Decision makers often want to identify the individuals for whom some intervention or treatment will be most effective in order to decide who to treat. In such cases, decision makers would ideally like to rank potential recipients of the treatment according to their individual causal effects. However, the historical data available to estimate the causal effects could be confounded, and as a result, accurately estimating the effects could be impossible. We propose a new and less restrictive assumption about historical data—called the ranking preservation assumption (RPA)—under which the ranking of the individual effects can be consistently estimated even if the effects themselves cannot be accurately estimated. Importantly, we find that confounding can be helpful for the estimation of the causal-effect ranking when the confounding bias is larger for individuals with larger causal effects, and that even when this is not the case, any detrimental impact of confounding can be corrected with larger training data when the RPA is met. We then analytically show that the RPA can be met in a variety of scenarios, including common business applications such as online advertising and customer retention. We support this finding with an empirical example in the context of online advertising. The example also shows how to evaluate the decision making of a confounded model in practice. The main takeaway is that what might traditionally be considered “good” data for causal estimation (i.e., unconfounded data) may not be necessary to make good causal decisions, so treatment assignment methods may work better than we give them credit for in the presence of confounding.

Key words: causal decision making, causal effect estimation, confounding, learning to rank

1. Introduction

Decision makers often want to identify the individuals for whom some intervention or treatment will be most effective, particularly when facing budget constraints (Bhattacharya and Dupas 2012). Typical examples include business applications such as targeted advertising (Miller and Hosanagar 2020) or customer retention (Ascarza 2018), where firms are interested in giving a limited number of special offers (treatments) to those customers for whom the offers can increase profits the most. So, the goal is to maximize the causal impact of the limited number of treatments that are offered.
An increasingly popular approach to address this type of decision-making tasks is to use machine learning (ML) on data from past treatment assignments to estimate a causal-effect (or uplift) model (Gutiérrez and Gérardy 2017). Using ML to estimate an uplift model allows the decision maker to estimate how the treatment effect varies from one individual to another based on their attributes (e.g., preferences, behaviors, history with the firm). So, the model can be used to predict the effect on each individual who might receive the treatment in order to rank them according to the size of their effect predictions. Then, those with the largest effect predictions are treated.

However, one major concern is the possibility of confounding in the training data. Confounding occurs when the individuals who received the treatment (the treated) and the individuals who did not receive it (the untreated) are systematically different. For example, if an ad (the treatment) was targeted primarily at people who were interested in the advertised product, then the treated would have been more likely to make a purchase even without the ad, and the predictions could suffer from an upward bias. We could falsely attribute to the ad a positive “causal effect” that should be attributed to another systematic difference between treated and untreated.

For this reason, the literature in policy learning has mostly focused on settings where controlled experiments are used to collect unconfounded data. However, conducting controlled experiments is not always feasible and can be prohibitively expensive even in applications like targeted advertising (Gordon et al. 2022), where experimentation is relatively common. There are a few studies that discuss policy learning with observational data (Athey and Wager 2021), but such studies typically assume that the confounding can somehow be corrected. The conventional perspective is that correcting for confounding is desirable (if not necessary) for causal inference.

Our study builds on a nascent stream of research that distinguishes between causal decision making and causal effect estimation (Fernández-Loría and Provost 2022b). One key distinction is how confounding affects these two tasks. For example, Yang et al. (2020) point out that confounding bias in the effect estimates does not result in a loss in decision-making value unless the bias changes the sign of the effect estimates. In fact, confounding bias can even improve decision making when larger causal effects are more likely to be overestimated (Fernández-Loría and Provost 2019).

What remains unclear is what kind of confounded data can be useful for decision making. Many studies in the causal inference literature have proposed non-testable assumptions that, when met in the confounded data, imply that the causal effect estimation improves with larger data. Well-known examples include ignorability (Rosenbaum and Rubin 1983) and the back-door criterion (Pearl 2009). Causal decision making can improve with larger data when these assumptions are violated (Fernández-Loría and Provost 2019), but it is unclear what conditions the confounded data must meet for this to occur. In response to previous calls for research (Eckles 2022, McFowland III 2022, Fernández-Loría and Provost 2022b), we address this gap in the literature by characterizing
settings where good causal decisions can be made even though accurate effect estimation is not possible because of violations of conventional causal assumptions.

As our main contribution, we formalize a new and less restrictive assumption—that we call the **ranking preservation assumption (RPA)**—under which it is possible to use confounded data to identify the individuals for whom a specific treatment is most effective even though the effects themselves cannot be accurately estimated (Section 2). When the RPA is met, any detrimental impact of confounding on the estimation of the causal-effect ranking can be corrected with larger training data. In fact, confounding can be helpful for the estimation of the causal-effect ranking when the confounding bias is larger for individuals with larger causal effects. We provide an empirical illustration of this result in the context of an online advertising application (Section 4).

A question that naturally arises is “When should we expect the confounded estimates to preserve the causal-effect ranking?” As a second contribution, we provide a two-part answer. First, we look at the type of settings where we should expect the confounded data to be useful to learn a model that can rank individuals according to their treatment effects. We theoretically derive sufficient conditions in which the RPA holds, and find that some of these conditions closely match common business applications such as advertising and customer retention (Section 3). The implication is that there is a variety of scenarios where treatment assignment methods could work perfectly fine despite the use of flawed training data, even if such data suffers from unobserved confounding that cannot possibly be corrected.

Then, we show how to determine in practice whether a confounded model is useful for ranking individuals according to their treatment effects. We focus on settings where practitioners can get their hands on at least some unconfounded data and show how the practitioners could then use that data to evaluate the confounded model (Section 4).

Overall, this study develops the perspective that what might traditionally be considered “good” data for causal estimation (i.e., unconfounded data) may not be necessary to make good causal decisions. This perspective is quite important in practice because acquiring data to estimate causal effects accurately is often complicated and expensive.

