this alternative assumption as,
\[
S_{TV}(\Lambda) = \{f_{01}(y) \mid \delta(f_{01}(y), f_{00}(y)) \leq \Lambda\},
\]
where \(\Lambda\) represents the maximal total variation distance between \(f_{01}\) and \(f_{00}\).

Under the framework of distributionally robust optimization, \(S_{TV}\) forms an ambiguity set that defines the set of potential distributions for \(f_{01}\). If we wish to obtain the lower bound of the ATT, we consider the following robust optimization problem:
\[
\bar{\tau} = \min_{f_{01} \in S_{TV}(\Lambda)} \tau(p, f_{11}, f_{01}).
\]
The solution, \( \tilde{\tau} \), represents the most conservative ATT among all the distributions that are consistent with this ambiguity set.

In general, we may consider any valid ambiguity set \( \mathcal{S} \) over \((f_{10}, f_{01})\) and define the causal effect of interest as \( \Psi(p_1, f_{11}, f_{00}, f_{10}, f_{01}) \). Suppose, without loss of generality, that a greater value of the causal effect is desirable. Then, the distributionally robust causal inference for \( \Psi \) under the ambiguity set \( \mathcal{S} \) can be written as the following optimization problem:

\[
\min_{f_{10}, f_{01} \in \mathcal{S}} \Psi(p_1, f_{11}, f_{00}, f_{10}, f_{01}),
\]

where the empirical optimization problem replaces \( p_1, f_{11}, f_{00} \) with its empirical estimates.

We next demonstrate how this proposed distributionally robust causal inference framework can be applied to various settings.

### 2.3 The Marginal Sensitivity Model

We first show that our distributionally robust causal inference framework introduced above encompasses the commonly used marginal sensitivity model of Tan (2006) as a special case. The marginal sensitivity model implies that the odds ratio of treatment assignment probability with and without conditioning on the unobserved pre-treatment covariates \( U_i \) is bounded by the sensitivity parameter \( \Gamma \geq 1 \) with probability one,

\[
\frac{1}{\Gamma} \leq \frac{\pi(X_i, U_i)/(1 - \pi(X_i, U_i))}{\pi(X_i)/(1 - \pi(X_i))} \leq \Gamma,
\]

where \( e(X_i) = \mathbb{P}(T_i = 1 | X_i) \). If we simplify the notation by suppressing the conditioning on observed confounders \( X_i \) as done above, then this equation becomes:

\[
\frac{1}{\Gamma} \leq \frac{\pi(U_i)/(1 - \pi(U_i))}{p_1/p_0} \leq \Gamma,
\]

Let us consider the standard IPW estimator of the ATT, which can be written as:

\[
\frac{1}{n_1} \sum_{i=1}^{n} Y_i T_i - \frac{1}{n} \sum_{i=1}^{n} \frac{\pi(U_i)}{1 - \pi(U_i)} Y_i (1 - T_i).
\]

where \( n_1 \) represents the number of treated units and \( n \) is the total number of units. Under the marginal sensitivity model with the sensitivity parameter \( \Gamma \), robust causal inference based on the IPW estimator of the ATT can be written as:

\[
\min_{\pi(U_i)} \frac{1}{n_1} \sum_{i=1}^{n} Y_i T_i - \frac{1}{n} \sum_{i=1}^{n} \frac{\pi(U_i)}{1 - \pi(U_i)} Y_i (1 - T_i),
\]

s.t. \( \frac{p_1}{p_0} \frac{\pi(U_i)}{1 - \pi(U_i)} \leq \frac{\Gamma p_1}{p_0}, \ \forall i \)

\[
\sum_{i: T_i = 0} \frac{\pi(U_i)/(1 - \pi(U_i))}{p_1/p_0} = n_0, \quad \sum_{i: T_i = 1} \frac{p_1/p_0}{\pi(U_i)/(1 - \pi(U_i))} = n_1.
\]
The following proposition shows that the marginal sensitivity model can be written as a special case of the proposed distributionally robust causal inference framework. All proofs appear in the supplementary appendix.

**Proposition 1** (Marginal sensitivity model). The marginal sensitivity model given in Equations (11)–(13) is equivalent to the following distributionally robust causal inference problem:

\[
\min_{f_{01} \in S_{01}^{MS}(\Gamma)} \tau(p, f_{11}, f_{01}),
\]

with the following ambiguity sets,

\[
S_{01}^{MS}(\Gamma) = \left\{ f_{01}(y) \mid F_{01}(y) = \hat{F}_0(y; w), \sum_{i : T_i = 0} w_i = 1, \frac{1}{\Gamma} \leq n_0 w_i \leq \Gamma \forall i \right\},
\]

where \( \Gamma \geq 1 \) and \( \hat{F}_t(y; w) \) is the weighted empirical CDF of observed outcome under the treatment condition \( T_i = t \) and a vector of observational weights \( w \).

Proof is given in Appendix A.1.

This ambiguity set provides an intuitive interpretation. For example, the set of all possible distributions for \( f_{01}(y) \) are those created by weighting samples of \( f_{00}(y) \) with an undersampling or oversampling factor of at most \( \Gamma \). Therefore, the marginal sensitivity model essentially assumes that the \( f_{01}(y) \) distribution is similar to the \( f_{00}(y) \) distribution up to some reweighting.

One important feature of the marginal sensitivity model is that when modeling the distribution of a counterfactual outcome \( f_{01}(y) \), it uses the information from the distribution of observed outcome under the control condition, i.e., \( f_{00}(y) \), rather than from \( f_{11}(y) \), which is the distribution of observed outcome under the treatment condition. This approach will yield a non-informative bound if the unobserved confounders affect the outcome in a substantial way, leading to a large value of \( \Gamma \). In such settings, the distance between \( f_{01}(y) \) and \( f_{00}(y) \) may be too great for the marginal sensitivity model to be useful. We now propose an alternative sensitivity model, called “distributional sensitivity model,” that addresses this limitation.

### 2.4 The Distributional Sensitivity Model

The distributional sensitivity model is based on the assumption that the distribution of the observed outcome of the treatment group \( f_{11}(y) \) is sufficiently informative about the distribution of its counterfactual outcome \( f_{01}(y) \). Similarly, if one is interested in the average treatment effect for the control (ATC), we may assume that \( f_{00}(y) \) is informative about \( f_{10}(y) \) for the control group. This assumption is credible if the treatment effect is much smaller than the effect of unobserved confounders on the outcome. In such settings, the within-group comparison is likely to be more informative than the comparison between
the treatment and control groups, which is the basis of the marginal sensitivity model. In addition, unlike the marginal sensitivity model, the distributional sensitivity model allows for the potential lack of overlap between the treatment and control groups.

Specifically, in the case of the ATT, the distributional sensitivity model assumes that \( f_{01}(y) \) and \( f_{11}(y) \) have a similar distribution up to a location shift \( c \). This type of location shift model has been used as a justification for the use of rank-sum tests in randomized experiments (Lehmann, 2006). The ambiguity set for \( f_{01}(y) \) is given by:

\[
S_{01}^{DS}(\Gamma, \delta) = \left\{ f_{01}(y) \mid F_{01}(y) = \tilde{F}_0(y; w), \sum_{i:T_i=0} w_i = 1, \ 0 \leq w_i n_0 \leq \Gamma \ \forall i, \right. \\
\left. \min_c KS(F_{01}(y), F_{11}(y + c)) \leq \delta \right\},
\]

where \( \Gamma \geq 1 \) and \( KS(F(y), G(y)) = \max_y |F(y) - G(y)| \) represents the Kolmogorov-Smirnov distance between the distributions \( F \) and \( G \). Note that the inequality on \( w_i \) along with the normalization of the weights imply that at least \( 1/\Gamma \) of the weights would be non-zero. Unlike the marginal sensitivity model, we allow the weights to be zero to account for the potential lack of common support between \( f_{00}(y) \) and \( f_{01}(y) \). We then bound the treatment effect subject to this ambiguity set.

