Feature selection in stratification estimators of causal effects: lessons from potential outcomes, causal diagrams, and structural equations

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Abstract. What is the ideal regression (if any) for estimating average causal effects? We study this question in the setting of discrete covariates, deriving expressions for the finite-sample variance of various stratification estimators. This approach clarifies the fundamental statistical phenomena underlying many widely-cited results. Our exposition combines insights from three distinct methodological traditions for studying causal effect estimation: potential outcomes, causal diagrams, and structural models with additive errors.

Key words and phrases: Causal inference, Dimension reduction, Propensity score, Regularized regression, Semi-supervised learning, Variable selection.

1. INTRODUCTION

This paper considers the problem of estimating an average treatment effect from observational or experimental data, provided that a sufficient set of control variables are available. We pose the question: might the statistical precision of our estimates improve if we used only a subset of the available controls or possibly a dimension reduced transformation of them? This question is evergreen in the applied social sciences (see Leamer (1983) or Hernan and Robins (2022), page 195), but is surprisingly tricky to navigate for many applied researchers. In this paper, we break the problem down by considering the somewhat stylized situation of discrete covariates with finite support, where we are able to conduct a thorough variance analysis.

This paper examines this question in detail using tools from three distinct formalisms: potential outcomes, causal diagrams, and structural equations. We show (Section 2) that a key condition licensing valid causal inference from observational data can be expressed equivalently in each of the three distinct frameworks (conditional unconfoundedness, the back-door criterion, and exogenous errors), allowing us to alternate between perspectives as is convenient pedagogically. Importantly, this equivalence is established in terms of a generic function of observed covariates, meaning that it covers not only variable selection, but "feature selection"; this generality means that insights built on this equivalence apply seamlessly to modern methods such as regression trees or neural networks, which implicitly introduce potentially non-invertible transformations of the observed covariates.

For clarity, we focus on the simplified (yet fairly common in practice) setting of discrete covariates with finite support, which allows us to derive finite sample properties of common stratification estimators, including widely-used linear regression and propensity score methods. Section 3 presents two novel-but-elementary results that will be used to re-analyze earlier theoretical results pertaining to regression adjustment for causal effect estimation. The first result defines the notion of a minimal control function, allowing us to distinguish between necessary and sufficient statistical control for causal effect estimation. The second result is
a finite-sample analysis of stratification estimators of average causal effects in the setting of discrete control variables with finite support. This finite-sample analysis, presented in Theorem 2, articulates the conditions by which a control function may be viewed as optimal in the sense of minimum variance.

Section 4 collects concrete examples illustrating practical implications of the theory presented in Section 3, detailing how these results relate to previous literature, both classic and contemporary. By bringing together these profound results in the context of a common statistical framework, we hope to harmonize their insights for practitioners.

Section 5 concludes by discussing further connections to previous literature.

2. FORMAL FRAMEWORKS FOR CAUSAL INFERENCE

Let $Y$ be the outcome/response of interest, $Z$ be a binary treatment assignment, and $X$ be a vector of covariates drawn from covariate space $\mathcal{X}$, all denoted here as random variables. For a sample of size $n$, observations are assumed to be drawn independently as triples $(X_i, Y_i, Z_i)$, for $i = 1, \ldots, n$. The goal of causal effect estimation is to understand how the response variable $Y$ changes according to hypothetical manipulations of the treatment assignment variable, $Z$. For simplicity, we will refer to our observational units as “individuals”, although of course in applications that need not be the case.

The essential challenge to causal estimation is that only one of the two possible treatment assignments can be observed; as a consequence, if individuals who happen to receive the treatment differ systematically from those who do not, either in terms of their likely response value or in terms of how they respond to treatment, naive comparisons between the treated and untreated units will not simply reflect the causal impact of the treatment — the treatment effect is said to be confounded with other aspects of the population. The field of causal inference has proposed and developed a variety of techniques for coping with this difficulty, the most common of which is some form of regression adjustment (meant here to include propensity score estimators and matching estimators, etc), which entails estimating average causal effects as (weighted) averages of (estimated) conditional expectations. The key assumption that justifies this process is referred to as conditional unconfoundedness, which asserts that the measured covariates adequately account for all of the systematic differences between the treated and untreated individuals in our observational sample; formalizing this assumption can be approached in a number of ways, which we turn to now. Only after the notation of these formalisms has been introduced can our causal estimand, and the class of estimators we will study, be precisely defined.

2.1 Potential outcomes

The potential outcomes framework casts causal inference as a missing data problem: causal estimands are contrasts between pairs of outcomes that are mutually unobservable — when we see one, we cannot see the other. At present, the standard reference for the potential outcomes framework is Imbens and Rubin (2015), which contains extensive citations to the primary literature.

Let $Y^{\dagger}$ and $Y^{\circ}$ refer to the “potential outcomes” when $Z = 1$ and $Z = 0$. For individual $i$, the individual treatment effect will be defined as the difference between the potential outcomes:

$$\tau_i = Y^{\dagger}_i - Y^{\circ}_i.$$ 

Other treatment effects, such as a ratio rather than a difference, are sometimes considered, but in this paper we focus on the difference. Because the potential outcomes $(Y^{\dagger}, Y^{\circ})$ are never observed simultaneously, individual treatment effects can never be estimated directly.

However, average treatment effects can be identified (learned from data) provided certain assumptions are satisfied. The causal estimand this paper will focus on is the average treatment effect, or ATE:

$$(1) \bar{\tau} \equiv E[Y^{\dagger} - Y^{\circ}].$$

The precise population over which this expectation is taken will be discussed in more detail in section 2.5. The standard assumptions that allow this average effect to be estimated are:
1. Stable unit treatment value assumption (SUTVA), which consists of two conditions:
   a) *Consistency*: The observed data is related to the potential outcomes via the identity
      \[ Y = Y^1 Z + Y^0 (1 - Z), \]
      which describes the “gating” role of the observed treatment assignment, \( Z \).
   b) *No Interference*: for any sample of size \( n \) with \( Y \in Y \) and \( Z \in Z \), \((Y^1_i, Y^0_i) \perp \perp Z_j\) for all \( i, j \in \{1, \ldots, n\} \) with \( j \neq i \), which rules out interference between observational units.

2. Positivity: \( 0 < P(Z = 1 | X = x) < 1 \) for all \( x \in X \)

3. Conditional unconfoundedness: \((Y^1, Y^0) \perp \perp Z | X\)

Imagining concrete violations of these conditions is intuition-building. Consistency can be violated under non-compliance, so that treatment assignment doesn’t match treatment actually received. No interference can be violated, for example, if we were studying the effect of individual tutoring on student grades in a certain classroom and students study together; Jimmy’s treatment assignment may impact Sally’s grade. Positivity is violated if certain individuals can never receive treatment, rendering their contribution to the average treatment effect unlearnable. And finally, conditional unconfoundedness can be violated, for example, if both treatment assignment and the outcome variable share a common cause. However, this is not the only way conditional unconfoundedness can be violated, and exploring other possibilities in full generality is the topic of the remainder of the paper.

Taken together, the above assumptions enable identification of average treatment effects because they imply the following equality, the left-hand side of which is estimable:

\[ E_X [E[Y | X, Z = 1] - E[Y | X, Z = 0]] = E[Y^1 - Y^0]. \]

In more detail, the equivalence is established as follows:

\[
E_X [E[Y | X, Z = 1] = E_X [E[Y^1 Z + Y^0 (1 - Z) | X, Z = 1]] \\
= E_X [E[Y^1 | X, Z = 1] = E[Y^1]] .
\]

\[
E_X [E[Y | X, Z = 0] = E_X [E[Y^1 Z + Y^0 (1 - Z) | X, Z = 0]] \\
= E_X [E[Y^0 | X, Z = 0] = E[Y^0]].
\]

An alternative parametrization is:

\[ Y_i = Y^0_i + \tau_i Z_i \]

where

\[ \tau_i = Y^1_i - Y^0_i, \]

which emphasizes that \( \tau_i \) itself can differ across units and, as a random variable, can be dependent on the treatment assignment so that \( \tau \notin Z \). This treatment effect parametrization will be used extensively in our exposition.

This paper is focused on the following question: If \( X \) satisfies conditional unconfoundedness, might there be a function of \( X \) with a reduced range that also satisfies conditional unconfoundedness? That is, can \( X \) be reduced in dimension while still providing valid causal effect estimation? Answering this question requires a more detailed examination of how conditional unconfoundedness is achieved in any particular data generating process, which is facilitated by the introduction of causal diagrams.

### 2.2 Causal diagrams

#### 2.2.1 Graph theory for causal identification

Causal diagrams provide a more fine-grained look at confounding, as they consider the full joint distribution of the response, treatment, and control variables regressors. The graphical approach to causality has its earliest roots in the work of Sewell Wright (Wright, 1918, 1920, 1921), but attained its mature modern form in
the prodigious work of Judea Pearl (Pearl, 1987; Pearl and Verma, 1987, 1995; Pearl, 1995).
See Pearl (2009a) for a textbook treatment and comprehensive references. The presentation
here loosely follows the expository treatment in Shalizi (2021).

Recall that any joint density over \( p \) random variables may be expressed in compositional
form, as a product of conditional densities:

\[
f(x_1, x_2, \ldots, x_p) = f(x_1) f(x_2 | x_1) f(x_3 | x_1, x_2) \cdots f(x_p | x_1, x_2, \ldots, x_{p-1}),
\]

where the density functions \( f(\cdot) \) and \( f(\cdot | \cdot) \) refer to different densities depending on their
arguments. The labeling of the variables is arbitrary, and so we can chain together these
marginal and conditional distributions in any order (though of course that will lead to different
forms). Some of these variables might exhibit conditional independence, meaning that, for
example

\[
f(x_1 | x_2, x_3) = f(x_1 | x_2)
\]

which is equivalently expressed as

\[
X_1 \perp \perp X_3 | X_2.
\]

The relationship to directed (acyclic) graphs (DAG) is straightforward: draw a node for each
variable and draw a line from \( X_j \) going into \( X_i \) if \( X_j \) appears in the conditional distribution
of \( X_i \). This graph is directed, with the arrow pointing from \( X_j \) to \( X_i \). We say that \( X_j \) is a
“parent” of \( X_i \) and that \( X_i \) is the “child” of \( X_j \).

From the graph, the joint distribution may be expressed as

\[
f(x_1, \ldots, x_p) = \prod_{j=1}^{p} f(x_j | \text{parents}(x_j)).
\]

This leads us to the Markov property, which is

\[
X_j \perp \perp \text{non-descendants}(X_j) | \text{parents}(X_j),
\]

where “descendant” refers to children, grandchildren, great-grandchildren, etc. We can see
this by dividing through by the marginal distribution of parents(\( X_j \)) and observing that the
resulting distribution is a product of terms involving either \( X_j \) or non-descendants(\( X_j \)), but
not both. The Markov property allows one to efficiently deduce conditional independence
relationships and underpins Pearl’s algorithm (which will be described shortly).

Finally, a complete treatment of confounding in the causal diagram framework requires the
following definition:

**Definition 1.** A collider is a node/variable \( V \) in a DAG that sits on an undirected path
between two other nodes/variables, \( X_j \) and \( X_i \), and the paths both have arrows pointing into
\( V \).

Conditioning on a collider induces dependence between its parents. For a classic example
of this phenomenon, suppose that a certain college grants admission only to applicants with
high test scores and/or athletic talent. Even if we grant that in the general population these
talents may be independent, but among admitted students, these two attributes become highly
dependent. If we know that a student is not athletic, then we know for sure that they must be
academically gifted and vice-versa. While this is a basic result in probability theory, Pearl’s
work emphasized its significance to the problem of regression adjustment for causal effect
estimation.