### 2. Ranking by Causal Effects

This study considers settings where a decision maker wants to treat the individuals on whom some treatment will have the largest expected causal effect. Formally, let \( C \in \mathbb{R} \) be the causal effect, and let \( X \) be a vector of \( p \) individual-level attributes (features) that the decision maker observes, where \( X \in \mathbb{R}^p \). Then, the **conditional average treatment effect (CATE)** is:

\[
\beta(x) = \mathbb{E}[C|X = x]. \tag{1}
\]
We also refer to Equation 1 as the causal model. Additionally, let $\beta(i)$ be the $i$-th largest CATE among the individuals that the decision maker could potentially treat:

$$\beta(i) = \beta(x_i),$$

where $x_i$ are the features for the individual with the $i$-th largest CATE.

This study considers settings where the decision maker’s goal is to treat the $k$ individuals with the largest CATEs. Therefore, the following is an optimal treatment assignment policy:

$$a^*(x) = \mathbf{1}\{\beta(x) \geq \beta(k)\}, \forall x \in \mathbb{R}^p,$$

where $a^*(x) = 1$ when an individual should be treated, and $a^*(x) = 0$ otherwise.

The challenge in our setting is that we cannot directly estimate the causal model $\beta$ because causal effects are not observable. Instead, causal effects must be estimated based on the outcomes of treated and untreated individuals. Formally, let $Y$ be the baseline outcome (the outcome if individuals are not treated), let $Y + C$ be the treatment outcome (the outcome if individuals are treated), and let $T$ be the treatment assignment ($T = 1$ for treated individuals, and $T = 0$ for untreated individuals). Equation 4 defines an alternative model that can be estimated, which we call the feasible model or the conditional average difference in outcomes (CADO):

$$\tilde{\beta}(x) = \mathbb{E}[Y + C|T = 1, X = x] - \mathbb{E}[Y|T = 0, X = x],$$

$$= \mathbb{E}[C|T = 1, X = x] + (\mathbb{E}[Y|T = 1, X = x] - \mathbb{E}[Y|T = 0, X = x]).$$

The CATE (Equation 1) and the CADO (Equation 4) are the same when the causal effect and the baseline outcome are conditionally independent of the treatment assignment given the features:

$$T \perp\!
\perp C, Y | X.$$

This (non-testable) assumption is known in the causal inference world as unconfoundedness, ignorability (Rosenbaum and Rubin 1983), the back-door criterion (Pearl 2009), and exogeneity (Wooldridge 2015). This assumption is met when the treatment assignment does not provide any information about the baseline outcome or the causal effect after conditioning on the features. If this is not the case, then it is not possible to unbiasedly estimate the causal model (Equation 1) using the feasible model (Equation 4).

---

1 If the decision maker wants to treat the individuals who have a CATE larger than some threshold $\delta$ (e.g., when treating the individuals for whom the benefit of doing so is larger than some cost), then $k$ corresponds to the index that satisfies $\beta(x_k) > \delta \geq \beta(x_{k+1}).$

2 The baseline outcome and the treatment outcome are equivalent to potential outcomes (Rubin 1974). Formally, let $Z(0)$ and $Z(1)$ be the potential outcomes when untreated and treated, respectively. Then, $Z(0) = Y,$ and $Z(1) = Y + C.$ We define causal effects as $C$ rather than $Z(1) - Z(0)$ because doing so is convenient for the analysis in Section 3.
However, accurately estimating the causal model is not the goal here. Instead, we are interested in assessing whether the feasible model is good for decision making. Suppose that the feasible model is used to treat the $k$ individuals with the largest CADOs. Then, we say that the feasible model is a regret minimizer when the treatment assignments made with the feasible model are the same as if the causal model had been used:

$$\tilde{a}(x) = 1\{\tilde{\beta}(x) \geq \tilde{\beta}(k)\} = a^*(x), \forall x \in \mathbb{R}^p,$$

where $\tilde{\beta}(k)$ is the $k$-th largest CADO.

**Theorem 1.** The feasible model is a regret minimizer for a fixed $k$ if and only if:

$$\tilde{\beta}(x_i) \geq \tilde{\beta}(k), \forall i \leq k; \quad (7)$$

$$\tilde{\beta}(x_i) < \tilde{\beta}(k), \forall i > k. \quad (8)$$

The proof is in Appendix A. Theorem 1 implies that accurate estimations of causal effects are not necessary to make optimal decisions. If the $k$ individuals with the largest CADOs ($\tilde{\beta}$) are also the $k$ individuals with the largest CATEs ($\beta$), then the feasible model is a regret minimizer.

Figure 1 illustrates this. The blue solid line is the CADO as a function of the CATE. The vertical dashed line is the $k$-th largest CATE, and the horizontal dashed line is the $k$-th largest CADO. The feasible model correctly identifies the $k$ individuals with the largest effects whenever the blue line appears only on the upper-right quadrant (Equation 7) and the lower-left quadrant (Equation 8). In this example, the feasible model is not good for causal effect estimation due to confounding, but it correctly identifies the $k$ individuals with the largest causal effects.

Nevertheless, one concern in this example is that the feasible model may or may not be a regret minimizer depending on the number of individuals that the decision maker wants to treat. For instance, if the decision maker changes $k$ so that $\beta(k) = 3$ or $\beta(k) = 5$, then the feasible model does not correctly identify the top $k$ individuals. More generally, the feasible model is a regret minimizer for any $k$ if and only if it can rank all individuals according to their causal effects.

**Theorem 2.** The feasible model is a regret minimizer for any $k$ if and only if:

$$\tilde{\beta}(x_i) > \tilde{\beta}(x_j), \forall i, j : \beta(x_i) > \beta(x_j). \quad (9)$$

The proof is in Appendix B. We call Equation 9 the ranking preservation assumption or RPA, and it is met when larger CADOs also imply larger CATEs. Figure 2 illustrates an example in which the RPA is met. As opposed to Figure 1, Figure 2 shows that the feasible model is a regret minimizer regardless of $k$. The $k$ individuals with the largest CADOs always correspond to the $k$ individuals with the largest CATEs.
Figure 1   Comparison of the causal model and the feasible model. The feasible model does not approximate the causal model accurately, but it can identify the $k$ individuals with the largest effects (upper-right quadrant).

Figure 2   The feasible model preserves the ranking of the causal model. For any $k$, the feasible model can identify the $k$ individuals with the largest effects (upper-right quadrant).