This ambiguity set for the distributional sensitivity model is similar to the one used for the marginal sensitivity model. They both construct the distribution of counterfactual outcome for the treated group \( f_{01}(y) \) as a weighted distribution of the observed outcome for the control group \( f_{00}(y) \). The key difference, however, is that under the distributional sensitivity model, \( f_{01}(y) \) is assumed to have a distributional shape similar to that of \( f_{11}(y) \) up to a location shift.

Next, we show that this distributional shape constraint on \( F_{01} \) holds if the heterogeneity of treatment effect due to unobserved variables \( U_i \) is sufficiently small. Such an assumption may be more credible than the assumption of the marginal sensitivity model that limits the impact of unobserved variables on the treatment and outcome.

**Proposition 2.** Assume that \( F_{01} \) and \( F_{11} \) are minimally \( k \)-Lipschitz, where \( k > 0 \) is the smallest real number that satisfies for all \( x, y \in \mathbb{R} \):

\[
|F_{01}(x) - F_{01}(y)| \leq k|x - y|, \quad |F_{11}(x) - F_{11}(y)| \leq k|x - y|
\]

Then, we have:

\[
\min_c KS(F_{01}(y), F_{11}(y + c)) \leq 3 \left( \frac{k \sigma_{01}}{2} \right)^{2/3},
\]

where \( \sigma_{01} = \sqrt{\mathbb{V}[Y_i(1) - Y_i(0) \mid T_i = 1]} \).

Proposition 2 characterizes an upper bound of the location-shift KS distance in terms of the heterogeneity of treatment effect due to unobserved variables. In particular, under mild
regularity conditions, the KS distance is small if the outcome distributions more diffuse than the distribution of treatment effect (i.e., \( k \ll 1/\sigma_0 \)). Note that in most common distribution families, \( k \) is inversely proportional to the standard deviation. For example, a minimally \( k \)-Lipschitz uniform, normal, and exponential distribution has the variance of exactly \( 1/12k^2 \), \( 1/2\pi k^2 \), and \( 1/k^2 \), respectively. Thus, if both \( F_01 \) and \( F_{11} \) follow one of these distributions, the upper bound is given by,

\[
\min_c KS(F_{01}(y), F_{11}(y + c)) \leq \begin{cases} 
\left( \frac{3\sigma_0}{\sigma} \right)^{2/3} & \text{for uniform} \\
3 \left( \frac{\sigma_0}{2\sqrt{2}\pi\sigma} \right)^{2/3} & \text{for normal} \\
3 \left( \frac{\sigma_0}{2\pi} \right)^{2/3} & \text{for exponential}
\end{cases}
\]

respectively, where \( \sigma = \max\{\sqrt{\mathbb{E}[Y_i(1) - T_i = 1]}, \sqrt{\mathbb{E}[Y_i(0) - T_i = 1]}\} \). Thus, in these cases, our KS distance bound scales as \( O\left((\sigma_0/\sigma)^{2/3}\right) \). Note that the \( 2/3 \) exponent can be tightened if we assume the existence of higher moments of the distribution of the treatment effect.

In sum, our distributional shape constraint holds with a small value of \( \delta \) if the heterogeneity of treatment effect is smaller than that of the outcome variable, i.e., \( \sigma_{01} \ll \sigma \).

Beyond its connection to the heterogeneity of treatment effect, another distinct feature of the distributional sensitivity model is the way it uses observed data. Indeed, at first sight, it may appear that the distributional sensitivity model utilizes information from \( f_{01} \) and \( f_{11} \) in an asymmetric way. Specifically, we use the weighted samples from \( f_{00} \) to estimate \( f_{01} \), while bounding sampling weights \( w_i \) by limiting the difference in shape between \( f_{01} \) and \( f_{11} \) after a location shift. We show, however, that there exists a duality between bounds on sampling weights and metric bounds. This is because a restriction on sampling weights implies a greater degree of similarity between the two distributions. The following theorem establishes that a bound on sampling weights can be rewritten as a bound on a distance metric between \( f_{01} \) and \( f_{00} \).

**Theorem 1** (Duality between sampling weights and distance metrics). The ambiguity set of the distributional sensitivity model, \( S_{01}^{DS}(\Gamma, \delta) \) in Equation (14), can be equivalently characterized as:

\[
S_{01}^{DS}(\Gamma, \delta) = \left\{ f_{01}(y) \mid F_{01}(y) = \hat{F}_0(y; w), \sum_{i: T_i=0} w_i = 1, \ w_i \geq 0, \right. \\
\left. d_0(F_{01}(y), F_{00}(y)) \leq \frac{\Gamma - 1}{n_0}, \ d_1(F_{01}(y), F_{11}(y)) \leq \delta \right\}, \tag{15}
\]

where \( d_1(F, G) = \min_c KS(F(y), G(y + c)) \) is a metric, and

\[
d_0(F, G) = \begin{cases} 
\max_y \lim_{\epsilon \to 0^-} \left( F(y) - G(y) \right) - \left( F(y + \epsilon) - G(y + \epsilon) \right) & 2 \leq \Gamma, \\
\max_y \max_{\epsilon \to 0^-} \left( F(y) - G(y) \right) - \left( F(y + \epsilon) - G(y + \epsilon) \right), 0) & 1 \leq \Gamma < 2
\end{cases}
\]
over the space of piecewise constant cumulative distribution functions, is a quasimetric when 1 \leq \Gamma < 2 and a metric when \Gamma \geq 2.

To implement the distributional sensitivity model, we rewrite Equation (14) as a mixed integer linear optimization problem, which can be solved using existing software packages such as Gurobi Optimization (2020). Specifically, for the $KS$ distance metric constraint, we note that the size of the location shift $c$ is bounded as follows,

$$ \min_{c \in \mathbb{R}} KS(F_{01}(y), F_{11}(y + c)) = \min_{|c| \leq \max_i |Y_i - \min_i Y_i|} KS(F_{01}(y), F_{11}(y + c)). $$

Thus, we discretize $c$ with a small value of $\epsilon := |\max_i Y_i - \min_i Y_i|/m > 0$, and then optimize over the set $L = \{-|\max_i Y_i - \min_i Y_i|, -|\max_i Y_i - \min_i Y_i| + \epsilon, \ldots, -|\max_i Y_i - \min_i Y_i| + 2m\epsilon\}$. This yields,

$$ \min_{c \in \mathbb{R}} KS(F_{01}(y), F_{11}(y + c)) \approx \min_{j \in \{0, 1, \ldots, 2m\}} KS(F_{01}(y), F_{11}(y - |\max_i Y_i - \min_i Y_i| + j\epsilon)) $$$$ = \min_{j \in \{0, 1, \ldots, 2m\}} \max_{y} |F_{01}(y) - F_{11}(y + c_0 + j\epsilon)| $$

$$ \approx \min_{j \in \{0, 1, \ldots, 2m\}} \max_{k \in \{0, 1, \ldots, 2m\}} |F_{01}(\min_i Y_i + k\epsilon) - F_{11}(\min_i Y_i + c_0 + (j + k)\epsilon)|, \quad (16) $$

where $c_0 = -|\max_i Y_i - \min_i Y_i|$. Using this discretization, we can rewrite the optimization problem as the following mixed-integer linear (feasibility) optimization problem:

$$ \min_{w, d^k, d, z} \frac{1}{n_1} \sum_{i : T_i = 1} Y_i - \sum_{i : T_i = 0} w_i Y_i $$

subject to

$$ \sum_{i : T_i = 0} w_i = 1 \quad (17) $$

$$ 0 \leq w_i \leq \frac{\Gamma}{n_0} \quad \forall i \in \{1, \ldots, n\} \quad (18) $$

$$ d \leq \delta \quad (19) $$

$$ d \geq d_j^k - z_j \quad \forall j \in \{0, 1, \ldots, 2m\} \quad (20) $$

$$ d_j^k \geq F_{01}(\min_i Y_i + k\epsilon) - F_{11}(\min_i Y_i + c_0 + (j + k)\epsilon) \quad \forall j, k \in \{0, 1, \ldots, 2m\} \quad (21) $$

$$ d_j^k \geq F_{11}(\min_i Y_i + c_0 + (j + k)\epsilon) - F_{01}(\min_i Y_i + k\epsilon) \quad \forall j, k \in \{0, 1, \ldots, 2m\} \quad (22) $$

$$ \sum_{i} z_j = 2m - 1 \quad \text{where } z_j \in \{0, 1\} \quad \forall j \in \{0, 1, \ldots, 2m\}. \quad (23) $$

2.5 The Difference-in-Differences Design

We now extend the distributional sensitivity model to the difference-in-differences (DiD) design where the baseline (i.e., pre-treatment) outcome is available for both the treatment and
control groups. We use $Y_b(0, U)$ to denote the baseline outcome that is realized prior to the administration of the treatment. Under this setting, therefore, along with $(f_{00}, f_{01}, f_{10}, f_{11})$, we can define two additional distributions:

$$f_{bt}(y) = P(Y_b(0, U) = y \mid T(U) = t)$$  \hspace{1cm} (24)

for $t = 0, 1$. Note that we observe samples from both distributions $f_{bt}$ and $f_{bt'}$.