With a DAG in hand, it is possible to deduce – rather than assume – conditional uncon-
foundedness: Pearl developed an algorithm for determining subsets of variables in \( X \) (i.e.,
its coordinate dimensions) that define valid regression estimators. The inputs to this algo-
rithm are a directed acyclic graph (DAG) that characterizes the causal relationships between
variables; such a graph describes a particular compositional representation of the joint distribution, reflecting conditional independences that are implied by the stipulated causal relationships. The prohibition on cycles rules out positive feedback self-causation. Here we present Pearl’s algorithm in a somewhat simplified form, assuming that the graph contains no descendants of \( Z \) other than \( Y \).

Given an input DAG, \( G \) and a subset of nodes \( S \), the “backdoor” algorithm proceeds as follows:

1. Identify all (undirected) paths between \( Z \) and \( Y \).
2. Consider each variable along each of these paths and make sure that at least one of them is “blocked”.
   a) A variable \( W \) is blocked if
      i. \( W \) is not a collider and is in the set \( S \) or
      ii. \( W \) is a collider and neither \( W \) nor any of its descendants is in the set \( S \).
3. Return \( \text{TRUE} \) if every “backdoor” path between \( Z \) and \( Y \) (all paths except the direct causal arrow from \( Z \) to \( Y \)), is blocked. Otherwise return \( \text{FALSE} \).

Sets of variables satisfying the backdoor criterion — those sets where the algorithm returns \( \text{TRUE} \) — are valid adjustment sets in the sense that \( Y \) and \( Z \) would be conditionally independent, given those variables, if there were no causal relationship between \( Y \) and \( Z \). By ruling out all other possible sources of association, any observed association may be interpreted as arising from a causal relationship.

2.2.2 Functional causal models. Causal DAGs may be associated with a functional causal model, a set of deterministic functions that take as inputs elements of \( X \) as well as independent (“exogenous”) error terms. The basic triangle confounding graph corresponding to an \((X, Y, Z)\) triple satisfying conditional unconfoundedness is shown in Figure 1. The corresponding functional causal model can be expressed as

\[
\begin{align*}
Z &\leftarrow G(X, \epsilon_z) \\
Y &\leftarrow F(X, Z, \epsilon_y)
\end{align*}
\]

where \( X, \epsilon_z \) and \( \epsilon_y \) are mutually independent (though all three may be vector-valued with non-independent elements). The exogenous errors \((\epsilon_z, \epsilon_y)\) that appear in a single equation are suppressed in the graph. All of the stochasticity is inherited from the exogenous variables, while all of the deterministic relationships are reflected in the functions \( G(\cdot) \) and \( F(\cdot) \), which are explicitly endowed with a causal interpretation. Specifically, the potential outcomes are given by:

\[
\begin{align*}
Y^1 &\leftarrow F(X, 1, \epsilon_y) \\
Y^0 &\leftarrow F(X, 0, \epsilon_y)
\end{align*}
\]

where \((X, \epsilon_y)\) are drawn from their marginal distributions, irrespective of the value of the treatment argument. As was mentioned previously, throughout this paper we assume that \( X \) does not contain any causal descendants of \( Z \).
Consider two ways to conceptualize the data generating process for both the potential outcome pairs, \((Y_0, Y_1)\), and the observed response \(Y\). On the one hand, the potential outcomes can be generated from the functional causal model, by fixing the \(Z\) argument to 0 or 1, irrespective of the implied distribution of \(Z\). Procedurally, this would look like drawing \(X\) from its marginal distribution, drawing \(\epsilon_y\), and evaluating \(F(X, 0, \epsilon_y)\) and \(F(X, 1, \epsilon_y)\). The observed data can then be constructed via the consistency assumption \(Y = F(X, 1, \epsilon_y)Z + F(X, 0, \epsilon_y)(1 - Z)\). Equivalently, \(Y\) may be drawn directly via \(F(X, Z, \epsilon_y)\), where \(Z\) (the observed treatment assignment) was drawn according to \(Z \mid X\) (as specified by the CDAG). This equivalence is especially instructive as to why \(Y \mid Z = z\) and \(Y^z\) do not generally have the same distribution and, furthermore, why \(Y \mid Z = z, X = x\) and \(Y^z \mid X = x\) do have the same distribution (assuming, as we have above, that \(X\) is causally exhaustive).

The role of \(\epsilon_y\) in defining the distribution of the potential outcomes is worth considering in more detail. Note that for a binary \(Z\), any functional causal model \(F\) may be rewritten as

\[
F(X, Z, \epsilon_y) = F(X, 0, \epsilon_y) + Z [F(X, 1, \epsilon_y) - F(X, 0, \epsilon_y)] = \mu(X, \epsilon_y) + Z\tau(X, \epsilon_y).
\]

This formulation invites us to consider that \(\epsilon_y\) may be multivariate, distinct elements of which may affect \(\mu(X, \epsilon_y)\) and \(\tau(X, \epsilon_y)\). Three particular cases are especially notable:

1. \(\mu(X, \epsilon_y) = \mu(X) + \epsilon_y\) and \(\tau(X, \epsilon_y) = \tau(X)\): here, \(\epsilon_y\) has the same effect on the two potential outcomes \(F(X, 1, \epsilon_y)\) and \(F(X, 0, \epsilon_y)\), so that their joint distribution is singular.
2. \(\mu(X, \epsilon_y) = \mu(X) + \epsilon_y, 0\) and \(\tau(X, \epsilon_y) = \tau(X) + (\epsilon_y, 1 - \epsilon_y, 0)\) where the exogenous error is partitioned as \(\epsilon_y = (\epsilon_y, 0, \epsilon_y, 1)\). Here, \(\epsilon_y, 0\) and \(\epsilon_y, 1\) are distinct random variables that separately define the potential outcome distributions so that one effect of the treatment is in changing which exogenous influences affect the response.
3. \(\mu(X, \epsilon_y) = \mu(X) + \epsilon_y, \mu\), \(\tau(X, \epsilon_y) = \tau(X) + \epsilon_y, \tau\), where the exogenous errors are partitioned as \(\epsilon_y = (\epsilon_y, \mu, \epsilon_y, \tau)\). In this case, a distinct set of causal factors dictate exogenous variation in the prognostic (baseline) response and exogenous variation in the treatment effect itself. For example, variation in the baseline response may be due to environmental factors that are independent from genetic factors dictating one’s response to a new drug.

These three cases are visualized in Figure 2 with \(\tau(X) = 1\). Empirically, these cases are indistinguishable in that they are “observationally equivalent” — because the potential outcomes are never jointly observed, most aspects of their joint distribution are fundamentally unidentified.

With a more detailed causal graph, a more detailed assessment of conditional unconfoundedness can be made. For instance, consider Figure 3, which is equivalent to the standard triangle digram in the sense that controlling for all of the elements of \(X = (X_1, X_2, X_3, X_4)\) indeed satisfies conditional unconfoundedness. However, Pearl’s algorithm reveals that \((X_1, X_2)\) would suffice. By positing more information about the joint distribution of \(X\), it is possible to absorb \(X_3\) into \(\epsilon_z\) and \(X_4\) into \(\epsilon_y\), while redefining \(X = (X_1, X_2)\), bringing us back to the triangle graph, but with a reduced set of control variables.

### 2.3 Structural equations: Mean regression models with exogenous additive errors

Finally, the classic econometric literature approaches causality in terms of mean regression models with additive (but not necessarily homoskedastic) error terms, which are referred to as “structural” models (although the term is often used informally and imprecisely in the applied literature). Heckman and Vytlacil (2005) reviews the structural model approach in econometrics in depth, noting that such methods have their origin in the study of dynamic macroeconomic systems. A seminal reference is Haavelmo (1943). The mean regression perspective arises naturally if one takes a linear regression model as a starting point, but is straightforward to motivate starting from a generic functional causal model.
FIG 2. Left panel: Potential outcome distributions with a common additive univariate error and a homogeneous treatment effect (which shifts the line up one unit from the diagonal), articulated in Case 1 below. Right panel: Potential outcome distributions with a homogeneous treatment effect and distinct additive bivariate errors, $\epsilon_y,0$ and $\epsilon_y,0$, shown here with a positive correlation less than one, articulated in Case 2 below.

FIG 3. An elaboration of the triangle graph, depicting $X_1$ and $X_2$ as confounders, $X_4$ as a pure prognostic variable, and $X_3$ is an instrument.

Define

$$
\begin{align*}
\mu(x) & \equiv E(F(x,0,\epsilon_y)), \\
\tau(x) & \equiv E(F(x,1,\epsilon_y)) - \mu(x), \\
v(x,\epsilon_y) & \equiv F(x,0,\epsilon_y) - \mu(x), \\
\delta(x,\epsilon_y) & \equiv F(x,1,\epsilon_y) - F(x,0,\epsilon_y) - \tau(x)
\end{align*}
$$

(5)

giving a “structural model”

$$
Y = \mu(x) + v(x,\epsilon_y) + (\tau(x) + \delta(x,\epsilon_y))Z
$$

(6)

where $v(x,\epsilon_y)$ and $\delta(x,\epsilon_y)$ are deterministic functions, both of which are mean zero integrating over $\epsilon_y$ (for any $x$): $E(v(x,\epsilon_y)) = 0$ and $E(\delta(x,\epsilon_y)) = 0$. In this formulation, conditional unconfoundedness may be expressed in terms of independence of the treatment, $Z$, and the error terms $v(x,\epsilon_y)$ and $\delta(x,\epsilon_y)$. Such models are commonly used in a simplified form, where $\delta(x,\epsilon_y)$ is assumed to be identically zero and $\tau(x)$ is assumed to be constant in $x$, but such assumptions are not intrinsic to the formalism.

2.4 Relating the three frameworks

If every node in a causal diagram is observable, all remaining factors determining $Y$ are attributable to the exogenous errors, which are, by definition, independent of the treatment assignment. In that case, it is easy to forge a connection between the three formalisms, as they all assert that

$$
Y \mid X = x \overset{d}{\sim} Y \mid X = x, Z = z,
$$

(7)
where (recall) $Y^z = F(x, z, \epsilon_y)$, with distribution induced by the distribution over $\epsilon_y$. The above assertion essentially declares that the estimable conditional distributions which appear on the right hand side warrant a causal interpretation.

For sets of control variables that are not exhaustive, more care is needed in translating the formalisms, but a precise relationship can be obtained, as spelled out in the following lemma.

**Lemma 1.** The assertions below (with their corresponding causal framework labeled in brackets) stand in the following logical relationship: $1 \Rightarrow 2 \Leftrightarrow 3$.

1. $S = s(X)$ satisfies the back-door criterion. [Causal DAGs]
2. $S = s(X)$ satisfies conditional unconfoundedness: $(Y^0, Y^1) \perp Z \mid S$. [Potential Outcomes]
3. The response $Y$ can be represented in terms of a mean regression model with error terms $(\upsilon(s, X, \epsilon_y), \delta(s, X, \epsilon_y)) \perp Z \mid s(X) = s$. [Structural Equations]

**Proof.** Let $X$ denote all of the variables in a complete causal diagram with the exception of the treatment variable $Z$ and response variable $Y$, and consider the following causal model, written in terms of functional equations, potential outcomes, and a structural mean regression with additive exogenous errors:

$$
Z \leftarrow G(X, \epsilon_z),
Y^z \leftarrow F(X, z, \epsilon_y) = \mu(X) + \upsilon(X, \epsilon_y) + (\tau(X) + \delta(X, \epsilon_y))z,
\begin{bmatrix}
Y^0 \\
Y^1
\end{bmatrix} \leftarrow
\begin{bmatrix}
\mu(X) + \upsilon(X, \epsilon_y) \\
\mu(X) + \tau(X) + \upsilon(X, \epsilon_y) + \delta(X, \epsilon_y)
\end{bmatrix}.
$$

(8)

To see that 1 implies 2, recall that 1 means that $S$ renders the treatment and response conditionally independent in the modified DAG with no causal arrow between $Z$ and $Y$. But it is precisely such a graph that defines the relationship between $Z$ and the potential outcomes $Y^0 = F(X, 0, \epsilon_y)$ and $Y^1 = F(X, 1, \epsilon_y)$, as shown in Figure 4.