2.1. Estimation of the Ranking
We analyze next the implications of confounding bias for the estimation of the causal-effect ranking. With this purpose in mind, we re-define the CADO in Equation 4 as:

$$\tilde{\beta}(x) = \beta(x) + \alpha(x),$$  \hspace{1cm} (10)

where $\alpha(x)$ is the bias due to confounding in the data. In practice, CADOs are not used to make decisions because effects are estimated from data. We define the CATE estimates as:

$$\hat{\beta}(x) = \tilde{\beta}(x) + \xi,$$  \hspace{1cm} (11)
where $\xi$ is a random variable that represents the estimation error produced by an ML algorithm.\footnote{Note that $\xi$ corresponds to error in the estimation of $\hat{\beta}$. It does not correspond to the “idiosyncratic error” that is typically used in econometrics to describe the impact of unobserved factors on the dependent variable.}

Equation (11) can also be interpreted as the \textbf{estimated model}. $E[\xi]$ corresponds to bias in the ML algorithm, whereas deviations in $\xi$ represent variance in the ML algorithm due to sampling error.

We are interested in recovering the ranking of causal effects ($\beta$) using causal effect estimates ($\hat{\beta}$). In our analysis, we use the Kendall rank correlation coefficient (Kendall 1938) as a measure of the estimated model’s ability to recover the ranking. Assuming there are $n$ potential candidates for the treatment, the \textbf{rank correlation} ($\tau$) between the CATEs and the CATE estimates is:

$$
\tau = \frac{2}{n(n-1)} \sum_{i<j} \text{sgn}(\beta(x_i) - \beta(x_j))\text{sgn}(\hat{\beta}(x_i) - \hat{\beta}(x_j))
= \frac{2}{n(n-1)} \sum_{i<j} \text{sgn}(\hat{\beta}(x_i) - \hat{\beta}(x_j)).
$$

(12)

Note that $-1 \leq \tau \leq 1$. The estimated model preserves the ranking only when $\tau = 1$. However, in most practical settings, the estimates will not perfectly preserve the ranking because of confounding, bias in the modeling procedure, or sampling error. Therefore, we use the expected rank correlation next to assess how confounding can affect the estimation of the causal-effect ranking.

\textbf{Theorem 3.} The expected rank correlation between the CATEs and the CATE estimates is:

$$
E[\tau] = \left( \frac{4}{n(n-1)} \sum_{i<j} P[\beta_\Delta + \alpha_\Delta + \xi_\Delta > 0] \right) - 1,
$$

(13)

where:

$$
\beta_\Delta = \beta(x_i) - \beta(x_j) > 0,
\alpha_\Delta = \alpha(x_i) - \alpha(x_j),
\xi_\Delta = (\xi|X = x_i) - (\xi|X = x_j).
$$

The proof is in Appendix C. Theorem 3 shows that the expected rank correlation improves when $\alpha_\Delta > 0$ and decreases when $\alpha_\Delta < 0$. In other words, the quality of the ranking improves when the rate of change in the bias ($\alpha_\Delta$) is positive, and the larger the rate of change the better. As a result, confounding helps to estimate the ranking when the confounding bias is larger for individuals with larger effects (i.e., when $\alpha_\Delta$ is always positive). We show an empirical example in Section 4.

We can obtain further insights about the implications of confounding for the estimation of the ranking by making additional assumptions about the ML algorithm.
**Theorem 4.** Suppose that the estimated model \((\hat{\beta})\) is learned with an ML algorithm that is a consistent estimator of \(\hat{\beta}\). Then, if the RPA is met, \(\tau\) converges in probability to 1 as the size of the training data increases.

The proof is in Appendix D. Theorem 4 implies that, when the RPA is met, any detrimental impact of confounding on the estimation of the ranking can be corrected with larger training data if the ML algorithm is powerful enough to learn the CADOs. In other words, the causal decision making improves with larger training data if the RPA is met.

These results are well-aligned with Fernández-Loría and Provost (2019), who showed that using large-but-confounded data to estimate models can lead to decisions that are better than when using unconfounded-but-smaller data. We contribute to their results by showing that the same observations apply when the models are used to rank individuals according to their causal effects.

Specifically, Theorem 4 implies an interesting trade-off between large-but-confounded data and unconfounded-but-smaller data. On the one hand, confounding lowers performance (as defined in Equation 13) when \(\alpha_\Delta < 0\). On the other hand, if the RPA is met, Theorem 4 implies that any detrimental impact of confounding on performance becomes irrelevant with large enough training data. Additionally, if the ML algorithm is a consistent estimator, estimation errors decrease with larger training data. Therefore, a model trained with large-but-confounded data could perform better than a model trained with unconfounded-but-smaller data, because errors due to confounding could be offset by having less estimation errors.

### 3. Settings Where the Causal-Effect Ranking Is Preserved

We introduce next a theoretical analysis to help us gain some intuition on the type of settings where we could expect the RPA to hold. The purpose of this analysis is to show that there is a variety of scenarios where the RPA could be met and that some of these scenarios closely match common applications in which ranking individuals according to their causal effects is desirable.

#### 3.1. Data Generating Process

Whether the RPA is met or not will ultimately depend on the data generating process of \(T\), \(C\), and \(Y\). As part of our theoretical analysis, we make two assumptions about the data generating process. The first is that past treatment assignments are determined by an unobserved utility \(U\):

\[
T = 1\{U > 0\}. \tag{14}
\]

The second is that \(Y\), \(C\), and \(U\) are normally distributed when conditioned on \(X\):

\[
\begin{pmatrix}
Y \\
C \\
U
\end{pmatrix} \mid X \sim \mathcal{N}
\begin{pmatrix}
\mu(X) \\
\beta(X) \\
\nu(X)
\end{pmatrix},
\begin{pmatrix}
\sigma_Y^2 & \rho_{YC}\sigma_Y\sigma_C & \rho_{YU}\sigma_Y\sigma_U \\
\rho_{YC}\sigma_Y\sigma_C & \sigma_C^2 & \rho_{CU}\sigma_C\sigma_U \\
\rho_{YU}\sigma_Y\sigma_U & \rho_{CU}\sigma_C\sigma_U & \sigma_U^2
\end{pmatrix},
\tag{15}
\]
From an econometrics perspective, Equation 15 is the distribution of the error terms (i.e., the deviations) in $Y$, $C$, and $U$ given $X$. So, it describes how $Y$, $C$, and $U$ relate to each other after all the information in $X$ has been accounted for.