Under the DiD design, researchers typically assume the following parallel trend assumption to estimate the counterfactual outcome mean for the treated units,

$$E[Y(0, U) \mid T(U) = 1] = E[Y_b(0, U) \mid T(U) = 1] + \{E[Y(0, U) \mid T(U) = 0] - E[Y_b(0, U) \mid T(U) = 0]\}. \hspace{1cm} (25)$$

This assumption, however, may be violated in practice. Thus, we consider the following relaxation by allowing an $\epsilon$ distance between the distributions of $Y$ on the left and right hand side of Equation (25) under some distance function $d$:

$$d(F_{01}, F_{b1+00-b0}(y)) \leq \epsilon, \hspace{1cm} (26)$$

where $F_{b1+00-b0}(y) = P(Y_b(0, U_1) + Y(0, U_0) - Y_b(0, U_0) \leq y \mid T(U_1) = 1, T(U_0) = 0)$ is identifiable from the observed data. In particular, we can choose the function $d$ to be the difference in expectations, i.e., $d(F(x), G(x)) = |E_F(x) - E_G(x)|$, so that it is interpretable and we can recover the commonly used DiD assumption when the distance is zero.

This leads to the following distributional sensitivity model,

$$\min_{f_{01} \in S^{DID}_{01}(\Gamma, \delta, \epsilon)} \Psi(p_1, f_{11}, f_{00}, f_{10}, f_{01}), \hspace{1cm} (27)$$

where

$$S^{DID}_{01}(\Gamma, \delta, \epsilon) = \left\{ f_{01}(y) \mid F_{01}(y) = \hat{F}_0(y; w), \sum_{i: T_i = 0} w_i = 1, \right. $$

$$\left. 0 \leq w_i \leq \frac{\Gamma}{n_0} \forall i, \min c KS(F_{01}(y), F_{11}(y + c)) \leq \delta, \right. $$

$$\left. d(F_{01}(y), F_{b1+00-b0}(y)) \leq \epsilon \right\}. \hspace{1cm} (28)$$

Furthermore, the above distributional sensitivity model can be extended to the nonlinear change-in-changes (CIC) model proposed by Athey and Imbens (2006). The key assumption of the nonlinear CIC model can be written as,

$$F_{01}(y) = F_{b1}(F_{b0}^{-1}(F_{00}(y))). \hspace{1cm} (29)$$

We consider a relaxation of this assumption by allowing an $\epsilon$ distance between the distributions on the two sides of Equation (29). Note that we can estimate $F_{b1}(F_{b0}^{-1}(F_{00}(y)))$.
consistently. Thus, the distributional sensitivity model for the nonlinear CIC model can be written as:

$$\min_{f_{01} \in S_{01}^{CIC}(\Gamma, \delta, \epsilon)} \Psi(p_1, f_{11}, f_{00}, f_{10}, f_{01}),$$  \hspace{1cm} (30)$$

where

$$S_{01}^{CIC}(\Gamma, \delta, \epsilon) = \left\{ f_{01}(y) \mid F_{01}(y) = \hat{F}_0(y; w), \sum_{i: T_i = 0} w_i = 1, \right. \left. 0 \leq w_i \leq \frac{\Gamma}{n_0} \quad \forall i, \min_c KS(F_{01}(y), F_{11}(y + c)) \leq \delta, \right. \left. d(F_{01}(y), F_{b1}(F_{00}(y))) \leq \epsilon \right\}. \hspace{1cm} (31)$$

2.6 Instrumental Variables

We next show that the proposed distributionally robust causal inference framework can also be applied to instrumental variables methods (Angrist et al., 1996). Specifically, we first examine the estimation of the ATT, which is not identifiable, and then investigate the robustness of an instrumental variables (i.e., Complier Average Treatment Effect) estimate to the potential violation of exclusion restriction. For simplicity, we consider the settings, in which the treatment assignment (rather than actual treatment receipt) is either randomized or unconfounded given a set of pre-treatment covariates.

Specifically, let $Z_i$ represent a binary encouragement variable which is equal to 1 if unit $i$ is encouraged to receive the treatment and is equal to 0 otherwise. We use $T_i \in \{0, 1\}$ to represent the indicator variable for the actual receipt of treatment. We use $T_i(z, U_i)$ to denote the potential value of the treatment receipt variable under the encouragement condition $Z_i = z$ where $U_i$ represents the unobserved confounders. The actual treatment is then given by $T_i = T_i(Z_i, U_i)$.

Throughout this section, along with the unconfoundedness of the instrument, we assume the following monotonicity assumption, which is one of the main assumptions of instrumental variables estimation,

$$T(1, U) \geq T(0, U). \hspace{1cm} (32)$$

In the current setting, the assumption implies that there is no defier who would receive the treatment only when they are not encouraged. Finally, we can define the potential outcome as $Y(t, z, U)$ where the observed outcome is equal to $Y = Y(T, Z, U)$. Note that we do not impose the exclusion restriction assumption, which states that the instrument affects the outcome only through the treatment. This allows us to evaluate robustness against a potential violation of the exclusion restriction.

Define the following conditional distributions of potential outcomes,

$$f_{tz}(y) = P(Y(t', z', U) = y \mid T_i(z, U) = t, Z = z), \hspace{1cm} (33)$$
for \( t, t', z, z' \in \{0, 1\} \). Note that we can only identify the distributions \( f_{t z}^{t' z'} \) from the observed data where \( t = t' \) and \( z = z' \).

Under this setting, the ATT is defined as,

\[
\text{ATT} = \frac{p_{11}}{p_1} \left( \int y f_{11}^{11}(y) dy - \int y f_{11}^{01}(y) dy \right) + \frac{p_{10}}{p_1} \left( \int y f_{10}^{10}(y) dy - \int y f_{10}^{00}(y) dy \right),
\]

where \( p_{t z} = n_{t z} / n \), \( p_t = p_{t0} + p_{t1} \), and \( n_{t z} = \sum_{i=1}^{n} I\{T_i = t, Z_i = z\} \) denote the sample size and proportion for each observed strata defined by \( T_i = t \) and \( Z_i = z \), respectively, i.e., and. Thus, we need to estimate the unidentifiable distributions, i.e., \( f_{0 z}^{0 z} \) with \( z = 0, 1 \).