To see that 2 and 3 are equivalent, re-parametrize the additive error model in terms of $S$, as follows:

$$
Y^z \leftarrow \mu(s) + \upsilon(s, X, \epsilon_y) + (\tau(s) + \delta(s, X, \epsilon_y))z
\mu(s) \equiv \mathbb{E}(\mu(X) \mid S(X) = s)
\tau(s) \equiv \mathbb{E}(\tau(X) \mid S(X) = s)
\upsilon(s, X, \epsilon_y) \equiv \mu(X) - \mu(s) + \upsilon(X, \epsilon_y)
\delta(s, X, \epsilon_y) \equiv \tau(X) - \tau(s) + \delta(X, \epsilon_y).
$$

(9)

For a fixed value of $s$, the mean terms $\mu(s)$ and $\tau(s)$ are constant, so that $(Y^0, Y^1)$ stands in a one-to-one relationship with $\upsilon(s, X, \epsilon_y)$ and $\delta(s, X, \epsilon_y)$; therefore if the former are independent of $Z$, then so must be the latter, and vice-versa.

\[\square\]
2.5 Estimands, estimators, and sampling distributions

As described previously, by treatment effect, we mean the difference between the treated and untreated potential outcomes. By average treatment effect, we mean the average of this difference over some population of individuals. The functional causal model and a distribution over the exogeneous errors define an infinite hypothetical population from which the observed data is assumed to be a random sample. From this perspective, the population average treatment effect (PATE) may be expressed as

$$E(\tau(X) + \delta(X, \epsilon)) = E(\tau(X)),$$

where $\tau$ is a fixed-but-unknown function and the expectation is taken with respect to the data generating process defined by the CDAG and the associated functional causal model, so that $X$ and $\epsilon$ are both being averaged over.

Other average causal effects, differing in terms of the (sub)population over which the average is taken, are likewise readily defined in terms of the functional causal model (FCM). For instance, if we wish to restrict our attention to the average treatment effect among individuals in our observed sample, we may define our estimand as the sample average treatment effect, or SATE:

$$\frac{1}{N} \sum_{i=1}^{N} (\tau(x_i) + \delta(x_i, \epsilon_i)).$$

Note that the SATE and the PATE differ from one another in that, in general,

$$E(\tau(X)) \neq \frac{1}{N} \sum_{i=1}^{N} \tau(x_i)$$

and

$$\frac{1}{N} \sum_{i=1}^{N} \delta(x_i, \epsilon_i) \neq E(\delta(X, \epsilon)) = 0.$$

In this paper, we will compare stratification estimators of the PATE, evaluating them in terms of their finite sample variance over repeated sampling of independent draws from $(X_i, Y_i, Z_i)$. While it would be possible to consider the sampling distribution over $(Y_i, Z_i)$ for a fixed vector of observed covariates $x_i$, doing so would make cross comparison of different stratifications impossible, because the sampling distribution would be over-specified relative to the coarser stratification. Because the PATE is of wide applied interest, we argue that averaging over observed control variables $X_i$ is sensible and all of our results are derived in this setting.

Another average treatment effect of broad interest is the conditional average treatment effect (CATE), which defines an average treatment effect conditional on a set of covariate values. The population CATE,

$$E(\tau(X) + \delta(X, \epsilon) \mid X = x) = E(\tau(X) \mid X = x),$$
takes an expectation with respect to a conditional sampling distribution \( \tau(X) \mid X = x \), where \( \{X = x\} \) may denote a set of covariates rather than a single value. While the focus of this paper is on the PATE, its insights extend automatically to the population CATE.

The CATE is sometimes mistakenly reported in the literature as the \textit{individual treatment effect} (ITE), which is a separate estimand that is only identified with more restrictive assumptions. The ITE is defined at the unit level as the difference in potential outcomes. For unit \( i \), the ITE is given by

\[
F(X_i, Z_i = 1, \epsilon_{i,y,1}) - F(X_i, Z_i = 0, \epsilon_{i,y,0}).
\]

This is unidentified without further assumptions on the nature of the error term, as in general \( \epsilon_{i,y,1} \neq \epsilon_{i,y,0} \); see Figure 2.

### 3. MINIMAL AND OPTIMAL STATISTICAL CONTROL

#### 3.1 The principal deconfounding function

Although conditional unconfoundedness is central to our conception of causal effect estimation, in fact it is a stronger than necessary assumption for identifying the ATE. More specifically, one only needs a function \( s(x) \) that satisfies \textit{mean conditional unconfoundedness}.

**Definition 2.** A function \( s \) on covariate space \( \mathcal{X} \) is said to satisfy \textit{mean conditional unconfoundedness} if

\[
Z \perp \perp (\mu(X), \tau(X)) \mid s(X).
\]

**Lemma 2.** \textit{Mean conditional unconfoundedness is a sufficient condition for estimating average treatment effects.}

**Proof.** Denote the causal model as

\[
Y^z \leftarrow \mu(X) + v(X, \epsilon_g) + (\tau(X) + \delta(X, \epsilon_g))z
\]

where \( \epsilon_g \perp (Z, X) \), \( \mathbb{E}(v(x, \epsilon_g)) = 0 \), and \( \mathbb{E}(\delta(x, \epsilon_g)) = 0 \) for all \( x \). We aim to show that

\[
\mathbb{E}(Y^z \mid s(X) = s) = \mathbb{E}(Y \mid s(X) = s, Z = z),
\]

from which the result follows by the estimability of the right hand side for both \( z = 0 \) and \( z = 1 \). Recalling the relationship between \( Y^z \) and \( Y \mid Z = z \) described in Section 2.2.2, this is equivalent to showing that

\[
\mathbb{E}(\mu(X) + v(X, \epsilon_g) + (\tau(X) + \delta(X, \epsilon_g))z \mid s(X) = s) = \mathbb{E}(\mu(X) + v(X, \epsilon_g) + (\tau(X) + \delta(X, \epsilon_g))z \mid s(X) = s, Z = z),
\]

where the expectation over \( (X, \epsilon_g) \) is with respect to its marginal distribution on the left hand side and with respect to its conditional distribution, given \( Z = z \), on the right hand side. By the independence of \( \epsilon_g \), the mean zero errors for each \( x \), and the linearity of expectation, this reduces to showing that

\[
\mathbb{E}(\mu(X) + \tau(X)z \mid s(X) = s) = \mathbb{E}(\mu(X) + \tau(X)z \mid s(X) = s, Z = z).
\]

By the assumption of mean conditional unconfoundedness, \( Z \perp \perp (\mu(X), \tau(X)) \mid s(X) \), and the result follows.

Mean conditional unconfoundedness can be used to define a \textit{minimal} control function, but first we must recall the definition of the propensity score (Rosenbaum and Rubin, 1983), which we will denote by \( \pi(\cdot) \).
DEFINITION 3. The propensity score, based on a vector of control variables \( x \), is the conditional probability of receiving treatment:

\[
\pi(x) \equiv \mathbb{P}(Z = 1 \mid X = x).
\]

It is common to interchangeably refer to the propensity score, which emphasizes a specific numerical value, \( \pi(x) \), and the propensity function, which emphasizes the mapping, \( \pi : \mathcal{X} \rightarrow (0, 1) \).

In turn, we have:

DEFINITION 4. The principal deconfounding function is given by following conditional expectation:

\[
\lambda(x) = \mathbb{E}(\pi(X) \mid \mu(X) = \mu(x), \tau(X) = \tau(x)).
\]

THEOREM 1. The principal deconfounding function is the coarsest function satisfying mean conditional unconfoundedness.

PROOF. By iterated expectation, \( Z \mid \mu(X), \tau(X) \) is a Bernoulli random variable with probability \( \lambda(X) \), therefore

\[
\mathbb{E}(Z \mid \tau(X), \mu(X), \lambda(X)) = \mathbb{E}(Z \mid \lambda(X)),
\]

which shows that \( Z \perp \perp (\mu(X), \tau(X)) \mid \lambda(X) \) because \( Z \) is binary.

Furthermore, \( |\lambda(\mathcal{X})| \) is minimal: it takes exactly as many values as there are unique conditional distributions of \( Z \mid \mu(X), \tau(X) \). In more detail, suppose \( s(x) \) is coarser than \( \lambda(x) \) so that there exists \( x_1 \) and \( x_2 \) such that \( s(x_1) = s(x_2) \) but \( \lambda(x_1) \neq \lambda(x_2) \). But \( \lambda(x_1) \neq \lambda(x_2) \) implies \( (\mu(x_1), \tau(x_1)) \neq (\mu(x_2), \tau(x_2)) \), which in turn shows that

\[
Z \not\perp \perp \mu(X), \tau(X) \mid s(X)
\]

so mean conditional unconfoundedness is violated. \( \square \)

3.2 Optimal stratification for causal effect estimation

Recognizing that valid control features are non-unique raises the question: which control features are the best ones? To make this question precise, we study the finite sample variance of fixed-strata estimators, restricting our attention to a vector of discrete control variables.

Without loss of generality, discrete control variables with finite support can be represented as a single covariate taking \( K = |\mathcal{X}| \) distinct values. For example, a length \( d \) vector of binary covariates would be represented as a single variable taking \( 2^d \) values. This assumption is mathematically convenient and, by setting \( K \) large enough, can capture most empirical applications to a satisfactory degree of realism. (We revisit the plausibility of this assumption in the discussion section.) In the mathematical formalism and discussion of this paper, we will use the words “strata” and “features” interchangeably, to refer to functions of this single categorical variable.

In detail, this paper considers the following data generating process:

\[
\mathcal{X} = \{1, \ldots, K\},
\]

\[
\pi : \mathcal{X} \mapsto (0, 1),
\]

\[
Z \sim \text{Bernoulli}(\pi(X)),
\]

\[
Y \leftarrow \mu(X) + \nu_X + (\tau(X) + \delta_X)Z
\]

where \( \mathbb{E}(\nu_x) = 0 \) and \( \mathbb{E}(\delta_x) = 0 \) for all \( x \) so that \( \mu(x) = \mathbb{E}(Y \mid X = x, Z = 0) \) and \( \mu(x) + \tau(x) = \mathbb{E}(Y \mid X = x, Z = 1) \). Lastly, let the random variable \( N \) denote the overall sample size and define subset-specific sample sizes as follows:

- \( N_x \): the number of observations with \( X = x \),
• $N_{x,z}$: the number of observations with $X = x$ and $Z = z$.

We define the stratification estimator using a stratification function $s(\mathcal{X})$, which returns $J \leq K$ discrete function values. We compute the average difference in outcomes between the treated and control groups separately for individuals in each of the $J$ strata, so that

$$\bar{T}_{\text{strat}}^s = \sum_{j \in s(\mathcal{X})} \frac{N_j}{n} \{ \bar{Y}_{j,1} - \bar{Y}_{j,0} \}$$

where $N_j = \sum_{i=1}^n 1 \{ s(X_i) = j \}$

$$N_{j,0} = \sum_{i=1}^n 1 \{ s(X_i) = j \} \{ Z_i = 0 \} \quad N_{j,1} = \sum_{i=1}^n 1 \{ s(X_i) = j \} \{ Z_i = 1 \}$$

$$\bar{Y}_{j,0} = \frac{1}{N_{j,0}} \sum_{i=1}^n Y_i 1 \{ s(X_i) = j \} \{ Z_i = 0 \} \quad \bar{Y}_{j,1} = \frac{1}{N_{j,1}} \sum_{i=1}^n Y_i 1 \{ s(X_i) = j \} \{ Z_i = 1 \}$$

Note that if we choose the trivial stratification $s(x) = x$, we stratify completely on all $K$ unique levels of $\mathcal{X}$.