Thus, given some individual with feature values $x$, $\mu(x)$ is the individual’s expected outcome if not treated, $\beta(x)$ is the expected effect on the individual (the CATE), and $\nu(x)$ is the expected utility associated with treating that individual. Additionally, $\sigma_a^2$ is the degree of heterogeneity in variable $a$ that is not captured by $X$. For example, $\sigma_C^2$ represents the degree to which $C$ varies across individuals that have the same feature values, so it represents unobserved heterogeneity in causal effects. Finally, $\rho_{ab}$ is the correlation between variables $a$ and $b$ when conditioned on $X$. For instance, $\rho_{CU}$ is the correlation between $C$ and $U$ given $X$. So, if the unconfoundedness assumption holds (Equation 5), then $\rho_{YU} = \rho_{CU} = 0$.

As with unconfoundedness, Equation 15 is a non-testable characterization of the data generating process. We assume normality to facilitate the analytical derivations and gain some intuition on how confounding can affect decision making. Similar assumptions are also prevalent in many popular statistical models. For example, probit regression is often used to model variables with binary outcomes such as $T$, which is analogous to assuming that $U$ follows a normal distribution when conditioned on $X$. Similarly, some important properties of linear regression models only hold when the conditional distribution of the dependent variable is normal, as in Equation 15.

We suspect that the following result could potentially be generalized to a broader class of distributions, perhaps in a similar fashion to how theoretical results for matching methods have been generalized to spherically symmetrical distributions (Rubin and Thomas 1992; Rubin and Stuart 2006). However, we leave this for future research.

### 3.2. Sufficient Conditions That Preserve the Ranking

We discuss next sufficient (but not necessary) conditions for the RPA to be met.

**Theorem 5.** Given Equations 14 and 15, and assuming $\tilde{\beta}$ is always differentiable with respect to $\beta$, the ranking preservation assumption is met if:

$$U \perp\!\!\!\!\perp X|C,$$

and

$$\rho_{CU} \rho_{YU} (\mathbb{P}[T = 1|X = x] - 0.5) \geq 0, \forall x \in \mathbb{R}^p.$$  \hspace{1cm} (17)$$

The proof is in Appendix E.

Equation 16 means that the unobserved utility of assigning past treatments ($U$) is conditionally independent of the features ($X$) given the causal effect ($C$). Intuitively, this condition implies that past treatment assignments are primarily driven by causal effects (e.g., individuals with larger
Table 1  Scenarios under which the causal-effect ranking is preserved despite confounding.

| Scenario | Past treatment assignments have... |
|----------|----------------------------------|
| 1        | a positive...                    |
| 2        | a positive...                    |
| 3        | a negative...                    |
| 4        | a negative...                    |
|          | correlation with causal effects. |
|          | correlation with baseline outcomes. |
|          | 50% probability of occurring. |

Effects are generally more likely to be treated (rather than other individual-level characteristics). While this condition may look similar to the unconfoundedness assumption (Equation 5), they are quite different. On the one hand, Equation 16 implies that individuals with the same causal effect are equally likely to be treated regardless of other observed attributes. On the other hand, Equation 5 implies that individuals with the same observed attributes are equally likely to be treated regardless of their baseline outcomes and causal effects. The latter is a stronger assumption because it requires that $\rho_{YU} = \rho_{CU} = 0$.

Equation 17 shows that there are three quantities that determine whether the RPA is met. We have: $\rho_{CU}$, the unobserved correlation between treatment assignments and causal effects; $\rho_{YU}$, the unobserved correlation between treatment assignments and baseline outcomes; and $(P[T = 1|X = x] - 0.5)$, which is positive when the probability of treatment is greater than 50%.

These quantities describe conditions, summarized in Table 1, that are sufficient (but not necessary) for the RPA to be met. For example, according to Scenario 1 in Table 1, the RPA is met when the feasible model is learned using data in which:

1. treatment assignments have a positive correlation with causal effects,
2. treatment assignments have a positive correlation with baseline outcomes, and
3. treatment assignments have more than a 50% probability of occurring.

Targeted advertising is an application where this list of conditions could potentially be met. Condition 2 is likely to be met because advertisements are often targeted based on the probability of purchase, and Condition 1 is met when such a targeting strategy is an effective approach to increase purchases. For example, in an advertising campaign of a major food chain, Stitelman et al. (2011) found that displaying ads based on the estimated likelihood of conversion increased the volume of prospective new customers by 40% more than displaying ads to the general population. Fernández-Loría and Provost (2022a) also give modeling justifications for why targeting ads based on baseline outcomes can be a reasonable approach to maximize the causal impact of ads. Condition 3 is likely to be met if the opportunity cost associated with not advertising to good prospects is much larger than the treatment cost associated with advertising to bad prospects.

Note that Condition 3 may still not be met when the pool of potentially good prospects is too low, as that would discourage advertisers from targeting too many individuals.
As an additional example, Scenario 2 in Table 1 implies that the RPA is met when:
1. treatment assignments have a positive correlation with causal effects,
2. treatment assignments have a negative correlation with baseline outcomes, and
3. treatment assignments have less than a 50% probability of occurring.

Customer retention is an application where this list of conditions could be met. In a similar fashion to advertising, Condition 2 is likely to be met because retention offers are often targeted based on the probability of leaving, and Condition 1 is met when such a targeting strategy is an effective approach to increase retention. For example, Huang et al. (2015) deployed a churn prediction model in a telecommunications firm that increased the recharge rate of potential churners by more than 50% according to an A/B test. Condition 3 is likely to be met if the retention offer being sent is expensive (e.g., a discount conditional on the customer renewing the subscription), and as a result, the company is reluctant to target too many individuals.