Using our distributionally robust causal inference framework, we use the identifiable distribution \( f_{0 z}^{0 z} \) to infer the unidentifiable distribution \( f_{0 z}^{0 z} \) by assuming that \( f_{1 z}^{0 z} \) has a similar shape as \( f_{0 z}^{0 z} \) for \( z = 0, 1 \) up to a location shift of unknown size. Specifically, the ambiguity set for \( f_{1 z}^{0 z} \) under the distributional sensitivity model can be given by:

\[
S_{z}^{IV}(\Gamma, \delta) = \left\{ f_{1 z}^{0 z}(y) \mid F_{1 z}^{0 z}(y) = \hat{F}_{0 z}^{0 z}(y; w), \sum_{i:T_i=0, Z_i=z} w_i = 1, \right. \\
0 \leq w_i \leq \frac{\Gamma}{n_{0 z}} \forall i: T_i = 0, Z_i = z, \min_{c} KS(F_{1 z}^{0 z}(y), F_{1 z}^{1 z}(y + c)) \leq \delta \},
\]

for \( z = 0, 1 \). Under this setup, the standard exclusion restriction implies the following distributional equality that couples the unknown distributions,

\[
f_{t z}^{t 0} = f_{t z}^{t 1} \text{ for } t, z, z' \in \{0, 1\}.
\]

This is the distributional representation of the exclusion restriction assumption that \( Z \) only affects \( Y \) through \( T \). Note that in particular, the unknown distributions of interest in ATT \( f_{1 z}^{0 z} \) are coupled with the unknown distributions \( f_{1 z}^{0 1-z} \) as these constraints. We relax this assumption by allowing for a small violation of this assumption. Specifically, we consider an \( \epsilon \) distance between the distributions on the two sides of the equality in some distance function \( d \):

\[
d(F_{1 z}^{0 z}, F_{1 z}^{0 1-z}) \leq \epsilon \text{ for } z \in \{0, 1\}.
\]
Then, robust causal inference under the distributional sensitivity model that incorporates a relaxed version of exclusion restriction can be written as,

\[ S^IV_\varepsilon(\Gamma, \delta, \epsilon) = \left\{ f_{1z}(y), f_{1z-1}(y) \mid F_{1z}(y) = F_{1z}(y; w), \ F_{1z-1}(y) = F_{1z-1}(y; w) \right\} \]

\[
\sum_{i:T_i=0, Z_i=z} w_i = 1, \sum_{i:T_i=0, Z_i=1-z} w_i = 1, \\
0 \leq w_i \leq \frac{\Gamma}{n_{01}} \forall i : T_i = 0, Z_i = 1, \ 0 \leq w_i \leq \frac{\Gamma}{n_{00}} \forall i : T_i = 0, Z_i = 0, \quad (35) \\
\min_c KS(F_{0z}(y), F_{1z}(y + c)) \leq \delta, \min_c KS(F_{1z-1}(y), F_{1z-1}(y + c)) \leq \delta, \\
d(F_{1z}, F_{1z-1}) \leq \epsilon. \]

### 2.7 Incorporating High Dimensional Covariates

So far, we have focused on the settings, in which we condition on each value of covariates \( X_i \). In many observational studies, however, there exist a large number of pre-treatment covariates that need to be adjusted for. To handle such cases, we generalize our distributionally robust causal inference framework by directly controlling the differences in the observed covariates between the treatment and control groups.

Specifically, suppose that we have a total of \( J \) covariates. Let \( X_{ij} \) denote the \( j \)th covariate for unit \( i \) where \( j = 1, 2, \ldots, J \), and \( g_t(X_{ij}) \) represent the density function of \( X_{ij} \) for the units with the treatment status \( T_i = t \) where \( t = 0, 1 \). We use \( C(x_j) \) to denote the ambiguity set for the \( j \)th covariate. Then, the general optimization problem under our distributionally robust causal inference framework becomes,

\[
\min_{f_{10}(y), f_{00}(y) \in S(y), g_0(x_j), g_1(x_j) \in C(x_j) \forall j} \Psi(p_1, f_{11}, f_{00}, f_{10}, f_{01}), \quad (36)
\]

where \( S(y) \) is the distributionally robust set for the unobserved counterfactual outcomes (as in Sections 2.3 and 2.4), and \( C(x_j) \) are the ambiguity sets in which we limit the difference in the distribution of \( x_j \) under \( T = 0 \) and \( T = 1 \) to ensure covariate matching.

For illustration, we revisit the distributional sensitivity model for the ATT considered in Section 2.4. Under the high-dimensional setting, we may require \( C(x_j) \) to be such that the \( L_1 \) distance between first moments of \( X \) between \( f_{11} \) and \( f_{01} \) to be less than \( \epsilon \) (Zubizarreta, 2012):

\[
\sum_{j=1}^{J} \frac{1}{n_1} \sum_{i:T_i=1} X_{ij} - \frac{1}{n_0} \sum_{i:T_i=0} w_i X_{ij} \leq \epsilon. \quad (37)
\]
The equivalent Lagrangian form is,

$$\min_{w \in S_{01}^{\delta}(\Gamma, \delta)} \sum_{i: T_i = 1} Y_i - \frac{1}{n_0} \sum_{i: T_i = 0} w_i Y_i + \lambda \sum_{l=1}^J \left| \sum_{i: T_i = 1} X_{ij} - \frac{1}{n_0} \sum_{i: T_i = 0} w_i X_{ij} \right|.$$  (38)

We can then utilize this Lagrangian objective function and implement the constraints for the distributional sensitivity model as detailed in Equations (17)–(23).

3. Simulation Experiments

We conduct a simulation study to understand the empirical performance of the distributional sensitivity model. Specifically, we generate the data based on the following potential outcomes model,

$$T_i(u) = \text{Bern}(0.6u + 0.2),$$

$$Y_i(t, u) = (1 - u)(t - 0.5)v_i + u(t - 0.5)\eta_i + \theta_i + \epsilon_i,$$

where $v \sim N(0,1)$, $\nu_i \sim N(\tau_1, 1)$, $\eta_i \sim N(\tau_2, 1)$, $u \sim \text{Bern}(p)$, $\theta_i \sim N(0, 2)$ and $\epsilon_i \sim N(0, 0.1)$. Here, $u$ is an unobserved confounder such that when $u = 1$, $P(T = 1) = 0.8$ and $E[Y(1) - Y(0) | u = 1] = 0.8\tau_2 + 0.2\tau_1$, and when $u = 0$, $P(T = 1) = 0.2$ and $E[Y(1) - Y(0) | u = 1] = 0.2\tau_2 + 0.8\tau_1$. When $\tau_2 > \tau_1$, $u$ assigns a higher treatment probability to an individual with a higher treatment effect, and vice versa when $\tau_2 < \tau_1$. We also note that by design, all of the $f_{11}, f_{00}, f_{10}, f_{01}$ distributions are different so that no framework has a clear advantage.

The causal quantity of interest is the average treatment effect on the treated (ATT), which can be written as the following function of model parameters:

$$E[Y(1) - Y(0) | T = 1] = \frac{8p\tau_2 + 2(1 - p)\tau_1}{6p + 2}.$$}

We create multiple scenarios based on different values of $(\tau_1, \tau_2, p)$:

1. $(\tau_1, \tau_2, p) = (2, 3, 0.5),$
2. $(\tau_1, \tau_2, p) = (3, 2, 0.5),$
3. $(\tau_1, \tau_2, p) = (2, 3, 0.8).$

For each scenario, we generate a dataset with $n \in \{100, 200, 500\}$ samples over 1,000 independent runs. For each run, we examine the distributional sensitivity model of Section 2.4 with $\delta = 0.1$ and the marginal sensitivity model across different values of $\Gamma$ (we include experiments on other $\delta$ values in Appendix A.4). Across both sensitivity models, the value of $\Gamma$ is comparable because it provides identical upper bounds on the weights $w_i$.
Table 1 presents the bias and standard deviation of the ATT estimate for distributional and marginal sensitivity models, using their respective lower bound. As expected, both methods return conservative estimates of the ATT. We also find that the bias remain relatively
similar across different scenarios. However, the distributional sensitivity models generally produce less conservative results compared to the marginal sensitivity models, with a growing difference as we increase $\Gamma$. Appendix A.4 shows that this result still holds even if we increase $\delta$. In particular, at high levels of robustness, the distributional sensitivity model is able to estimate the ATT with a bias whose magnitude is half of the corresponding bias for the marginal sensitivity model. This shows that by using both $f_{00}$ and $f_{11}$, the distributional sensitivity model is able to generate less conservative estimates under a wide range of parameter values.