The following theorem describes when stratification beyond the minimal valid stratification, $\lambda(X)$, is beneficial, in terms of conditions on the underlying data generating process.

**Theorem 2.** Assume we have stratified on $\lambda(X)$ so that the average treatment effect is identified using a minimal deconfounding set. Consider a refinement of $\lambda$, $s(\mathcal{X})$, which also identifies the ATE: $s(x) \neq s(x')$ while $\lambda(x) = \lambda(x')$ for at least two $x, x' \in \mathcal{X}$. Define $\bar{T}_{\text{strat}}^s$ as a stratification estimator which uses level sets of $\lambda(X)$ to define strata and $\bar{T}_{\text{strat}}^\lambda$ as a stratification estimator which uses level sets of $s(\mathcal{X})$. Then $\mathbb{V}(\bar{T}_{\text{strat}}^s) < \mathbb{V}(\bar{T}_{\text{strat}}^\lambda)$ if $\nu < \eta$ where

$m(j) = |\{ s(x) : x \in \mathcal{X} \text{ such that } \lambda(x) = j \}|$

$\mathcal{B} = \{ j \in \lambda(\mathcal{X}) : m(j) > 1 \text{ and all sub-strata means and variances are constant} \}$

$\mathcal{C} = \{ j \in \lambda(\mathcal{X}) : m(j) > 1 \text{ and either the sub-strata means or variances are non-constant} \}$

$$\nu = \sum_{b \in \mathcal{B}} \left[ \mathbb{V} \left( \frac{N_b}{n} (\bar{Y}_{b,1} - \bar{Y}_{b,0}) \right) - \mathbb{V} \left( \sum_{t=1}^{m(b)} \frac{N_{b,t}}{n} (\bar{Y}_{b,t,1} - \bar{Y}_{b,t,0}) \right) \right]$$

$$\eta = \sum_{c \in \mathcal{C}} \left[ \mathbb{V} \left( \sum_{t=1}^{m(c)} \frac{N_{c,t}}{n} (\bar{Y}_{c,t,1} - \bar{Y}_{c,t,0}) \right) - \mathbb{V} \left( \frac{N_c}{n} (\bar{Y}_{c,1} - \bar{Y}_{c,0}) \right) \right]$$

and $\mathbb{V}(\bar{T}_{\text{strat}}^s) \geq \mathbb{V}(\bar{T}_{\text{strat}}^\lambda)$ otherwise.

A detailed proof is provided in Appendix A, but here we offer a sketch of the proof to build intuition. In comparing two stratifications, $\lambda$ and $s$, across discrete covariates $X$, we can partition the level sets of the two stratification functions as follows:

1. $\mathcal{A}$: values of $x \in \mathcal{X}$ for which both $\lambda$ and $s$ agree
2. $\mathcal{B}$: values of $x \in \mathcal{X}$ for which $s$ substratifies $\lambda$ but the mean and variance of $Y \mid Z$ are constant across substrata formed by $s$
3. $\mathcal{C}$: values of $x \in \mathcal{X}$ for which $s$ substratifies $\lambda$ and either the mean of $Y \mid Z$, the variance of $Y \mid Z$, or both vary across substrata formed by $s$

We ignore $\mathcal{A}$ and focus on $\mathcal{B}$ and $\mathcal{C}$. In the case of $\mathcal{B}$, $s$ performs “unnecessary” stratification, estimating and re-aggregating conditional means which are the same in the underlying data generating process, and thus incurs additional variance over the $\lambda$ stratification estimator. On the other hand, when we consider $\mathcal{C}$, $\lambda$ incurs additional variance over $s$ by failing to control for differences in the $Y \mid Z$.

In summary, $\mathcal{B}$ induces a variance penalty on $s$ relative to $\lambda$ by “overstratification”, while $\mathcal{C}$ induces a variance penalty on $\lambda$ relative to $s$ by “understratification.” Which estimator is...
preferred depends on the magnitude of these competing effects, as articulated in the \( \nu < \eta \) inequality above. The practical upshot of this theorem is that stratification that accounts for substantial variation in the response will tend to reduce variance of the treatment effect estimator (whether or not it is confounded in the sense of covarying with propensity to receive treatment), while stratification that accounts only for variation in treatment assignment will increase variance of the treatment effect estimator. This conclusion is illustrated in the examples of the following section.

4. VIGNETTES

This section collects examples illustrating the statistical trade-offs underlying feature selection for causal effect estimation that are articulated in Theorem 2. Many of the examples are interesting in their own right; connections to previous literature are provided throughout.

4.1 In what sense is randomization the “gold standard” for causal effect estimation?

It has become boiler-plate in reports on observational studies to remark that “in the absence of the gold standard of a randomized clinical trial, one may pursue statistical methods to control for confounding”. But in what sense is randomized treatment assignment the gold standard? Surely solid-state physicists do not randomize their lab conditions and hope their sample size is large enough to reveal interesting results. Famously, esteemed physicist Ernst Rutherford quipped “If your experiment needs statistics, you ought to have done a better experiment” (Hammersley (1962)). The intuition behind this remark is that it is control that is central, not randomization. See section 4.4 for a definition of a control feature that evokes the experimental notion of “control”.

Indeed, randomization is simply a way to guarantee control on average in the event that exact control is impossible, such as when crucial confounding factors are unobserved. This perspective in turn suggests that controlling for factors that we can observe and randomizing only for factors that we cannot observe would be the ideal approach. The following thought experiment amplifies this intuition.

Consider studying the effect of treatment \( Z \) on outcome \( Y \) in a sample of \( n \) pairs of identical twins and deciding how to allocate treatment across the \( 2n \) study participants. Completely randomized treatment assignment satisfies the assumptions outlined above and thus identifies the treatment effect. However, a naive randomization would sometimes accidentally treat both twins and leave other twin pairs untreated. This violates most people’s intuition about why twin studies are interesting and useful, which is that giving one twin the treatment and the other a placebo implicitly “controls for” all of the shared biological and environmental factors that may impact the treatment effect. Randomization within each twin pair can protect against unmeasured factors that may confound the result, such as (perhaps) which twin was born first.

In this case, both \( Z \) and the twin pair index, \( X \), are informative about the expected value of \( Y \). Now consider four possible approaches to study the effect of \( Z \) on \( Y \):

| Design | Estimator |
|--------|-----------|
| 1      | Complete randomization | Unadjusted mean difference |
| 2      | Twin pair randomization | Unadjusted mean difference |
| 3      | Complete randomization | Adjusted mean difference |
| 4      | Twin pair randomization | Adjusted mean difference |

where the unadjusted mean difference estimator is defined as

\[ \bar{\tau}_U = \bar{Y}_{Z=1} - \bar{Y}_{Z=0} \]

and the adjusted mean difference estimator is defined as

\[ \bar{\tau}_A = \sum_{x \in \mathcal{X}} \frac{n_x}{n} (\bar{Y}_{X=x,Z=1} - \bar{Y}_{X=x,Z=0}) \]

where \( \mathcal{X} \) is the set of twin pairs and \( X \) is a variable that indexes twin pairs.
Each of the four approaches above identifies the ATE. However, adjusting for twin pairs (approaches 3 and 4) will tend to reduce variance over the unadjusted alternatives (1 and 2) and, similarly, designs that incorporate twin pairs in randomization (2 and 4) will also see a reduction in variance over the completely randomized alternatives (1 and 3). These results are implicit in Theorem 2, which can be applied even if the propensity function is constant, as in a randomized trial.

As intuitive as this example may be, and despite its lesson being a straightforward implication of Theorem 2, regression adjustment for randomized trial data remains controversial. Freedman (Freedman, 2008a,b) criticized regression adjustment on the grounds that linear or linear logistic regression is potentially biased. Unfortunately, many researchers took this advice without first considering non-linear alternatives. Lin (2013) shows that regression adjustment in experimental data is not asymptotically unbiased if one entertains a richer set of interacted or saturated models, rather than a basic linear model. Of course, the stratification estimators studied here are fundamentally nonparametric and so are consistent with the conclusions of Lin (2013). At the same time, Theorem 2 concedes that for some data generating processes, undertaking a regression adjustment (via stratification) would simply produce unnecessary variability, specifically for data generating processes where the available control factors are not sufficiently predictive of the response. In many applied problems we find ourselves somewhere in between this case of mostly useless controls and the twin experiment situation of profoundly informative controls.

4.2 Propensity scores

Following the work of Rosenbaum and Rubin (1983), the propensity score (expression 11) has become a central element in many applied analyses of causal effects. In that paper, it was first shown that $\pi(x)$ satisfies conditional unconfoundedness, from which it follows that

$$\text{ATE} = E[Y_i - Y_0] = E[\pi(X)]E[Y | \pi(X), Z = 1] - E[Y | \pi(X), Z = 0].$$

This differs from the more general form of conditional unconfoundedness in that $\pi(X)$ is one-dimensional, while $X$ itself typically involves many controls.

An especially common use of the propensity score in practice is via the inverse-propensity weighted (IPW) estimator

$$\bar{\tau}_{\text{ipw}} = \frac{1}{N} \sum_{i=1}^{N} \left( \frac{Y_i Z_i}{\pi(X_i)} - \frac{Y_i (1 - Z_i)}{1 - \pi(X_i)} \right),$$

which is known to be consistent and has been widely studied theoretically.

Here we re-examine a curious result of Hirano, Imbens and Ridder (2003) which shows that an IPW estimator based on estimated propensity scores attains lower asymptotic variance than one based on the true propensity function. We can apply the finite-sample results of Theorem 2 to re-evaluate the meaning of this widely-known result by noting the following correspondence between IPW estimators and stratification estimators:

**Lemma 3.** The empirical inverse propensity weighting (IPW) estimator is equivalent to $\bar{\tau}_{\text{strat}}$ under the following conditions:

1. $X$ is discrete,
2. For all $x \in X$, $N_{x,1} > 0$ and $N_{x,0} > 0$,
3. The propensity weighting function is estimated nonparametrically as $\hat{\pi}(x) = N_{x,1}/N_x$ for each $x \in X$.
PROOF. By direct calculation,

\[ \tilde{\pi}_{ipw}^x = \frac{1}{n} \sum_{i=1}^{n} \left( \frac{Y_i Z_i}{\hat{\pi}(X_i)} - \frac{Y_i (1 - Z_i)}{1 - \hat{\pi}(X_i)} \right) \]

\[ = \frac{1}{n} \sum_{i=1}^{n} \left( \frac{Y_i Z_i}{N_{x_i,1}/N_{x_i}} - \frac{Y_i (1 - Z_i)}{N_{x_i,0}/N_{x_i}} \right) = \frac{1}{n} \sum_{i=1}^{n} \left( \frac{Y_i Z_i}{N_{x_i,1}} - \frac{Y_i (1 - Z_i) N_{x_i}}{N_{x_i,0}} \right) \]

\[ = \frac{1}{n} \sum_{x \in \mathcal{X}} \left( \frac{N_x}{N_{x,1}} \left( \sum_{i : X_i = x} Y_i Z_i \right) - \frac{N_x}{N_{x,0}} \left( \sum_{i : X_i = x} Y_i (1 - Z_i) \right) \right) \]

\[ = \frac{1}{n} \sum_{x \in \mathcal{X}} \left( \frac{N_x}{N_{x,1}} \bar{Y}_{x,1} - \frac{N_x}{N_{x,0}} \bar{Y}_{x,0} \right) = \frac{1}{n} \sum_{x \in \mathcal{X}} \frac{N_x}{N_{x,1}} \bar{Y}_{x,1} - \frac{N_x}{N_{x,0}} \bar{Y}_{x,0} = \tilde{\pi}_{strat}^x. \]

First, we give a finite-sample analogue of the Hirano, Imbens and Ridder (2003) result in the stratification context. Then, we demonstrate a modified estimator based on a known propensity score that improves upon the estimated propensity score IPW.