4. Empirical Example
This section has two purposes. First, it gives an empirical example of how confounding can improve the ability to rank individuals according to their causal effects even when it leads to highly inaccurate causal effect estimates. Second, it shows how to determine in practice whether a confounded model is useful to rank individuals according to their causal effects.

4.1. Application Setting
The data in our example was assembled by Criteo (an advertising platform) after running a controlled experiment in which advertising was randomly targeted to a large sample of online consumers. This data set was released to benchmark methods for uplift modeling (Diemert Eustache, Betlei Artem et al. 2018). We use visits as the outcome of interest. The data consist of 13,979,592 rows, each one representing a user with 11 features, a treatment indicator for the ad, and the label (i.e., whether the user made a visit or not). The treatment rate is 85%, the average visit rate for the untreated (the average baseline outcome) is 3.82%, and the average treatment effect (the ATE) is 1.03%. In our subsequent analyses, we use 70% of the data for training and 30% for evaluation.

We use gradient boosting based on decision trees to estimate causal effects by regressing the transformed outcome proposed by Athey and Imbens (2016). However, note that our theoretical results are agnostic to the ML algorithm that is used for estimating effects.

Note that other researchers have found that targeting based on churn propensity can be ineffective (Ascarza 2018).

See https://ailab.criteo.com/criteo-uplift-prediction-dataset/ for details and access to the data. We use the version of the data set that does not have leakage.

See https://lightgbm.readthedocs.io/en/latest/ for technical details.
4.2. Confounded Data

To create confounded data, we randomly drop 80% of the treated users who did not make a visit. This results in a confounded data set in which the visit rate for the treated is higher than for the untreated (partly) because of selection bias. The intent is to simulate data that results from targeting past ads to individuals who are more likely to visit, a common practice in advertising.

To illustrate how the causal effect estimation is affected by confounding, Figure 3 shows a scatter plot comparing the effect estimates of a model trained with confounded training data (\(\hat{\beta}_C\)) and a model trained with unconfounded training data (\(\hat{\beta}_U\)). The y-axis corresponds to the difference between the confounded and the unconfounded estimates, and the x-axis corresponds to the size of the unconfounded estimates. Dots above the dashed black line are cases where the confounded estimates are larger than the unconfounded estimates, whereas dots below the dashed line are cases where the confounded estimates are smaller. The blue solid line is a regression line that shows how the difference between the estimates changes as the size of the unconfounded estimates increases.

Figure 3 shows that most dots are above the dashed line, so the confounded estimates are generally larger than the unconfounded estimates. This is expected given the nature of the confounding. Additionally, the regression line has a positive slope, so the difference between the estimates tends to be larger for individuals with larger estimates. This suggests that the rate of change in the confounding bias is positive. In Section 2.1, we analytically showed that this can improve the estimation of the causal-effect ranking. We provide an empirical example of this below.
4.3. Evaluation

We assess next the performance of the ML algorithm when trained with confounded and unconconfounded data. We use three evaluation measures.

First, we use the Area Under the Qini Curve (AUQC) to empirically evaluate how good are the models at ranking individuals according to their CATEs. Qini curves are used in uplift modeling to depict the cumulative incremental gain in the outcome (visits in our example) that is achieved when the top percent of individuals with the largest CATE estimates are treated. Qini curves can be built using A/B test data, so practitioners that have access to experimental data can use this tool to visualize the performance of any model. A model’s AUQC is the model’s area under the curve divided by the maximum area that could possibly be under the curve. So, the AUQC summarizes the curve information in one number that is larger when the model is better at ranking individuals according to their causal effects. Figure 4 shows an example of a model’s Qini curve and its AUQC. In the figure, the model’s AUQC is 9% of the theoretically maximum AUQC.

As a second evaluation measure, we use the average causal effect for the top 10% of individuals with the largest CATE estimates—the Top 10% Uplift. Recall that the motivating task in this study is treating the top $k$ individuals with the largest effects. When we use the Top 10% Uplift as an evaluation measure, the $k$ corresponds to $n \times 10\%$, where $n$ is the number of potential candidates for the treatment. A larger Top 10% Uplift implies that the model is better at minimizing regret (i.e., finding the top 10% individuals with the largest effects).

In Section 2.1, we used the Kendall rank correlation coefficient to measure the estimated model’s ability to recover the CATE ranking. However, we cannot use this measure to evaluate performance empirically because in practice we never observe the CATEs for any given individual.
Finally, as a third evaluation measure, we use the mean absolute error (MAE) with respect to the ATE. This measure is used to assess how good are the models at causal effect estimation. A lower MAE implies that the model is better at causal effect estimation.

The evaluation is based on 100 different splits of the data into a training set and a test set. The results are reported in Figures 5, 6, and 7. The blue line is the performance of the ML algorithm with various sizes of unconfounded training data. The orange line is the performance of the ML algorithm with various sizes of confounded training data. Both lines are estimated using the same (unconfounded) test sets. The areas around the lines are 95% confidence intervals. Note that the confounded training data is generally smaller than the unconfounded training data of the “same size” because producing confounded data requires dropping 80% of the treated users who did not make a visit, as detailed in Section 4.2.

As Figure 5 shows, the confounded data leads to a larger MAE, so it is worse than the unconfounded data for estimating effects. In fact, the confounded data leads to an ATE estimate that is almost twice as large than what the ATE actually is (recall that the ATE in the entire sample is 1.03%). However, the confounded data generally leads to a larger AUQC (Figure 6) and a larger Top 10% Uplift (Figure 7), so it is better than the unconfounded data for ranking individuals according to their causal effects and finding the top 10% individuals with the largest causal effects.

5. Discussion

Although observational data often fails to produce the same estimates as controlled experiments due to confounding bias (Gordon et al. 2019), making decisions with confounded estimates can be perfectly fine (Fernández-Loria and Provost 2019, Yang et al. 2020). We propose the RPA to formally define when is fine to use confounded data to learn how to rank individuals according
Figure 6  Area Under the Qini Curve

Figure 7  Top 10% Uplift

to their causal effects. Critically, we show that when the RPA is met, any detrimental impact of confounding bias on the ranking estimation can be corrected with more training data. Furthermore, we theoretically derive conditions that are sufficient for the RPA to hold and find that those conditions closely match common business applications, such as targeted advertising and customer retention. Finally, we use data from a large-scale controlled experiment in online advertising to show an example in which confounding leads to highly inaccurate effect estimates, but the estimates are useful to rank individuals according to their effects.