4. Empirical Evaluations

For empirical demonstration, we apply the proposed distributionally robust causal inference framework to the following diverse data sets:

1. **National Supported Work Demonstration (NSW) Dataset**: This is a well-known data set, which was originally used by LaLonde (1986) to evaluate the accuracy of various causal inference methods for observational studies. The data set has an experimental benchmark which we use to assess the performance of our methodology.

2. **Boston Medical Center (BMC) Diabetes Dataset**: We apply the distributional sensitivity model to this observational study and demonstrate how our model avoids a potentially erroneous medical conclusion.

3. **Student/Teacher Achievement Ratio (STAR) Dataset**: Like the analysis of the NSW data, we use the setup from Wilde and Hollister (2007) to construct a synthetic observational dataset from the original randomized STAR dataset. We then evaluate the performance of our methodology using the experimental estimate as a benchmark.

4.1 NSW Dataset

The National Supported Work Demonstration (NSW) was a temporary employment program to help disadvantaged workers by giving them work experience and counseling in a sheltered environment. Specifically, the NSW randomly assigned qualified applicants to treatment and control groups, where workers in the treatment group were given a guaranteed job for 9 to 18 months. The primary outcome of interest $Y$ is the (annualized) earnings in 1978, 36 months after the program. The experimental data in total contains $n = 722$ observations, with $n_1 = 297$ participants assigned to the treatment group and $n_0 = 425$ participants in the control group. There are 7 available pre-treatment covariates $X$ that records the demographics and pre-treatment earnings of the participants.

We consider the setup of LaLonde (1986), where non-experimental control groups were constructed from the Population Survey of Income Dynamics (PSID) and Current Population Survey (CPS). We replace the experimental controls with the non-experimental controls.
Table 2: Results of robust inference under the distributional sensitivity model for the NSW dataset with PSID controls. The table presents the estimate, standard error, and bias of the robust estimator under different robustness parameters $\Gamma$ and $\delta$.

| Model                                      | $\Gamma$ | $\delta$ | $\epsilon$ | Estimate | s.e.   | Bias   |
|--------------------------------------------|----------|----------|-------------|----------|--------|--------|
| Distributional sensitivity model           | 25       | 0.02     |             | -54.9    | 1585.0 | -940.9 |
|                                            | 12       | 0.02     |             | -163.0   | 1320.4 | -1049.0|
|                                            | 8        | 0.02     |             | -294.5   | 1166.2 | -1180.5|
|                                            | 25       | 0.03     |             | -304.3   | 1586.7 | -1190.3|
|                                            | 25       | 0.05     |             | -559.2   | 1592.8 | -1445.2|
| Distributional sensitivity model           | 25       | 0.02     | 100         | 320.7    | 1585.7 | -565.3 |
| under the difference-in-differences        | 25       | 0.02     | 200         | 220.7    | 1585.6 | -665.3 |
|                                            | 25       | 0.02     | 500         | -54.9    | 1585.0 | -940.9 |
|                                            | 25       | 0.03     | 500         | -79.2    | 1587.5 | -965.2 |
|                                            | 25       | 0.05     | 500         | -79.3    | 1591.0 | -965.3 |
| Regression (LaLonde, 1986)                 |          |          |             | -1228.0  | 896.0  | -2114.0|

Table 2: Results of robust inference under the distributional sensitivity model for the NSW dataset with PSID controls. The table presents the estimate, standard error, and bias of the robust estimator under different robustness parameters $\Gamma$ and $\delta$.

and apply the distributional sensitivity model $S_{01}^{DS}(\Gamma, \delta)$ to robustly infer the ATT. Since the treated units come from the randomized experiment, we use the estimated average treatment effect of $\$886$ in the original experiment as a benchmark ATT estimate.

To prevent numeric issues due to the wide range of incomes, we log-transform the outcome variable before applying our methodology. We also impose matching on the first moments using Equation (38) with $\lambda = 1000$, which was selected so that the characteristic magnitude of the Lagrangian objective is significantly larger than the ATT objective, enforcing best-possible first moment matching. We chose $\Gamma$ such that the minimum number of selected samples is 30, 60, and 100, corresponding to a $\Gamma$ of 25, 12, and 8 respectively. We further vary $\delta$ between 0.02 and 0.05 as without any weighting, the distributional KL distance between $Y(1)$ and $Y(0)$ is just over 0.2. Lastly, we consider the distributional sensitivity model under the difference-in-differences design as described in Section 2.5, where we treat the 1975 income as the baseline outcome, and utilize the mean difference metric $d(F, G) = |\mu(F) - \mu(G)|$.

The results for the PSID dataset are shown in Table 2. For the standard errors under the distributional sensitivity model, we calculate the standard errors by treating the weights as fixed and computing the conditional weighted standard error. It is well known that the experimental results based on the PSID datasets are difficult to recover (Dehejia and Wahba, 1999). We observe that linear regression model produces large negative biases relative to the benchmark value. In contrast, the distributional sensitivity model is designed to be conservative, but generally produces smaller biases across a wide range of robustness parameters. In particular, under the difference-in-differences design, the distributional sensitivity model yields even smaller bias.
Distributionally Robust Causal Inference

Table 3: Results of robust inference under the distributional sensitivity model and linear regression for the NSW dataset with different control data sets from LaLonde (1986). The linear regression results are from LaLonde (1986). We choose \( \Gamma = n_1/100 \) and \( \delta = 0.02 \) for these experiments. The table presents the estimate, standard error, and bias of the robust estimator.

| Dataset | Distributional sensitivity model | Linear regression |
|---------|---------------------------------|-------------------|
|         | Estimate | s.e. | Bias | Estimate | s.e. | Bias |
| PSID    | −54.9    | 1585.0 | −940.9 | −1228.0 | 896.0 | −2114.0 |
| CPS     | −252.6   | 1003.7 | −1138.6 | −805.0   | 484.0  | −1691.0 |
| CPS2    | −190.1   | 933.6  | −1076.1 | −319.0   | 761.0  | −1205.0 |
| CPS3    | 408.2    | 922.4  | −477.8  | 1466.0   | 984.0  | 580.0  |

To further illustrate the robustness of our results, we apply the distributional sensitivity model to the other non-experimental datasets from LaLonde (1986) (PSID2 and PSID3 were removed due to data issues), and compare our results with the resulted from the linear regression controlled on all variables from LaLonde (1986). Table 3 shows that our estimates are again conservative, as expected, but the biases are all approximately smaller than the standard error. The absolute magnitude of the biases from our methodology is consistently lower than the bias achieved through regression. Together with the results in Table 2, our findings demonstrate that the distributional sensitivity model is able to deliver robustness guarantees without incurring a large bias.

4.2 BMC Dataset

We next apply the distributional sensitivity model to the BMC dataset. The BMC dataset consists of visit records of 10,806 Type II diabetic patients from 1999 to 2014. Specifically, during each visit, the doctor would prescribe a treatment regimen for the patient for the next treatment period. The outcome variable is the change in Hemoglobin A1c (HbA1c), a key indicator of blood glucose, between the visit and the end of the treatment period. A negative outcome value is desirable because the key objective of the treatment regimes is to lower the blood glucose levels.

To avoid the complications due to potential carryover effects between visits, we focus only on the set of first patient visits. We construct a binary treatment by focusing on two commonly-prescribed treatments: the Metformin monotherapy (MET) and the Insulin monotherapy (INS). In total, we have \( n = 2,538 \) valid visits, where \( n_1 = 1869 \) visits prescribed Metformin and \( n_0 = 669 \) prescribed insulin. There are 22 pre-visit covariates that describe demographics, treatment history, and comorbidities of the patient.