4.2.1 "Noisy estimates of one". Denote a candidate propensity function by \( q : \mathcal{X} \rightarrow (0, 1) \), so that the corresponding IPW estimator is

\[ \tilde{\pi}_{ipw}^q = \sum_x \left( \frac{N_x}{N} \right) \tilde{\pi}_{ipw}^{q,x} \]

where

\[ \tilde{\pi}_{ipw}^{q,x} = \left( \frac{\hat{\pi}(x)}{q(x)} \bar{Y}_{x,Z=1} - \frac{1 - \hat{\pi}(x)}{1 - q(x)} \bar{Y}_{x,Z=0} \right) \]

and \( \hat{\pi}(x) = (N_{x,x=1}/N_x) \) is the proportion of treated units in each stratum.

Taking \( q(x) = p(x) \) is the “true propensity score” case, while letting \( q(x) = \hat{\pi}(x) \) is the “estimated propensity score” case. In the former case, the treated and untreated stratum averages are weighted by \( \hat{\pi}(x)/\pi(x) \) and \( (1 - \hat{\pi}(x))/(1 - \pi(x)) \), respectively; in the latter case the weights are identically one. This difference in weights leads to the following analogue of the result of Hirano, Imbens and Ridder (2003):

**Theorem 3.** There exists some \( \epsilon > 0 \) such that if \( |\mu(x)| + |\tau(x)| > \epsilon \) for at least one \( x \in \mathcal{X} \),

\[ \forall \left( \sum_{x \in \mathcal{X}} \tilde{\pi}_{ipw}^{\hat{\pi},x} \right) \leq \forall \left( \sum_{x \in \mathcal{X}} \tilde{\pi}_{ipw}^{\pi,x} \right). \]

Essentially, the random weights in the true propensity IPW are only adding variability, compared to the IPW based on estimated weights, where exact cancellation occurs. A proof may be found in the appendix. Of course, there are many other possible IPW estimators, such as those based on parametric estimates. However, any parametric form will have a similar problem to the true propensity IPW if exact cancellation is not obtained.

4.2.2 The dimension reduction benefit of known propensity scores. Armed with an understanding of why the estimated propensity weights outperform the true propensity weights permits us to consider a modified estimator that is able to make use of knowledge of the true propensity scores (should they be known). Suppose \( K_{\pi} = |\pi(\mathcal{X})| < |\mathcal{X}| = K \). If \( \pi \) were
known exactly prior to estimating the average treatment effect, this reduction in the strata
should confer a benefit in terms of variance reduction — there are simply fewer conditional
expectations to estimate and there are more data available for estimating each one. Moreover,
it is still possible to avoid the noisy-estimation-of-one effect by estimating the propensity
score values on the level sets of \( \pi \); letting \( \rho \in \pi(\mathcal{X}) \) denote a specific value in the range of \( \pi \)
gives

\[
\bar{\tau}_{ipw}^{\pi,\rho} = \left( \frac{\bar{\pi}(\rho)}{\bar{\pi}(\rho)} \bar{Y}_{\rho,Z=1} - \frac{1 - \bar{\pi}(j)}{1 - \bar{\pi}(\rho)} \bar{Y}_{\rho,Z=0} \right)
\]

(17)

More precisely:

**Corollary 1.** Suppose the following conditions hold:

1. If \( \pi(x) = \pi(x') \), then \( \mu(x) = \mu(x') \) and \( \tau(x) = \tau(x') \),
2. \( \mathbb{V}(\epsilon \mid X = x) = \sigma_j^2 \) for all \( x \) with \( \pi(x) = j \) and for all \( j \in \pi(\mathcal{X}) \), and
3. \( |\pi(\mathcal{X})| < |\mathcal{X}| \).

Then

\[
\mathbb{V} \left( \sum_{j \in \pi(\mathcal{X})} \bar{\tau}_{ipw}^{\pi,j} \right) \leq \mathbb{V} \left( \sum_{j \in \pi(\mathcal{X})} \left( \sum_{x \in \pi(x) = j} \frac{N_x}{N_j} \bar{\tau}_{ipw}^{\pi,x} \right) \right).
\]

This result formalizes the intuition that fewer strata implies a greater degree of aggregation
and that, with larger sample sizes in the remaining strata, estimation should be accordingly
more efficient. In other words, knowledge of the true propensity function permits feature
selection, after which the empirical propensities can be used in an IPW estimator (which is
equivalent to the stratification estimator on the selected features).

Condition one requires some explanation: in the fixed-strata case “over-stratification” can
actually be beneficial if the additional strata are predictive of the response itself and condition
one rules out this possibility, as it states that \( \mu-\tau \) is at least as coarse as \( \pi \). That is, in
addition to the “noisy-estimation-of-one” phenomenon, empirical estimates of the propensity
score can benefit from being defined on strata that are predictive of the response, but not the
treatment assignment; this benefit is not directly related to the true-versus-actual propensity
score question, but merely reflects the fact that controlling for prognostic factors can benefit
treatment effect estimation.

**4.2.3 The inefficiency of instrumental controls.** While a known propensity score can poten-
tially aid IPW estimation by preventing unnecessary stratification, an additional corollary of Theorem 2 tells us that stratification based on a known propensity function may produce
unnecessary stratification as a result of unconfounded variation in propensity scores, which we refer to as “instrumental” stratification.

**Corollary 2.** Define a stratification \( s \) such that \( |s(\mathcal{X})| < |\pi(\mathcal{X})| \) and define \( g : \pi(\mathcal{X}) \to s(\mathcal{X}) \) as a function that collapses level sets of \( \pi \) into level sets of \( s \). Let \( m(j) = |\{ \pi(x) : g(\pi(x)) = j \}| \) and suppose the following conditions hold:

1. There exist \( x, x' \) such that \( \pi(x) \neq \pi(x') \) while \( s(x) = s(x') \), \( \mu(x) = \mu(x') \), and \( \tau(x) = \tau(x') \),
2. \( \mathbb{V}(\epsilon \mid \pi(X) = p) = \sigma_j^2 \) for all \( x \) with \( \pi(x) = p \) and \( g(\pi(x)) = j \) and for all \( j \in s(\mathcal{X}) \).

Then,

\[
\mathbb{V} \left( \sum_{j \in s(\mathcal{X})} \bar{\tau}_{ipw}^{\pi,j} \right) \leq \mathbb{V} \left( \sum_{j \in s(\mathcal{X})} \left( \sum_{\pi: g(\pi) = j} \frac{N_{\pi}}{N_j} \bar{\tau}_{ipw}^{\pi,\pi} \right) \right).
\]

This corollary and the other results of this subsection provide rigorous finite-sample cor-
roboration of the advice offered in Hernan and Robins (2022) quoted in the introduction.
4.3 Generalized prognostic scores

In data generating processes where variation in \( \tau \) is independent of \( Z \), the prognostic score, \( \mathbb{E}(Y^0 \mid X = x) = \mu(x) \), is a sufficient control function. This follows because mean conditional unconfoundedness is satisfied trivially by \( s(X) = \mu(X) \) when \( \tau(X) \perp \! \! \! \perp Z \); see Lemma 2. Like the propensity score, the prognostic score can be estimated from partially observed data — the propensity score can be estimated from \((X, Z)\) pairs and the prognostic score can be estimated from control units only, \((X, Z = 0, Y)\), which in many contexts are more readily available than treated observations. See Hansen (2008) for a rigorous exposition of prognostic scores.

The vector-valued function \((\mu, \tau)\) is a “generalized” prognostic score, containing both the usual prognostic score, as well as the treatment effect itself. This version of the prognostic score has received little attention, presumably because it “begs the question”, in that one of its elements is the very estimand of interest. However, note that conditioning on a random variable is not about the values of that variable per se, but is rather about the level sets of the function defining that random variable. In particular, any one-to-one function of \( \mu - \tau \) also satisfies mean conditional unconfoundedness; knowledge of the treatment effect itself is not required, merely knowledge of which strata have distinct treatment effects. Note also that Theorem 2 suggests that prognostic strata are more desirable from an estimation variance perspective, suggesting, perhaps counterintuitively, that large control groups may be advantageous in practice and that investing in data collection of prognostic factors should be prioritized in cases where randomization of treatment assignment is not possible.

4.4 Constant control function

The previous two examples showed that propensity scores and prognostic scores are sufficient control functions; this example demonstrates a function that may be coarser than either one. Consider a function on \( X \) defined as follows:

**Definition 5.** A function \( s \) on \( X \) is a constant control function if for all \( x, x' \in X \) such that \( s(x) = s(x') \) at least one of the following holds

- \( \pi(x) = \pi(x') \),
- \( \mu(x) = \mu(x') \) and \( \tau(x) = \tau(x') \).

In other words, a constant control function is a coarsening of \( X \) such that on each level set defined by \( s \), either \( \pi(x) \) or \( (\mu(x), \tau(x)) \) are constant. The following lemma shows that a constant control function defines a random variable \( S = s(X) \) such that \( \mathbb{E}(Y \mid Z = z, S) = \mathbb{E}(Y^{z} \mid S) \).

**Lemma 4.** Assume \( X \) satisfies conditional unconfoundedness and consider the random variable \( S = s(X) \), where \( s \) is a constant control function; then \( S \) satisfies conditional unconfoundedness.

**Proof.** Consider the causal diagram of \((X, Z, Y)\) expanded to include random variables \( X_p = \pi(X), X_y = (\mu(X), \tau(X)) \), and \( X_c = s(X) \) for \( s \) defined above, depicted in panel (a) of Figure 7. Integrating out \( X \) leads to a probabilistic graphical model as shown in panel (b) of Figure 7; dashed lines denote not-necessarily causal probabilistic dependence and solid arrows denote causal relationships. The result follows by showing that \( X_p \perp \! \! \! \perp X_y \mid X_c \); in terms of the diagram this means that the curved dashed line does not exist. But this follows immediately from the definition of \( X_c \). For any value of \( X_c \), either \( X_p \) or \( X_y \) is constant, and so the conditional distribution of \( X_p \) and \( X_y \) is trivially a product distribution. \( \square \)

The intuition behind a constant control function is that one way to control for “systematic co-variation” is simply to remove all variation. Clearly, both \( \pi(X) \) and \( (\mu(X), \tau(X)) \) are
themselves constant control functions, as is $X$ itself. However, a constant control function may be coarser than either, as illustrated in Figure 8, which shows an example of a simple data generating process that has a constant control function. In this example, the CCDR comprises just two strata, although $\mu$ and $\pi$ take 10 and 11 unique values, respectively, and $|\mathcal{X}| = 20$. The treatment effect is heterogeneous but unconfounded: $\tau \sim U(5, 10)$. The second panel of Figure 8 shows the sampling distributions of four different stratification estimators: one using level sets of $\mu$, one using level sets of $\pi$, one using all 20 values of $x$, and one using the two values of the minimal constant control function, indicating if $x \leq 11$. All four stratification estimators are unbiased, but their differing variances exhibit a pattern consistent with Theorem 2: $\mu$ gives the lowest variance, followed by $x$, followed by the constant control function, followed by $\pi$.