These findings have important implications for practice because conducting experiments to collect unconfounded data is often (prohibitively) expensive or even impossible due to political, ethical, business, or other practical reasons. As a result, practitioners may be forced to build models with confounded data, which may be seen as an unfortunate necessity. Our results imply that treatment
assignment methods may work better than we give them credit for in the presence of confounding. In fact, the confounding could even be beneficial for decision making.

Nevertheless, as with most causal assumptions, the RPA is a condition that we normally cannot test empirically in practice. In Section 4, we demonstrated how practitioners can evaluate the decision making of confounded models by using unconfounded data as a test set. However, this approach raises two practically relevant questions.

(1) If it’s possible to collect unconfounded data to evaluate a confounded model, then why not just use the unconfounded data to train a new model? First, using confounded training data can lead to better results, as shown in our empirical results. Second, evaluating confounded models with unconfounded data will generally require a substantially smaller data investment than collecting enough training data to build a full-blown, unconfounded model. Both of these points are discussed in greater detail by Fernández-Loría and Provost (2019).

(2) What if it’s not possible to collect unconfounded data to evaluate the confounded model? For such settings, using sensitivity analysis to evaluate the decision making of the confounded model is a reasonable alternative. Fernández-Loría and Provost (2019) discuss and give an example of how sensitivity analysis techniques can be used to assess the potential impact of confounding on decision making rather than on effect estimation. Similar techniques could be adapted to assess how adversarial the confounding would need to be for the RPA to be violated.

In practice, however, choosing between confounded and unconfounded data may not be the best way to proceed. The combination of both could work better, and there are several pioneering efforts to combine observational and experimental data for causal inference (Peysakhovich and Lada 2016, Rosenman et al. 2020, Kallus et al. 2018, Fernández-Loría and Provost 2020). Similar assumptions to the RPA could be useful in determining (and possibly testing) when combining the two types of data leads to a better model for decision making than if only one type of data had been used. This presents a natural direction for future research.

We expect our findings to also be valuable in settings where data investment is commonplace, such as in multi-armed bandit (MAB) problems. The problem of treating the top $k$ individuals with the largest CATEs can be formulated as a contextual MAB problem with two arms: the treat arm (with a reward of $\beta(x) - \beta(k)$) and the do-not-treat arm (with a reward of 0). Our results imply that if the RPA is met, then the estimated model will select the correct arm in expectation. This suggests that, when the RPA is met, bootstrapping a MAB algorithm with confounded data can lead to lower regret than not bootstrapping the algorithm with any data.

Beyond these practical implications, we hope our study will help inform future methodological developments. For instance, in our empirical example, we produced confounded data by dropping 80% of the treated individuals that did not make a visit. This resulted in worse causal effect
estimates but higher performance at identifying the individuals with the largest causal effects. Introducing confounding as we did in our example can be seen as an instance of undersampling, a technique that is often used in predictive modeling to improve classification performance.

In line with this result, past studies have shown that learning to classify individuals into optimal actions can lead to better decisions than learning to accurately estimate effects (Lemmens and Gupta 2020, Fernández-Loría et al. 2022, Zhang et al. 2012). Similarly, methods designed to identify the subpopulations most affected by a treatment can perform better at this task than methods designed for causal effect estimation (McFowland III et al. 2018). So, we hope our study will help inspire new techniques specifically designed for the estimation of the causal-effect ranking.

Appendix A: Proof of Theorem 1

We start with the “if” part of the proof. We want to show that \( \tilde{a}(x_i) = a^*(x_i) \quad \forall i \). That is:

\[
1 \{ \tilde{\beta}(x_i) > \tilde{\beta}(k) \} = 1 \{ \beta(i) > \beta(k) \} \quad \forall i. 
\]

If \( i \leq k \), then Equation 7 implies that the left-hand side is equal to one. The right-hand side would then also be equal to one because \( \beta(i) \) is the \( i \)-th largest CATE. If instead \( i > k \), then Equation 8 implies that the left-hand side is equal to zero. By definition of \( \beta(i) \), the right-hand side would then also equal to zero. Therefore, the left-hand side and the right-hand side are always the same.

Now, for the “only if” part, assume that \( \tilde{a}(x_i) = a^*(x_i) \quad \forall i \). First, let \( i \leq k \). We have by definition that \( \beta(i) \geq \beta(k) \). This means that \( a^*(x_i) = \tilde{a}(x_i) = 1 \), and so \( \tilde{\beta}(x_i) \geq \tilde{\beta}(k), \forall i \leq k \). Next, consider \( i > k \). Then, \( \beta(i) < \beta(k) \), which implies that \( a^*(x_i) = \tilde{a}(x_i) = 0 \). So, it follows that \( \tilde{\beta}(x_i) < \tilde{\beta}(k), \forall i > k \), as required. Q.E.D.

Appendix B: Proof of Theorem 2

First, we want to show that \( \tilde{\beta} \) is a regret minimizer if the RPA is met. Without loss of generality, set \( k \) so that \( \tilde{\beta}(k) = \tilde{\beta}(x_j) \). We have that \( \beta(i) \geq \beta(j) \), \( \forall i \leq j \) by definition, and thus \( \tilde{\beta}(x_i) \geq \tilde{\beta}(k), \forall i \leq j \). We also have that \( \beta(x_i) < \beta(x_k), \forall i > j \), so \( \tilde{\beta}(x_i) < \tilde{\beta}(k), \forall i > k \). Therefore, by Theorem 1, \( \tilde{\beta} \) is a regret minimizer.

Now, for the “only if” part, we do a proof by contradiction. Assume that \( \tilde{\beta} \) is a regret minimizer for any \( k \), and there exists a pair of positive integers \( i, j \) for which \( \tilde{\beta}(x_i) \leq \tilde{\beta}(x_j) : \tilde{\beta}(i) > \beta(j) \). This implies that \( i < j \).