We are interested in the ATT as well as the average treatment effect for the control (ATC). We apply the distributional sensitivity model while balancing the first moments of all covariates with \( \lambda = 1000 \) to enforce best-possible first moment matching. We choose the same sen-
Table 4: Results of the distributional sensitivity model on the BMC dataset. We show the estimates of both observed and counterfactual mean outcomes along with the estimates of the ATT and ATC.

| Method                              | $E[Y(1) \mid T = 1]$ | $E[Y(0) \mid T = 1]$ | ATT     | $E[Y(1) \mid T = 0]$ | $E[Y(0) \mid T = 0]$ | ATC  |
|-------------------------------------|------------------------|------------------------|---------|------------------------|------------------------|------|
| Doubly Robust                       | $-0.87$                | $-0.12$                | $-0.75$ | $-1.66$                | $-0.89$                | $-0.77$|
| Causal Forests                      | $-0.87$                | $-0.47$                | $-0.70$ | $-1.84$                | $-0.89$                | $-0.96$|
| Distributional sensitivity model     | $-0.87$                | $-0.51$                | $-0.36$ | $-0.72$                | $-0.89$                | $0.17$|

The results are shown in Table 4. Our outcome variable of interest is the change in HbA1c level, therefore the estimated $E[Y(1)]$ and $E[Y(0)]$ represent our estimates of the change in HbA1c for the treated groups and untreated groups specifically, while ATT and ATC records the estimated change in HbA1c that results from the treatment. We find that the estimates based on Causal Forests and the doubly-robust estimator have negative estimates of both ATT and ATC. This suggests that being prescribed Metformin lowers the level of blood glucose, benefiting patients.

However, the negative ATC estimate is primarily due to a large negative estimate of the counterfactual mean outcome, i.e., $E[Y(1) \mid T = 0]$. Although the lack of experimental data makes it difficult to assess the accuracy of these estimates, it is highly unlikely that any single individual can produce a change of 1.5 points or greater in HbA1c in a single period. Indeed, under the standard settings, a reduction of HbA1c by 0.5 points or greater is already considered clinically significant (Spaulonci et al., 2013). Thus, the absolute outcome estimates of Causal Forests and the doubly robust estimator appear to be overstating the efficacy of MET.

In contrast, the estimates based on the distributional sensitivity model are much more realistic. The ATT estimate shows an effect of moderate size while the ATC estimate is slightly positive. The results therefore suggest that the effectiveness of MET is far from conclusive.

4.3 STAR Dataset

As the third and final application, we analyze the STAR dataset using the distributional sensitivity model. The STAR project is a randomized experiment stratified on the school level that randomly assigned over 7,000 students across 79 schools to three different groups: small class, regular class without aid, and regular class with a full-time teacher’s aid (Mosteller,
The experiment began when students entered kindergarten and continued through third grade, with assessments at the end of each grade.

We focus on the comparison of two groups — small class and regular class without aid. Our binary treatment assignment variable is equal to 1 if a student is assigned to a small class and is 0 otherwise. For the outcome variable, we consider the reading score at the end of kindergarten. There are a total of ten pre-treatment covariates, including four demographic characteristics of students (gender, race, birth month, birth year) and six school characteristics (urban/rural, enrollment size, grade range, number of students on free lunch, number of students on school buses, and percentage of white students).

We construct a non-experimental control group from the STAR by broadly following the strategy of Wilde and Hollister (2007). First, we subset the data to select 28 schools that had over 50 participating students during kindergarten. Second, to create a non-experimental control group for each school, we randomly permute at the school level for the students in the control group to simulate a setting where the control group does not come from experimental data (in this case coming from experimental data from another school). Third, for each school, we apply the distributional sensitivity model to robustly estimate the school-level ATT. Finally, we compute the stratified ATT estimate by weighting the school-level ATT estimates across all schools by the number of student participants.

The results are shown in Table 5. Standard errors are calculated using the same method as in Section 4.1. We find that when with \( \Gamma = 1.1 \), the estimate is once again not statistically significantly different from the experimental benchmark. As \( \Gamma \) grows, however, the estimated ATT quickly becomes negative. This is due to the fact that each school has a small number of samples (50–100 students), and thus a larger \( \Gamma \) results in an estimate based on the unidentifiable distributions with just a few samples. This suggests that with a small sample size, the distributional sensitivity model may yield an overly conservative estimate.

| \( \Gamma \) | \( \delta \) | Estimate | s.e.  | Bias  |
|----------|----------|----------|------|-------|
| 1.1      | 0.3      | 5.67     | 1.38 | -2.01 |
| 1.25     | 0.3      | 4.27     | 1.42 | -3.41 |
| 1.4      | 0.3      | 0.76     | 1.46 | -6.92 |
| 1.6      | 0.3      | -2.33    | 1.52 | -10.01|

Table 5: Results of the distributional sensitivity model on the STAR dataset under different robustness parameters.

5. Conclusion

In this work, we proposed a new robust causal inference framework with distributionally robust optimization. We showed how the commonly used marginal sensitivity model is a special case of our framework, and proposed a new model, the distributional sensitivity
model, that overcomes its limitation and has a wide applicability. We illustrate, on both synthetic and empirical data, that the distributional sensitivity model is able to generate estimates that are only moderately conservatively biased and offers robust guarantees.

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Appendix A. Supplementary Appendix

A.1 Proof of Proposition 1

Proof. By algebraic manipulation, the marginal sensitivity model is equivalent to:

\[
\begin{align*}
\min_{\pi} \frac{1}{n_1} \sum_{i: T_i = 1} Y_i - \frac{p_0}{n_0} \sum_{i: T_i = 0} Y_i \frac{\pi(U_i)}{1 - \pi(U_i)} \\
\text{s.t.} \quad \frac{p_1}{\Gamma p_0} \leq \frac{\pi(U_i)}{1 - \pi(U_i)} \leq \frac{\Gamma p_1}{p_0} \forall i \\
\sum_{i: T_i = 0} \frac{\pi(U_i)/(1 - \pi(U_i))}{p_1/p_0} = n_0, \quad \sum_{i: T_i = 1} \frac{p_1/p_0}{\pi(U_i)/(1 - \pi(U_i))} = n_1
\end{align*}
\]

where \( n_t = np_t \) for \( t = 0, 1 \). Define \( \lambda_i = p_0 \pi(U_i)/(p_1 (1 - \pi(U_i))) \) and we can rewrite the above formulation, after some rearrangement, as:

\[
\begin{align*}
\min_{\lambda_i} \frac{1}{n_1} \sum_{i: T_i = 1} Y_i - \sum_{i: T_i = 0} \frac{\lambda_i}{n_0} Y_i \\
\text{s.t.} \quad \frac{1}{\Gamma} \leq \lambda_i \leq \Gamma \forall i \\
\sum_{i: T_i = 0} \lambda_i n_0 = 1, \quad \sum_{i: T_i = 1} \frac{1}{n_1 \lambda_i} = 1.
\end{align*}
\]

Now, consider the empirical version of the distributionally robust causal inference problem for the ATT under our framework shown in Equation (5):

\[
\begin{align*}
\min_{f_{01}, f_{10} \in S} \frac{1}{n_1} \sum_{i: T_i = 1} Y_i - \int y f_{01}(y) dy
\end{align*}
\]

Therefore, we find that the problem in Equations (A1)–(A3) correspond exactly to the distributionally robust causal inference problem under our framework with the following ambiguity sets:

\[
S^MS_{01}(\Gamma) = \left\{ f_{01}(y) \mid F_{01}(y) = \hat{F}_0(y; w), \; \sum_{i: T_i = 0} w_i = 1, \; \frac{1}{\Gamma} \leq n_0 w_i \leq \Gamma \forall i, j \right\}
\]