4.5 Independent variables in both $\pi$ and $(\mu, \tau)$.

When the coordinates of $X$ (the nodes in the graph) are all mutually independent, a valid control set is the elements $X_j$ occurring in both the propensity model and (at least one of) the prognostic and moderation models. For example, this was the strategy used in concocting the example DGP presented in section 4.10. As a more general example, if $\pi(x_1, \ldots, x_d) = \pi(x_1, x_2)$, $\mu(x_1, \ldots, x_d) = \mu(x_2, x_3)$, and $\tau(x_1, \ldots, x_d) = \tau(x_4, x_5)$, and $X_1 \perp \perp X_j$ for $j \neq$
If the coordinate dimensions of $X$ are independent, then which variables $X_j$ appear (or not) in the eight possible combinations of $\pi$, $\mu$, and $\tau$ can be used to characterize four relevant variable types with respect to treatment effect estimation: necessary controls, pure prognostic variables, instruments, and extraneous (or noise) variables. The above Venn diagram depicts these eight regions, shaded according to these designations. Variables in the cross-hatched region are necessary controls, as they appear in both $\pi$ and either $\mu$ or $\tau$ (or both). The gray shaded region corresponds to pure prognostic variables, appearing in $\mu$ or $\tau$ (or both), but not appearing in $\pi$. The white region corresponds to instruments, variables which appear in $\pi$, but neither in $\mu$ nor $\tau$. Variables outside of the three circles are entirely irrelevant to either the outcome or the treatment. Such designations become considerably more complicated when the elements of $X$ are not independent (cf. Example 4.6).

Consider the causal diagram in Figure 5. Either the propensity controls $(X_1, X_3)$ or the prognostic-moderation controls $(X_2, X_4)$ are adequate for statistical control. However, Pearl’s algorithm tells us that “mixed” variables also suffice, such as $(X_1, X_4)$ or $(X_2, X_3)$. Interestingly, such examples show that the notion of “instrumental” variables and “prognostic” variables are context dependent. Specifically, relative to a conditioning set of $(X_2, X_3)$, additional stratification using $X_4$ is prognostic, while additional stratification on $X_1$ would be instrumental. Theorem 2 suggests that adding prognostic controls is often desirable, while adding instruments should be avoided, but such designations will fluctuate depending on what has already been included.

This example also illustrates a limitation of the triangle graph. Suppose that only $(X_2, X_3)$ were available for measurement. The resulting diagram for just those two controls (Figure 6) is not the usual causal diagram, because $X_2$ has no causal impact on $Z$, while $X_3$ has no causal impact on $Y$. Accordingly, there is no unaugmented CDAG describing $(X_2, X_3, Z, Y)$; instead, we must denote merely statistical relationships using dashed lines. When a practitioner invokes conditional unconfoundedness in the potential outcomes framework, it therefore does not imply the triangular CDAG.

Similarly, invoking (conditionally) exogenous errors does not imply that the resulting mean components of the structural model are causal. In more detail, if the potential outcomes are defined in terms of the CDAG on the full set $(X_1, X_2, X_3, X_4)$, a structural model can be derived that only involves $(X_2, X_3)$, as follows:
Noting that the resulting error terms now depend not only \( \epsilon (18) \),

But this follows from the fact that \( x \),

However, reflecting on invertible transformations such as

on how the control variables are parametrized. This scenario is not commonly discussed, pre-

4.7 Sets satisfying the back-door criterion according to a transformed CDAG.

This example considers a data generating process that admits distinct CDAGs, depending

on how the control variables are parametrized. This scenario is not commonly discussed, presumably because observed measurements are taken to be designated by “nature”, so to speak.

However, reflecting on invertible transformations such as \((x_1, x_2) \rightarrow (x_1, x_1/x_2)\) highlights that functional causal models are certainly subject to changes of variables.

More concretely, consider the following DGP:

\[
X_j \overset{iid}{\sim} \text{Bernoulli} \left( p_j \right) \\
\pi(X) = \beta_0 + \beta_1 (2X_1X_2 - X_1 - X_2 + 1) + \beta_2X_3 \\
Z \overset{iid}{\sim} \text{Bernoulli} \left( \pi(X) \right) \\
\mu(X) = \alpha_0 + \alpha_1 (2X_1X_2 - X_1 - X_2 + 1) + \alpha_2X_4 \\
\tau(X) = \tau \quad \text{(constant treatment effect)} \\
Y = \mu(X) + \tau(X)Z + \epsilon, \quad \epsilon \sim \mathcal{N}(0, \sigma^2) 
\]

Next, define random variable \( W = (2X_1X_2 - X_1 - X_2 + 1) \), regarding \( X_2 \) as the exogeneous variable in the functional model for \( W \mid X_1 \). Additionally, suppressing \( X_3 \) and \( X_4 \), as they represent exogenous variation, yields the causal graph in Figure 11.

From this graph, it is clear that conditioning on \( W \) satisfies conditional unconfounded-

ness. Most interestingly, \(|\mu(X)| = |\pi(X)| = 4\). while \(|W| = 2\); thus \( W \) provides the smallest possible random variable. Indeed, the level sets of \( W \) are exactly the level sets of \( \lambda \):

\[
\mathbb{E}(\pi(X) \mid \mu(X)) = \mathbb{E}(\pi(X) \mid W, X_4) = \mathbb{E}(\pi(X) \mid W).
\]

4.8 Sets that induce collider bias in a graph without colliders

We see in Section 4.7 that conditioning on synthetic “features” that combine existing vari-

ables can lead to smaller control sets than their component variables. It is thus perhaps nat-

ural to consider machine learning useful in searching for and constructing such sets. It is
true that certain combinations of confounding variables may create a synthetic, minimal de-
confounders. However, it is also possible to combine two independent variables to create a
“collider” (defined in Section 2.2) which confounds the causal effect of $Z$ on $Y$ after condi-
tioning.

Consider the graph in Figure 12 and define its data generating equations as

\[
Y \sim \mathcal{N}(\alpha X_2 + \tau Z, \sigma^2)
\]

\[
Z \sim \text{Bernoulli}\left(\frac{1}{4} + X_1/2\right)
\]

\[
X_1, X_2 \sim \text{Bernoulli}\left(\frac{1}{2}\right)
\]

From this graph, we can see that the average causal effect of $Z$ on $Y$ is identified uncondi-
tional of $X_1$ and $X_2$, though we may condition on either or both variables. Suppose we
construct two synthetic variables

\[
\tilde{X}_A = \min\left\{X_1, X_2\right\}
\]

\[
\tilde{X}_B = a[1\{X_1 == 1\}1\{X_2 == 1\} + 1\{X_1 == 0\}1\{X_2 == 0\}] + b[1\{X_1 == 1\}1\{X_2 == 0\}] + c[1\{X_1 == 0\}1\{X_2 == 1\}]
\]

where the unique values of categorical $\tilde{X}_B$ may be treated as strata of a conditioning set.

We show in the simulation results in Table 1 that conditioning on either $\tilde{X}_A$ or $\tilde{X}_B$ biases
the average treatment effect, while conditioning on both $X_1$ and $X_2$ does not. Note that both
$\tilde{X}_A$ and $\tilde{X}_B$ are constructed in a manner not unlike the “feature learning” step of common
machine learning algorithms, such as neural networks and decision trees.

4.9 Sets satisfying the back-door criterion with respect to a mean CDAG.

The structural model perspective permits us to produce, starting from a given CDAG, a
modified causal diagram that reflects only the mean dependencies. For estimation of average

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**FIG 10. Causal graph in terms of original covariates**

**FIG 11. Causal graph under transformed covariates**

**FIG 12. Graph with no confounding and no colliders**

**TABLE 1**

Simulation results for 10,000 simulations of $n = 1,000$ of the above DGP

| Control Set | Bias   | Log-likelihood |
|-------------|--------|----------------|
| $\emptyset$ | 0.00   | -3.143         |
| $X_2$       | 0.00   | -2.352         |
| $X_1, X_2$  | 0.00   | -2.350         |
| $\tilde{X}_A$ | -1.72 | -2.967         |
| $\tilde{X}_B$ | 2.50  | -2.798         |
causal differences, such a graph suffices to identify valid control variable sets that are potentially smaller than any control set satisfying the back-door criterion on the original CDAG.

For example, consider the following data generating process:

\[
X \sim \text{Bernoulli}(1/2),
\]
\[
Z \sim \text{Bernoulli}(1/4 + X/2)
\]
\[
Y \sim \mathcal{N}(\tau Z, (\sigma + X)^2)
\]

For this DGP, \(\mu(X) = 0\) and \(\tau(X) = \tau\) are both constant in \(X\), which implies that the null set satisfies mean conditional unconfoundedness; even though \(X\) is a common cause of \(Z\) and \(Y\), it only affects the variance of \(Y\), but not the mean. Therefore, the full joint distribution of \(X, Z, Y\) is the triangle diagram of Figure 6, while Figure 13 depicts the joint distribution of \((X, Z, \mathbb{E}(Y \mid X, Z))\), in which \(X\) is unconnected to \(\mathbb{E}(Y \mid X, Z) = \mathbb{E}(Y \mid Z)\).

Note that while mean conditional unconfoundedness identifies the ATE, it does not identify other causal estimands. For instance, consider the quantile treatment effect (QTE), for \(q \in (0, 1)\):

\[
F_{Y^1}(q) - F_{Y^0}(q)
\]

where \(F^{-1}\) denotes an inverse cumulative distribution function. Integrating out \(X, Y \mid Z = z\) is a mixture of two normal random variables, with PDF and CDF defined as

\[
f(y \mid Z = z) = w_z \phi(y, \tau z, (\sigma + 1)^2) + (1 - w_z) \phi(y, \tau z, \sigma^2),
\]
\[
F(y \mid Z = z) = w_z \Phi(y, \tau z, (\sigma + 1)^2) + (1 - w_z) \Phi(y, \tau z, \sigma^2)
\]

where \(w_z = \mathbb{P}(X = 1 \mid Z = z)\). By contrast, the PDF and CDF of \(Y^z\) are given by

\[
f(y^z \mid Z = z) = \frac{1}{2} \phi(y, \tau z, (\sigma + 1)^2) + \frac{1}{2} \phi(y, \tau z, \sigma^2),
\]
\[
F(Y^z \mid Z = z) = \frac{1}{2} \Phi(y, \tau z, (\sigma + 1)^2) + \frac{1}{2} \Phi(y, \tau z, \sigma^2).
\]

Because \(X \not\perp Z\), \(w_z \neq 1/2\) and therefore

\[
F_{Y^1}(q) - F_{Y^0}(q) \neq F_{Y|Z=1}(q) - F_{Y|Z=0}(q),
\]

as illustrated in Figure 14.

\[Fig 13. \text{Mean causal graph}\]

### 4.10 Partial randomization

Some estimands require weaker assumptions than estimating the average treatment effect over the whole population does. For example, the **average treatment effect among the treated**, or ATT, is defined as \(\mathbb{E}(Y^1 - Y^0 \mid Z = 1) = \mathbb{E}(Y^1 \mid Z = 1) - \mathbb{E}(Y^0 \mid Z = 1)\). This estimand is important in the program evaluation literature, see for example Heckman (1996) and Heckman, Ichimura and Todd (1997).

\(^1\)In our experience, this potential outcomes notation for the ATT can give students fits, particularly the \(\mathbb{E}(Y^0 \mid Z = 1)\) term. Such students may find the structural equation notation to be somewhat more transparent: \(\mathbb{E}(\tau(X) \mid Z = 1)\) makes it clear that the probabilistic impact of conditioning on \(Z = 1\) is to modify the distribution over \(X\) defining the expectation; there is no opportunity for cognitive interference from the fact that the “\(z\)” in \(Y^z\) is different from that in the condition \(Z = z\).
An illustration of a confounded quantile treatment effect with unconfounded ATE. The top two panels depict the density and CDF functions of the DGP from section 4.9 for the four combinations of $X \in \{0, 1\}$ and $Z \in \{0, 1\}$. For each value of $X$ the change in the quantile is a constant shift to the right. The second row shows the densities of the potential outcome distributions and the conditional distribution of $Y \mid Z$, respectively, with $X$ integrated out. In both cases, the resulting density is a mixture of two normals with different variances and a common mean. However, the potential outcomes densities are just translations of the same mixture density, whereas the conditional distribution of $Y \mid Z$ also differs in terms of the mixture weights. The bottom row depicts the same relationship, but in terms of the CDFs. Attempts to estimate the quantile treatment effect — shown here as the distance between the black and grey curves at the horizontal dashed line in left panel — using the analogous distance from the right panel would misestimate the effect.
Here we use structural model notation to compare the ATT to the ATE, as relates to the “naive” contrast that compares the average response among treated individuals to the average response among the untreated individuals. In terms of the population, the naive contrast estimates \( E(Y \mid Z = 1) - E(Y \mid Z = 0) \). In terms of the structural model, this is equivalent to
\[
E(\mu(X) + \tau(X) \mid Z = 1) - E(\mu(X) \mid Z = 0).
\]
By definition, the exogenous errors are mean zero and vanish from the above expression. Now, randomization of \( Z \) implies that \( (\mu(X), \tau(X)) \perp \perp Z \), which in turn implies that \( E(\mu(X) \mid Z = 1) = E(\mu(X) \mid Z = 0) \) and therefore that
\[
E(\mu(X) + \tau(X) \mid Z = 1) - E(\mu(X) \mid Z = 0) = E(\tau(X) \mid Z = 1),
\]
the ATT. Randomization further implies that \( E(\tau(X) \mid Z = 1) = E(\tau(X)) \), so that the ATE and the ATT are the same.