Applying Theorem 1 with \( k = i \), we have that \( \tilde{\beta}(x_j) < \beta(i) \leq \tilde{\beta}(x_i) \), which is a contradiction. So, either \( \tilde{\beta} \) is not a regret minimizer when \( k = i \) or \( \tilde{\beta}(x_i) \leq \tilde{\beta}(x_j) \) is not possible. Q.E.D.

Appendix C: Proof of Theorem 3

By definition (Equation 12):

\[
\tau = \frac{2}{n(n-1)} \sum_{i<j} \text{sgn}(\tilde{\beta}(x_i) - \hat{\beta}(x_j))
\]

Taking the expectation results in:

\[
\mathbb{E}[\tau] = \frac{2}{n(n-1)} \sum_{i<j} \mathbb{E}[\text{sgn}(\tilde{\beta}(x_i) - \hat{\beta}(x_j))],
\]

\[
= \frac{2}{n(n-1)} \sum_{i<j} \mathbb{P}[\tilde{\beta}(x_i) - \hat{\beta}(x_j) > 0] - \mathbb{P}[\tilde{\beta}(x_i) - \hat{\beta}(x_j) < 0],
\]
\[
\begin{align*}
&= \frac{2}{n(n-1)} \sum_{i<j} \left( 2P[\hat{\beta}(x_i) - \hat{\beta}(x_j) > 0] - 1 \right), \\
&= \left( \frac{4}{n(n-1)} \sum_{i<j} P[\beta_{\Delta} + \alpha_{\Delta} + \xi_{\Delta} > 0] \right) - 1,
\end{align*}
\]
where on the last line we denote: \( \beta_{\Delta} = \beta(x_i) - \beta(x_j), \alpha_{\Delta} = \alpha(x_i) - \alpha(x_j), \xi_{\Delta} = \xi(x_i) - \xi(x_j) \). Q.E.D.

**Appendix D: Proof of Theorem 4**

If the ML algorithm used to learn \( \hat{\beta} \) is a consistent estimator of \( \tilde{\beta} \), then \( \hat{\beta}(x) \) converges in probability to \( \tilde{\beta}(x) \) as the size of the training data increases. This implies that \( \hat{\beta}(x_i) - \hat{\beta}(x_j) \) converges in probability to \( \tilde{\beta}(x_i) - \tilde{\beta}(x_j) \). Therefore, according to the definition of \( \tau \) in Equation 12, \( \tau \) converges in probability to:

\[
\frac{2}{n(n-1)} \sum_{i<j} \text{sgn}(\hat{\beta}(x_i) - \hat{\beta}(x_j)).
\]

By definition of the index in \( x_i \) and \( x_j \), we have that \( \beta(x_i) > \beta(x_j) \). If the RPA is met, then \( \tilde{\beta}(x_i) > \tilde{\beta}(x_j) \), and \( \tau \) converges in probability to:

\[
\frac{2}{n(n-1)} \sum_{i<j} 1 = 1.
\]

Q.E.D.

**Appendix E: Proof of Theorem 5**

Equation 15 is:

\[
\hat{\beta}(x) = E[C|T = 1, X = x] + (E[Y|T = 1, X = x] - E[Y|T = 0, X = x]).
\]

Equation 15 implies that \( E[C | U, X = x] = \beta(x) + \alpha_C(U - \nu(x)) \), where \( \alpha_C = \rho_C \sigma_C / \sigma_U \) and \( \sigma_U = \rho_C \sigma_C / \sigma_U \). Using the property of iterated expectations and Equation 14, we also have that \( E[C | T = 1, X = x] = E[E[C | U, X = x] | T = 1, X = x] \). Replacing on the right-hand side, we have that:

\[
E[C|T = 1, X = x] = \beta(x) + \alpha_C(E[U | T = 1, X = x] - \nu(x)),
\]

and therefore:

\[
\hat{\beta}(x) = \beta(x) + \alpha_C(E[U | T = 1, X = x] - \nu(x)) + (E[Y|T = 1, X = x] - E[Y|T = 0, X = x]).
\]

Similarly, Equation 15 implies that \( E[Y | U, X = x] = \mu(x) + \alpha_Y(E[U | X = x] - \nu(x)) \), where \( \alpha_Y = \rho_Y \sigma_Y / \sigma_U \). As before, applying the property of iterated expectations, we have the following relationship for \( i = 0, 1 \):

\[
E[Y | T = i, X = x] = E[E[Y | U, X = x] | T = i, X = x],
\]

\[
= \mu(x) + \alpha_Y(E[U | T = i, X = x] - \nu(x)).
\]

This implies that:

\[
E[Y | T = 1, X = x] - E[Y | T = 0, X = x] = \alpha_Y(E[U | T = 1, X = x] - E[U | T = 0, X = x]),
\]

and therefore:

\[
\hat{\beta}(x) = \beta(x) + \alpha_C(E[U | T = 1, X = x] - \nu(x)) + \alpha_Y(E[U | T = 1, X = x] - E[U | T = 0, X = x]).
\]
Now, we must compute the expression for the derivative of $\tilde{\beta}(x)$ with respect to $\beta(x)$. The former follows from the definition of the RPA and the fact that $\partial \tilde{\beta}(x) / \partial \beta(x) > 0$. Equation 14 implies that $T = 1 \iff U > 0$ and $T = 0 \iff U \leq 0$. Therefore, we have that:

$$\tilde{\beta}(x) = \beta(x) + \alpha_c (E[U \mid U > 0, X = x] - \nu(x)) + \alpha_y (E[U \mid U > 0, X = x] - E[U \mid U \leq 0, X = x]).$$

To obtain the derivative of $\tilde{\beta}(x)$ with respect to $\beta(x)$, we need the following derivative:

$$\frac{\partial E[U \mid U > 0, X = x]}{\partial \beta(x)} = \frac{\partial \nu(x)}{\partial \beta(x)} \frac{\partial E[U \mid U > 0, X = x]}{\partial \nu(x)},$$

which is a consequence of the chain rule. From Theorem 2 of [Horrace (2015)]#, we know that the second term of the previous product is:

$$\frac{\partial E[U \mid U > 0, X = x]}{\partial \nu(x)} = \frac{\text{Var}[U \mid U > 0, X = x]}{\sigma^2_U}. $$