\qed
A.2 Proof of Proposition 2

Denote $\mu_{01} = \mathbb{E}[Y_i(1) - Y_i(0) \mid T_i = 1]$. Then, for any $\epsilon > 0$, we have:

$$\min_c KS(F_{01}(y), F_{11}(y + c)) \leq KS(F_{01}(y), F_{11}(y + \mu_{01}))$$

$$= \max_c |\mathbb{P}(Y_i(0) \leq c \mid T_i = 1) - \mathbb{P}(Y_i(1) \leq c + \mu_{01} \mid T_i = 1)|.$$ 

$$\leq \max_c |\mathbb{P}(Y_i(0) \leq c \mid T_i = 1) - \mathbb{P}(Y_i(0) \leq c - \epsilon \land Y_i(1) - Y_i(0) \leq \epsilon + \mu_{01} \mid T_i = 1)|$$

$$\leq \max_c |\mathbb{P}(Y_i(0) \leq c \mid T_i = 1) - \mathbb{P}(Y_i(0) \leq c - \epsilon \mid T_i = 1) - \mathbb{P}(Y_i(1) - Y_i(0) \leq \epsilon + \mu_{01} \mid T_i = 1) + 1|$$

$$= \max_c \{\mathbb{P}(Y_i(0) \leq c \mid T_i = 1) - \mathbb{P}(Y_i(0) \leq c - \epsilon \mid T_i = 1)\} + 1 - \mathbb{P}(Y_i(1) - Y_i(0) \leq \epsilon + \mu_{01} \mid T_i = 1)$$

$$\leq k\epsilon + (1 - \mathbb{P}(Y_i(1) - Y_i(0) \leq \epsilon + \mu_{01} \mid T_i = 1))$$

$$\leq k\epsilon + \frac{\sigma_{01}^2}{\epsilon^2}$$

where the last two inequalities follow from the $k$-Lipschitz condition and Chebyshev’s inequality. Now, since this is true for all $\epsilon > 0$, we minimize this expression over $\epsilon$. The minimum is reached at $\epsilon = \left(\frac{2\sigma_{01}^2}{k}\right)^{1/3}$, and thus we have:

$$\min_c KS(F_{01}(y), F_{11}(y + c)) \leq 3 \left(\frac{k\sigma_{01}}{2}\right)^{2/3}$$

A.3 Proof of Theorem 1

We begin by defining the following two ambiguity sets:

$$S_{01}^{DSa}(\Gamma, \delta) = \left\{ f_{01}(y) \mid F_{01}(y) = \hat{F}_0(y; \mathbf{w}), \quad \sum_{i:T_i = 0} w_i = 1, \right.$$ 

$$\left. 0 \leq w_i \leq \frac{\Gamma}{n_0} \quad \forall i, \quad \min_c KS(F_{01}(y), F_{11}(y + c)) \leq \delta \right\},$$

and

$$S_{01}^{DSb}(\Gamma, \delta) = \left\{ f_{01}(y) \mid F_{01}(y) = \tilde{F}_1(y; \mathbf{w}), \quad \sum_{i:T_i = 0} w_i = 1, \quad w_i \geq 0, \right.$$ 

$$\left. d_0(F_{01}(y), F_{00}(y)) \leq \frac{\Gamma - 1}{n_0}, \quad d_1(F_{01}(y), F_{11}(y)) \leq \delta \right\}.$$ 

Now, suppose $f_{01}(y) \in S_{01}^{DSa}(\Gamma, \delta)$. Then, we know that

$$F_{01}(y) = \tilde{F}_1(y; \mathbf{w}) = \sum_{i:T_i = 0} w_i F_{Y_i}(y),$$

and

$$F_{01}(y) = \hat{F}_0(y; \mathbf{w}) = \sum_{i:T_i = 0} w_i F_{Y_i}(y).$$
where \( \sum_{i:T_i=0} w_i = 1 \), \( F_{Y_i}(y) = 1\{y \geq Y_i\} \), and \( 0 \leq w_i \leq \frac{1}{n_0} \) holds. Let us first consider the case where \( \Gamma \geq 2 \). Then we have, by definition of \( d_0 \):

\[
\begin{align*}
    d_0(F_{01}(y), F_{00}(y)) &= \max_{i} \left| w_i - \frac{1}{n_0} \right| \\
    &\leq \max \left\{ \frac{\Gamma - 1}{n_0}, \frac{1}{n_0} \right\} \\
    &\leq \frac{\Gamma - 1}{n_0}.
\end{align*}
\]

where we used the fact that \( F_{00}(y) = \sum_{i:T_i=0} F_{Y_i}(y)/n_0 \). Therefore, we have \( f_{01}(y) \in S^{DS}(\Gamma, \delta) \).

Next, we prove the reverse direction. Let \( f_{01}(y) \in S^{DSb}(\Gamma, \delta) \). Again, let us first consider the case where \( \Gamma \geq 2 \). By definition of \( S^{DSb}(\Gamma, \delta) \), we have:

\[
\begin{align*}
    d_0(F_{01}(y), F_{00}(y)) &= \max_{i} \left\{ w_i - \frac{1}{n_0}, 0 \right\} \\
    &\leq \frac{\Gamma - 1}{n_0}.
\end{align*}
\]

This implies \( \max_i w_i \leq \Gamma/n_0 \). Thus, we have that \( f_{01}(y) \in S^{DSa}(\Gamma, \delta) \). For the case where \( \Gamma < 2 \), by definition of \( S^{DSb}(\Gamma, \delta) \), we have:

\[
\begin{align*}
    d_0(F_{01}(y), F_{00}(y)) &= \max_{i} \max \left\{ w_i - \frac{1}{n_0}, 0 \right\} \\
    &\leq \frac{\Gamma - 1}{n_0},
\end{align*}
\]

which similarly implies \( \max_i w_i \leq \Gamma/n_0 \). Therefore, we have shown \( f_{01}(y) \in S^{DSb}(\Gamma, \delta) \Rightarrow f_{01}(y) \in S^{DSa}(\Gamma, \delta) \), which, together with the above result, implies that \( S^{DSb}(\Gamma, \delta) = S^{DSa}(\Gamma, \delta) \).

We now prove that \( d_0 \) is a metric on the space of piecewise constant CDFs when \( \Gamma \geq 2 \) and a quasimetric when \( \Gamma < 2 \). A metric \( d \) must satisfy three properties:

1. \( d(x, y) = 0 \iff x = y \)
2. \( d(x, y) = d(y, x) \)
3. \( d(x, y) \leq d(x, z) + d(z, y) \)

A quasimetric \( d' \) needs to only satisfy properties 1 and 3.
We first consider the $\Gamma \geq 2$ case. By symmetry of the absolute value function, $d_0$ satisfies property 2. For property 1, if $F = G$, it is clear that $d(F, G) = 0$. If $d(F, G) = 0$, then it implies:

$$\max_y \left| \lim_{\epsilon \to 0^-} (F(y) - G(y)) - (F(y + \epsilon) - G(y + \epsilon)) \right| = 0$$

which leads to:

$$\lim_{\epsilon \to 0^-} (F(y) - G(y)) - (F(y + \epsilon) - G(y + \epsilon)) = 0 \quad \forall y$$  \hspace{1cm} (A5)

Note that $F, G$ are piecewise constant CDFs. Therefore, $F$ can be completely characterized by its $n_F$ jump points:

$$F(y) = \sum_{i=1}^{n_F} w_i \mathbf{1}\{y > y_i\},$$

where $\sum_{i=1}^{n_F} w_i = 1$. Then, we apply Equation (A5) at $y = y_i$ for all $i \in \{1, \ldots, n_F\}$, which yields:

$$\lim_{\epsilon \to 0^-} G(y) - G(y + \epsilon) = w_i$$

That is, $G$ also has a jump of magnitude $w_i$ at $y_i$ for all $i$. Since $\sum_{i=1}^{n_F} w_i = 1$, this means that $G$ cannot have any additional discontinuity points, and thus we have:

$$G(y) = \sum_{i=1}^{n_F} w_i \mathbf{1}\{y > y_i\} = F(y).$$

For property 3, we have:

$$d_0(F, G) + d_0(G, H) = \max_{y_1} \left| \lim_{\epsilon \to 0^-} (F(y_1) - G(y_1)) - (F(y_1 + \epsilon) - G(y_1 + \epsilon)) \right|$$

\[ + \max_{y_2} \left| \lim_{\epsilon \to 0^-} (G(y_2) - H(y_2)) - (G(y_2 + \epsilon) - H(y_2 + \epsilon)) \right| \]

\[ \geq \max_{y_1} \left| \lim_{\epsilon \to 0^-} (F(y_1) - G(y_1)) - (F(y_1 + \epsilon) - G(y_1 + \epsilon)) \right| \]

\[ + \left| \lim_{\epsilon \to 0^-} (G(y_1) - H(y_1)) - (G(y_1 + \epsilon) - H(y_1 + \epsilon)) \right| \]

\[ \geq \max_{y_1} \left| \lim_{\epsilon \to 0^-} (F(y_1) - G(y_1)) - (F(y_1 + \epsilon) - G(y_1 + \epsilon)) \right| \]

\[ + \left| \lim_{\epsilon \to 0^-} (G(y_1) - H(y_1)) - (G(y_1 + \epsilon) - H(y_1 + \epsilon)) \right| \]

\[ = \max_{y_1} \left| \lim_{\epsilon \to 0^-} (F(y_1) - H(y_1)) - (F(y_1 + \epsilon) - H(y_1 + \epsilon)) \right| \]

\[ = d_0(F, H), \]

as required. Therefore $d_0$ is a metric for $\Gamma \geq 2$. 