However, the above derivation also reveals that to estimate the ATT one only needs \( E(\mu(X) \mid Z = 1) = E(\mu(X) \mid Z = 0) \), or what we might call mean prognostic unconfoundedness, which itself follows from \( \mu(X) \perp \perp Z \), or prognostic unconfoundedness. Thus, when the ATT is the sole interest, one only needs to rule out prognostic confounding. Meanwhile, treatment effect confounding, \( \tau(X) \not\perp \not\perp Z \), entails that the ATT and ATE are different, so that the ATE remains unknown even with the ATT in hand.

Note that a similar argument works for \( E(\tau(X) \mid Z = 0) \), the average effect of the treatment on the control (untreated) population, or ATC. This is easiest to see by reparametrizing the structural model in terms of: \( Z^* = 1 - Z \), \( \mu^*(X) = \mu(X) + \tau(X) \), and \( \tau^*(X) = -\tau(X) \). It then follows that the ATC may be estimated from the naive contrast so long as \( \mu^*(X) \perp \perp Z \).

As it relates to feature selection, it is notable that a smaller feature set may allow estimating the ATT than would be required for estimating the ATE. The following DGP is a concrete example:

\[
X_1 \sim \text{Bernoulli}(1/2), \quad X_2 \sim \text{Bernoulli}(1/2),
\]
\[
Z \mid X_1, X_2 \sim \text{Bernoulli}(0.25 + 0.5X_2),
\]
\[
Y \mid X_1, X_2, Z \sim \mathcal{N}(X_1 + (1 + 2X_2)Z, \sigma^2).
\]
In this example, \( \tau(X) = \tau(X_2) = 1 + 2X_2, \mu(X) = \mu(X_1) = X_1 \), and the ATE is \( E(\tau(X)) = 1 + 2E(X_2) = 2 \). The ATT, on the other hand, is \( E(\tau(X) \mid Z = 1) = 1 + 2E(X_2 \mid Z = 1) = 3/2 \). It is a nice simulation exercise to demonstrate that the naive contrast is consistent for the ATT, but not the ATE.

### 4.11 A two-stage estimator using two distinct control features.

This example builds upon the ideas presented in the previous one, but returns to the goal of regression adjustments for the ATE.

Suppose we know that \( \mu(X) \perp \perp Z \mid s_1(X) \) and \( \tau(X) \perp \perp Z \mid s_2(X) \), for distinct functions (features) \( s_1 \) and \( s_2 \). One approach to estimating the ATE under this assumption would be to stratify on the common refinement of \( s_1(X) \) and \( s_2(X) \), thus guaranteeing that \( (\mu(X), \tau(X)) \perp \perp Z \mid s(X) = s_1(X) \lor s_2(X) \). But an alternative two-stage approach is possible, which requires estimating fewer individual strata means. The procedure is:

1. Estimate \( E(\mu(s_1(X)) = E(Y \mid Z = 0, s_1(X)) \) from the control data.
2. Define \( R = Y - \mu(s_1(X)) \).
3. Estimate \( E(R \mid Z = 1, s_2(X)) \) from the treated data.
4. Compute the ATE as \( E_X(\mathbb{E}(R \mid Z = 1, s_2(X))) \), where the outer expectation is over \( X \), with respect to its marginal distribution.
We may verify the validity of this estimator by first expressing the procedure as the following iterated expectation:

\[
E_X (E(Y - E(Y | Z = 0, s_1(X)) | Z = 1, s_2(X)))
\]

\[
= E_X (E(Y | Z = 1, s_2(X))) - E_X (E(Y | Z = 0, s_1(X)))
\]

\[
= E_X (E(\mu(X) + \tau(X) | Z = 1, s_2(X))) - E_X (E(\mu(X) | Z = 0, s_1(X)))
\]

\[
= E_X (E(\tau(X) | Z = 1, s_2(X))) + E_X (E(\mu(X) | Z = 1, s_2(X))) - E_X (E(\mu(X) | Z = 0, s_1(X))).
\]

By the assumption that \( \mu(X) \perp \perp Z | s_1(X) \), we find that \( E(\mu(X) | Z = 0, s_1(X)) = E(\mu(X) | Z = 1, s_1(X)) \), which in turn implies that the second and third terms above are both equal to \( E_X(\mu(X) | Z = 1) \) (just expressed as distinct iterated expectations) and thus cancel. By the assumption that \( \tau(X) \perp \perp Z | s_2(X) \), the remaining term is equal to \( E_X(\tau(X) | s_2(X)) \) and the desired result follows after taking the outer expectation: \( E(\tau(X)) = E_X(E(\tau(X) | s_2(X))_.

5. DISCUSSION

To conclude, we synopsize our results and discuss further relationships to previous literature.

5.1 Famous results or debates revisited

The discrete covariate setting studied here allowed us to revisit several important existing results from a unique perspective.

Virtues of the propensity score. Rosenbaum and Rubin (1983) is often cited in support of propensity score methods for causal inference, but its results are often over-stated. First, there is not one propensity score, but many, one corresponding to each valid set of control features. Second, a propensity score need not be minimal; it is the minimal balancing score for the complete set of features used to create it, but balancing on those features is not necessary to estimate causal effects. Third, a propensity score method that disregards important prognostic features can be much less efficient than a method that does incorporate such features.

Estimated versus True propensity scores. In practice, the propensity score (corresponding to a given set of control features) is rarely known and so must be estimated. Hirano, Imbens and Ridder (2003) is sometimes cited to put a positive spin on this state of affairs: estimating a propensity function is better than knowing it exactly! But the actual situation is more nuanced. The asymptotic analysis of Hirano, Imbens and Ridder (2003) comparing the IPW estimator using true versus estimated propensity scores conceals the variety of specific ways the two estimators differ. Viewing the IPW as a stratification estimator in the discrete covariate setting puts these distinctions into immediate relief. One, the IPW using the true propensity scores uses different strata weights than the one using the estimated propensity scores, resulting in a higher variance estimator. Two, the IPW based on a true propensity score is able to collapse unnecessary strata, which can reduce the variance of the estimator. Three, collapsing unnecessary strata does not always reduce the variance, because the “extraneous” strata may be informative about unconfounded variation in the response. That is, an IPW estimator based on estimated propensity scores can have lower variance than one based on a true propensity score because it performs an implicit regression adjustment that is essentially unrelated to the propensity score. To be sure, the mathematics of Hirano, Imbens and Ridder (2003) are consistent with our analysis, and one can parse their expressions for such meaning, but their analysis does not expose the importance of either variable selection or prognostic stratification.

Regression adjustments for randomized experiments. Freedman (2008a) is sometimes cited as a reason to avoid regression adjustment for causal effect estimation altogether. However, Freedman’s result was more about model specification — or misspecification — than
it was about regression adjustment per se. Provided that one undertakes a nonparametric adjustment, as advocated by Lin (2013), Freedman’s main concerns are addressed. However, nonparametric adjustment poses its own challenges, in the form of high-variance estimators. Whether or not the inclusion of strong prognostic features is enough to offset the increased variability that comes with estimating a nonparametric model with limited data is impossible to say in any generality. Theorem 2 approaches this question quantitatively.

The peril of colliders. Greenland, Pearl and Robins (1999) introduce the “M-Graph” and the problem of conditioning on unblocked colliders. The issue was vigorously debated in a series of articles and replies in *Statistics in Medicine* between 2007 and 2009. Rubin (2007) suggested that all available pre-treatment covariates should be included in the conditioning set of any observational causal analysis, while others (Shrier (2008); Sjölander (2009); Pearl (2009b)) contended that such a strategy could incur collider bias. Rubin (2009) responded that unblocked colliders are a stylized problem that has few practical ramifications. This exchange in turn motivated further research, including Ding and Miratrix (2015), Rohde (2019), and Cinelli, Forney and Pearl (2020). Here, we observed that should colliders appear in a set of control variables — along with the associated blocking variables — regularization can unintentionally induce collider bias, revealing that colliders are not only a problem when their parents are unobserved. In particular, regularized regression approaches will struggle with colliders that are blocked by only a propensity-side ancestor. Additionally, Section 4.8 demonstrated that composite features that combine non-collider variables can “feature engineer” a pseudo-collider; how likely this is to occur in practice for particular supervised learning algorithms is an interesting open question.

Conditional unconfoundedness versus mean conditional unconfoundedness. In a discussion of Angrist, Imbens and Rubin (1996), Heckman (Heckman, 1996) makes a point similar to the one we make in section 4.9, that conditional unconfoundedness is stronger than necessary for estimating certain treatment effects. Angrist rejoins that identification based on “functional form” is undesirable. Here, we have taken the perspective of Heckman, as mean conditional unconfoundedness is the key notion for defining the principal deconfounding function, so it is perhaps worthwhile to unpack why. Our interest was in understanding the conditions according to which a particular set of control variables would yield a valid stratification estimator. From this perspective, a more specific assumption is weaker than a more general one: Conditional unconfoundedness implies mean conditional unconfoundedness, but not the other way around. It is the specificity of the *estimand* that permits the weaker (more general) assumption on the DGP. As we explored in section 4.9, mean conditional unconfoundedness does not permit estimation of quantile treatment effects. In order for mean conditional unconfoundedness to license estimation of quantile treatment effects, one would need to impose additional restrictions on the DGP, such as a fixed distributional shape around the unconfounded mean. But that is not our suggestion (nor do we believe it was Heckman’s).

Interestingly, this distinction between conditional unconfoundedness and mean conditional unconfoundedness is at the heart of the difference between general causal diagrams and more traditional path analysis. By focusing on correlations, the path diagram must only respect the mean causal relationships. Sometimes this is described by saying that path analysis “has a structural model, but no measurement model” (Wikipedia).

Additionally, a number of elementary, but easily-overlooked, facts were clarified: regression, propensity score weighting (and, *a fortiori*, double robust estimators based thereon) are identical in the case of discrete covariates (cf. lemma 3); CDAGs are non-unique (cf. Section 4.7), and instrumental and prognostic variable designations are inherently contingent (cf. Section 4.6).

5.2 Methodological ecumenicalism

In section 2.4, it was shown that the potential outcomes, CDAG, and exogenous errors definitions of conditional unconfoundedness are substantively equivalent. This result allows
us to conveniently move between the conventions of these alternative frameworks, which implicitly emphasize distinct aspects of the problem they all address — estimating treatment effects from data.

For example, the causal graph approach reminds us that sets of valid control variables are not unique and, consequently, we must not speak of the propensity score, but rather a propensity score and, perhaps, many candidate propensity scores (cf. section 4.2). This observation is fundamental to understanding how regularization will impact bias due to feature selection on graphs including colliders and instruments.