Similarly:

$$\frac{\partial E[U \mid U \leq 0, X = x]}{\partial \nu(x)} = -\frac{\partial E[-U \mid -U > 0, X = x]}{\partial \nu(x)} = -\frac{\partial E[-U \mid -U > 0, X = x]}{\partial \nu(x)} \frac{\partial (-\nu(x))}{\partial \nu(x)} = V \text{ar}[U \mid -U > 0, X = x] = \frac{\text{Var}[U \mid U \leq 0, X = x]}{\sigma^2_U}.$$ 

Based on the formulations above, taking the derivative of $\tilde{\beta}(x)$ with respect to $\beta(x)$ results in:

$$\frac{\partial \tilde{\beta}(x)}{\partial \beta(x)} = \frac{1 + \alpha_c \frac{\partial \nu(x)}{\partial \beta(x)} \left( \frac{\text{Var}[U \mid T = 1, X = x]}{\sigma^2_U} - 1 \right)}{\alpha_y \frac{\partial \nu(x)}{\partial \beta(x)} \left( \frac{\text{Var}[U \mid T = 1, X = x]}{\sigma^2_U} - \frac{\text{Var}[U \mid T = 0, X = x]}{\sigma^2_U} \right)}.$$ 

Assuming $U \perp X \mid C$, then:

$$\nu(x) = E_c[E[U \mid C] \mid X = x] = E_c[E_u[E[U \mid C, X] \mid X = x] = E_c[E_u[\nu(X) + \rho_{CV} \sigma_u / \sigma_c](C - \beta(X)) \mid X = x] = E_c[E_u[\nu(X)] + \rho_{CV} \sigma_u / \sigma_c(C - E_x[\beta(X)]) \mid X = x] = E[|U| + \rho_{CV} \sigma_u / \sigma_c(E[C] \mid X = x) - E[C]] = E[|U| + \rho_{CV} \sigma_u / \sigma_c](\beta(x) - E[C]).$$

This implies that $\partial \nu(x) / \partial \beta(x) = \rho_{CV} \sigma_u / \sigma_c$. So, we have that:

$$\frac{\partial \tilde{\beta}(x)}{\partial \beta(x)} = 1 + \rho_{CV}^2 \left( \frac{\text{Var}[U \mid T = 1, X = x]}{\sigma^2_U} - 1 \right) \quad \rho_{CV} \sigma_y \left( \frac{\text{Var}[U \mid T = 1, X = x]}{\sigma^2_U} - \frac{\text{Var}[U \mid T = 0, X = x]}{\sigma^2_U} \right).$$
Note that:
\[
\rho_{CU}^2 \left( \frac{\text{Var}[U|T = 1, X = x]}{\sigma_U^2} - 1 \right) > -1
\]
because \(-1 < \rho_{CU} < 1\) and \(0 < \text{Var}[U|U > 0, X = x] < \sigma_U^2\) by Proposition 3 of [Horrace (2015)]. Therefore:
\[
\frac{\partial \tilde{\beta}(x)}{\partial \beta(x)} > \rho_{UY} \rho_{CU} \frac{\sigma_Y}{\sigma_C} \left( \frac{\text{Var}[U|T = 1, X = x]}{\sigma_U^2} - \frac{\text{Var}[U|T = 0, X = x]}{\sigma_U^2} \right).
\]
The RPA is met when the expression above is positive (i.e., when larger causal effects imply larger effect estimands). That is, when:
\[
\rho_{UY} \rho_{CU} \text{Var}[U|T = 1, X = x] - \text{Var}[U|T = 0, X = x]) > 0. \tag{18}
\]
Let:
\[
\Delta = \text{Var}[U|T = 1, X = x] - \text{Var}[U|T = 0, X = x]
\]
\[
= \text{Var}[U|U > 0, X = x] - \text{Var}[U|U \leq 0, X = x].
\]
To show that Equation \[18\] is positive when \(\rho_{UY} \rho_{CU}(P[T = 1|X = x] - 0.5) > 0\), we want to show that:
\[
\text{sgn}(\Delta) = \text{sgn}(P[T = 1|X = x] - 1/2). \tag{19}
\]
By the symmetry of the normal distribution around the mean, we know that:
\[
\text{Var}[U|U > \nu(x), X = x] = \text{Var}[U|U \leq \nu(x), X = x].
\]
Therefore, Equation \[19\] is met when \(\nu(x) = 0\) because then \(\Delta = 0\) and \(P[T = 1|X = x] = 0.5\). In addition, we know that \(P[T = 1|X = x] > 0.5\) when \(\nu(x) > 0\), and \(P[T = 1|X = x] < 0.5\) when \(\nu(x) < 0\). Therefore, we can show that Equation \[19\] is also met when \(\nu(x) \neq 0\) by showing that \(\partial \Delta/\partial \nu(x) > 0\), which would imply that \(\Delta > 0\) when \(\nu(x) > 0\) and \(\Delta < 0\) when \(\nu(x) < 0\).

By Theorem 3 of [Horrace (2015)], we have that \(\partial \text{Var}[U|U > 0, X = x]/\partial \nu(x) > 0\). Theorem 3 of [Horrace (2015)] also implies that:
\[
\frac{\partial \text{Var}[U|U \leq 0, X = x]}{\partial \nu(x)} = \frac{\partial \text{Var}[-U|U > 0, X = x]}{\partial \nu(x)} = \frac{\partial \text{Var}[-U|U > 0, X = x]}{\partial (-\nu(x))} \frac{\partial (-\nu(x))}{\partial \nu(x)} < 0.
\]
Therefore, we have that:
\[
\frac{\partial \Delta}{\partial \nu(x)} = \frac{\partial \text{Var}[U|U > 0, X = x]}{\partial \nu(x)} - \frac{\partial \text{Var}[U|U \leq 0, X = x]}{\partial \nu(x)} > 0
\]
because the above is the sum of a positive term minus a negative term (i.e., a positive quantity). This implies that Equation \[19\] is met, so the RPA is met. Q.E.D.

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