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We next prove the case where $\Gamma < 2$. Again, it is clear that if $F = G$, then we have $d_0(F, G) = 0$. If $d_0(F, G) = 0$, then we have:

$$\max_y \max_{\epsilon \to 0^-} \left\{ \lim_{\epsilon \to 0^-} (F(y) - G(y)) - (F(y + \epsilon) - G(y + \epsilon)), 0 \right\} = 0$$

This implies that:

$$\lim_{\epsilon \to 0^-} (F(y) - G(y)) - (F(y + \epsilon) - G(y + \epsilon)) \leq 0 \quad \forall y \tag{A6}$$

$$\lim_{\epsilon \to 0^-} (F(y) - G(y)) - (F(y + \epsilon) - G(y + \epsilon)) \leq 0 \quad \forall y$$

Let us consider the characterization of $F$ by its $n_F$ jump points:

$$F(y) = \sum_{i=1}^{n_F} w_i 1\{y > y_i\},$$

If we apply Equation (A6) to the $y_i$ for all $i \in \{1, \cdots, n_F\}$, and then sum up the equations, we have:

$$\lim_{\epsilon \to 0^-} \sum_{i=1}^{n_F} G(y_i) - G(y_i + \epsilon) \geq 1$$

However, since $G$ is a CDF, we have:

$$\lim_{\epsilon \to 0^-} \sum_{i=1}^{n_F} G(y_i) - G(y_i + \epsilon) \leq G(\max_i y_i) - G(\min_i y_i) \leq 1$$

Therefore, we have:

$$\lim_{\epsilon \to 0^-} \sum_{i=1}^{n_F} G(y_i) - G(y_i + \epsilon) = 1$$

Now assume that we have $\lim_{\epsilon \to 0^-} G(y_i) - G(y_i + \epsilon) < w_i$ for some $i$. Then, by Equation (A6) at $y = y_i$, we have:

$$\lim_{\epsilon \to 0^-} (F(y_i) - G(y_i)) - (F(y_i + \epsilon) - G(y_i + \epsilon)) = w_i - (G(y_i) - G(y_i + \epsilon)) > 0,$$

which leads to a contradiction. Therefore, we have that $\lim_{\epsilon \to 0^-} G(y_i) - G(y_i + \epsilon) \geq w_i$ for all $i$. But then since $\sum_{i=1}^{n_F} w_i = 1$, $G$ thus cannot have any additional jump points and therefore:

$$G(y) = \sum_{i=1}^{n_F} w_i 1\{y > y_i\} = F(y),$$

as required.
For property 3, we have:

\[
d_0(F, G) + d_0(G, H) = \max_{y_1} \max \left\{ \lim_{\epsilon \to 0^-} (F(y_1) - G(y_1)) - (F(y_1 + \epsilon) - G(y_1 + \epsilon)), 0 \right\} \\
+ \max_{y_2} \max \left\{ \lim_{\epsilon \to 0^-} (G(y_2) - H(y_2)) - (G(y_2 + \epsilon) - H(y_2 + \epsilon)), 0 \right\} \\
\geq \max_{y_1} \max \left\{ \lim_{\epsilon \to 0^-} (F(y_1) - G(y_1)) - (F(y_1 + \epsilon) - G(y_1 + \epsilon)), 0 \right\} \\
+ \max \left\{ \lim_{\epsilon \to 0^-} (G(y_1) - H(y_1)) - (G(y_1 + \epsilon) - H(y_1 + \epsilon)), 0 \right\} \\
\geq \max_{y_1} \max \left\{ \lim_{\epsilon \to 0^-} (F(y_1) - H(y_1)) - (F(y_1 + \epsilon) - H(y_1 + \epsilon)), 0 \right\} \\
= d_0(F, H).
\]

Thus, \(d_0\) is a quasimetric when \(\Gamma < 2\).
A.4 Additional Synthetic Experiments on Varying $\delta$

| Scenario 1          | $n = 100$ |          | $n = 200$ |          | $n = 500$ |          |
|---------------------|-----------|----------|-----------|----------|-----------|----------|
|                     | bias      | s.d.     | bias      | s.d.     | bias      | s.d.     |
| **Distributional Sensitivity** |           |          |           |          |           |          |
| $\Gamma = 2$        | -1.810    | 0.498    | -1.888    | 0.335    | -1.913    | 0.213    |
| $\Gamma = 3$        | -2.245    | 0.543    | -2.385    | 0.384    | -2.455    | 0.234    |
| $\Gamma = 5$        | -2.729    | 0.649    | -2.900    | 0.462    | -2.900    | 0.275    |
| **Marginal Sensitivity** |           |          |           |          |           |          |
| $\Gamma = 2$        | -1.938    | 0.465    | -1.954    | 0.316    | -1.944    | 0.207    |
| $\Gamma = 3$        | -2.506    | 0.470    | -2.506    | 0.348    | -2.550    | 0.218    |
| $\Gamma = 5$        | -3.164    | 0.529    | -3.175    | 0.391    | -3.192    | 0.241    |
| **Scenario 2**      |           |          |           |          |           |          |
| **Distributional Sensitivity** |           |          |           |          |           |          |
| $\Gamma = 2$        | -1.190    | 0.489    | -1.263    | 0.336    | -1.302    | 0.213    |
| $\Gamma = 3$        | -1.653    | 0.543    | -1.797    | 0.369    | -1.882    | 0.234    |
| $\Gamma = 5$        | -2.094    | 0.661    | -2.296    | 0.465    | -2.412    | 0.274    |
| **Marginal Sensitivity** |           |          |           |          |           |          |
| $\Gamma = 2$        | -1.321    | 0.455    | -1.330    | 0.321    | -1.333    | 0.208    |
| $\Gamma = 3$        | -1.916    | 0.474    | -1.953    | 0.339    | -1.973    | 0.216    |
| $\Gamma = 5$        | -2.518    | 0.546    | -2.577    | 0.394    | -2.594    | 0.233    |
| **Scenario 3**      |           |          |           |          |           |          |
| **Distributional Sensitivity** |           |          |           |          |           |          |
| $\Gamma = 2$        | -1.667    | 0.553    | -1.788    | 0.370    | -1.831    | 0.226    |
| $\Gamma = 3$        | -2.113    | 0.632    | -2.260    | 0.441    | -2.369    | 0.263    |
| $\Gamma = 5$        | -2.457    | 0.752    | -2.745    | 0.519    | -2.882    | 0.326    |
| **Marginal Sensitivity** |           |          |           |          |           |          |
| $\Gamma = 2$        | -1.847    | 0.513    | -1.870    | 0.345    | -1.865    | 0.218    |
| $\Gamma = 3$        | -2.466    | 0.549    | -2.455    | 0.392    | -2.475    | 0.244    |
| $\Gamma = 5$        | -3.047    | 0.598    | -3.090    | 0.435    | -3.092    | 0.271    |

Table A1: A Simulation Study of Distributional Sensitivity (with $\delta = 0.15$) and Marginal Sensitivity Models. The table presents the estimated bias and standard deviation of the ATT estimators for both models under difference scenarios, robustness ($\Gamma$) and number of samples $n$. 

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