The potential outcomes approach reminds us that the exogenous errors need not be common among the treatment arms (cf. figure 1). More generally, because the potential outcome notation is intrinsically individualized, it emphasizes the idea that some individuals in a population may have distinct causal diagrams; in particular, some arrows may not appear in every individual’s graph. This is not at odds with the graphical formalism; rather it emerges simply because the graph alone does not fully determine the data generating process. In this paper, this distinction is not particularly important, but in estimation techniques relying on instrumental variables, it becomes critical (Angrist, Imbens and Rubin, 1996).

From the exogeneous errors approach, we are reminded that full conditional unconfoundedness is not actually necessary to estimate particular causal effects (cf. section 4.9); we leverage this result in defining the principal deconfounding function.

Synthesizing the three methods also clarifies common misunderstandings that can occur when operating solely within a single framework; for example, a mean regression model with exogeneous additive errors need not be structural (e.g., causal) in all of its arguments — rather, the exogeneity of the errors narrowly licenses a causal interpretation in the treatment variable (cf. section 4.6).

5.3 On discrete covariates with finite support

The approach in this paper has been to consider stratification estimators in the case of discrete control variables with finite support. Discrete covariates are both common in practice (indeed, more common than continuous covariates) and pedagogically illuminating, and therefore worthy of careful study. We are aware that not everyone agrees; we read in the textbook of Imbens and Rubin (Section 12.2.2):

If...we view the covariates as having a discrete distribution with finite support, the implication of unconfoundedness is simply that one should stratify by the values of the covariates. In that case there will be, with high probability, in sufficiently large samples, both treated and control units with the exact same values of the covariates. In this way we can immediately remove all biases arising from differences between covariates, and many adjustment methods will give similar, or even identical, answers.

However, as we stated before, this case rarely occurs in practice. In many applications it is not feasible to stratify fully on all covariates, because too many strata would have only a single unit.

The differences between various adjustment methods arise precisely in such settings where it is not feasible to stratify on all values of the covariates, and mathematically these differences are most easily analyzed in settings with random samples from large populations using effectively continuous distributions for the covariates...[Therefore] for the purpose of discussing various frequentist approaches to estimation and inference under unconfoundedness...it is helpful to view the covariates as having been randomly drawn from an approximately continuous distribution.

To paraphrase, the two main premises of this quote are: a) confounding — and, more specifically, deconfounding — is relatively easy to understand in the case of discrete covariates with finite support, and b) complete stratification is infeasible in many applications. We agree with these statements. But the conclusion — that the stylized setting of continuous covariates is therefore better suited to studying statistical methods for causal inference — does not necessarily follow. Indeed, we employ a different stylized mathematical assumption —
that each strata has at least one treated-control contrast — and find that, even in that case, bias variance trade-offs emerge. More importantly, these trade-offs can be studied directly, without resorting to asymptotic arguments, which may be untrustworthy guides to a method’s operating characteristics in practice. For example, Hahn (2004) concludes that foreknowledge of which variables are instruments is asymptotically irrelevant for regression estimators of average treatment effects. As we have seen in Section 4 of this paper, being able to distinguish instruments from confounders is certainly relevant for finite-sample performance.

5.4 Relationship to semi-supervised learning

This paper considers the problem of feature selection for causal effect estimation when a propensity function is available, but a causal diagram is not. This assumption is of course implausible in many practical scenarios, although there are cases where it may be approximately true. For example, suppose that a researcher has a dataset with \( n \) complete observations of \((X, Z, Y)\) and \( m \) “partially observed” samples, where \( m \gg n \). Partial samples of \((X, Z)\) pairs could be used to more accurately estimate \( \pi(X) \), bringing their applied problem closer to the setting studied above. Similarly, partial samples on \((X, Z = 0, Y)\) could be used to better estimate \( \mu(X) \), which is particularly useful in the situation described in Section 4.11. Such scenarios may be plausible in electronic health records, for instance, in which a treatment (say, a new blood pressure medicine) is rarely administered but an outcome (say, blood pressure) is very commonly measured.

The idea of using large auxiliary datasets is common in machine learning, where it is known as semi-supervised learning (Zhu and Goldberg, 2009; Belkin, Niyogi and Sindhwani, 2006; Liang, Mukherjee and West, 2007). Using unlabeled data to estimate a propensity function in conjunction with machine learning or other regularization methods represent an exciting application of semi-supervised learning to the problem of causal effect estimation. While it is often easier to formalize and motivate the use of auxiliary data for prediction, rather than estimation, this paper shows that there is a role for function estimation techniques in machine learning causal inference.
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APPENDIX A: PROOF OF THEOREM 2

We consider a sample of \( n \) i.i.d. observations of \( (X, Y, Z) \) from the data generating process in Equation 12. We assume that there exists a function \( \lambda \) defined on \( \mathcal{X} \) such that the ATE is identified conditional on \( \lambda(X) \). We assume that \( |\lambda(X)| = J < K = |\mathcal{X}| \) so that the unique values of \( \lambda(X) \) define a non-trivial “coarsening” of \( X \). Consider a function \( s(X) \) such that there exists at least one pair \( x, x' \in \mathcal{X} \) such that \( s(x) \neq s(x') \) while \( \lambda(x) = \lambda(x') \). We assume that \( s(X) \) also identifies the ATE, so that conditioning on \( s(X) \) does not induce collider bias.

For each \( j \in \lambda(\mathcal{X}) \), there exist \( m(j) \geq 1 \) unique values of \( s(X) \), which we denote as \( \{j_1, \ldots, j_m\} \). By the definition of \( s(X) \), there exists at least one \( j \in \lambda(\mathcal{X}) \) such that \( m(j) > 1 \).

We define two stratification estimators of the ATE as follows:

\[
\begin{align*}
\tilde{\tau}^{\lambda}_{\text{strat}} &= \frac{1}{n} \sum_{j \in \lambda(\mathcal{X})} N_j \left( \bar{Y}_{j,1} - \bar{Y}_{j,0} \right) \\
\tilde{\tau}^s_{\text{strat}} &= \frac{1}{n} \sum_{j \in \lambda(\mathcal{X})} \left( \sum_{\ell=1}^{m(j)} \frac{N_{j,\ell}}{n} \left( \bar{Y}_{j,\ell,1} - \bar{Y}_{j,\ell,0} \right) \right)
\end{align*}
\]

\[
\begin{align*}
N_j &= \sum_{i=1}^{n} \{ \lambda(X_i) = j \} \\
N_{j,1} &= \sum_{i=1}^{n} \{ \lambda(X_i) = j \} \{ Z_i = 1 \} \\
N_{j,0} &= \sum_{i=1}^{n} \{ \lambda(X_i) = j \} \{ Z_i = 0 \} \\
\bar{Y}_{j,1} &= \frac{1}{N_{j,1}} \sum_{i=1}^{n} Y_i \{ \lambda(X_i) = j \} \{ Z_i = 1 \} \\
\bar{Y}_{j,0} &= \frac{1}{N_{j,0}} \sum_{i=1}^{n} Y_i \{ \lambda(X_i) = j \} \{ Z_i = 0 \}
\end{align*}
\]

Now, we consider a \( j \in \lambda(\mathcal{X}) \) with \( m(j) > 1 \). We introduce some notation.

\[
\begin{align*}
\mu_{j,1} &= \mathbb{E}(Y \mid \lambda(X) = j, Z = 1) \\
\mu_{j,0} &= \mathbb{E}(Y \mid s(X) = j, Z = 1) \\
\mu_{j,1,1} &= \mathbb{E}(Y \mid s(X) = j, Z = 1) \\
\mu_{j,0,0} &= \mathbb{E}(Y \mid s(X) = j, Z = 0) \\
\sigma_{j,1}^2 &= \mathbb{V}(Y \mid \lambda(X) = j, Z = 1) \\
\sigma_{j,0}^2 &= \mathbb{V}(Y \mid s(X) = j, Z = 1) \\
\sigma_{j,1,1}^2 &= \mathbb{V}(Y \mid s(X) = j, Z = 1)
\end{align*}
\]

By the law of iterated expectations and the law of total variance, it follows that

\[
\begin{align*}
\mu_{j,1} &= \mathbb{E}(\mathbb{E}(Y \mid s(X) = j, Z = 1) \mid \lambda(X) = j, Z = 1) = \mathbb{E}(\mu_{j,1,1} \mid \lambda(X) = j, Z = 1) \\
\sigma_{j,1}^2 &= \mathbb{E}(\mathbb{V}(Y \mid s(X) = j, Z = 1) \mid \lambda(X) = j, Z = 1) + \mathbb{V}(\mathbb{E}(Y \mid s(X) = j, Z = 1) \mid \lambda(X) = j, Z = 1) \\
\mu_{j,0} &= \mathbb{E}(\mu_{j,0,0} \mid \lambda(X) = j, Z = 1) \\
\sigma_{j,0}^2 &= \mathbb{E}(\sigma_{j,0}^2 \mid \lambda(X) = j, Z = 1)
\end{align*}
\]

We denote

\[
\begin{align*}
\tilde{\mu}_{j,1} &= \mathbb{E}(\mu_{j,1,1} \mid \lambda(X) = j, Z = 1) = \mu_{j,1} \\
\tilde{\sigma}_{j,1}^2 &= \mathbb{E}(\sigma_{j,1}^2 \mid \lambda(X) = j, Z = 1) \\
\nu(\mu_{j,1,1}) &= \mathbb{V}(\mu_{j,1,1} \mid \lambda(X) = j, Z = 1)
\end{align*}
\]
Conditioning on $N = \{N_{j,1}, \ldots, N_{j,m}, N_{j,0}, \ldots, N_{j,m,0}\}$, we see

$$
V \left( \sum_{\ell=1}^{m(j)} N_{j\ell} \bar{Y}_{j\ell,1} \mid N \right) = \sum_{\ell=1}^{m(j)} N_{j\ell}^2 V \left( \bar{Y}_{j\ell,1} \right) = \sum_{\ell=1}^{m(j)} N_{j\ell}^2 \frac{\sigma_{j,1}^2}{N_{j\ell,1}} = \sum_{\ell=1}^{m(j)} \frac{(N_{j\ell,1} + N_{j\ell,0})^2}{N_{j\ell,1}} \frac{\sigma_{j,1}^2}{N_{j\ell,1}}
$$

$$
V \left( N_{j} \bar{Y}_{j,1} \mid N \right) = N_{j}^2 V \left( \bar{Y}_{j,1} \right) = N_{j}^2 \frac{\sigma_{j,1}^2}{N_{j,1}} = \frac{(N_{j,1} + N_{j,0})^2}{N_{j,1}} \frac{\sigma_{j,1}^2}{N_{j,1}} = \frac{(\sum_{\ell=1}^{m(j)} (N_{j\ell,1} + N_{j\ell,0}))^2}{\sum_{\ell=1}^{m(j)} N_{j\ell,1}} \frac{\sigma_{j,1}^2}{N_{j,1}} + \nu (\mu_{j,1})
$$

and

$$
E \left( \sum_{\ell=1}^{m(j)} N_{j\ell} \bar{Y}_{j\ell,1} \mid N \right) = \sum_{\ell=1}^{m(j)} N_{j\ell} E \left( \bar{Y}_{j\ell,1} \mid N \right) = \sum_{\ell=1}^{m(j)} N_{j\ell} E \left( Y \mid s(X) = j\ell, Z = 1 \right) = \sum_{\ell=1}^{m(j)} N_{j\ell} \mu_{j\ell,1}
$$

$$
E \left( N_{j} \bar{Y}_{j,1} \mid N \right) = N_{j} E \left( \bar{Y}_{j,1} \mid N \right) = N_{j} E \left( Y \mid \lambda(X) = j, Z = 1 \right) = N_{j} \mu_{j,1} = \left( \sum_{\ell=1}^{m(j)} N_{j\ell,1} \right) \mu_{j,1}
$$

Thus, we have that

$$
V \left( \sum_{\ell=1}^{m(j)} N_{j\ell} \bar{Y}_{j\ell,1} \right) = E \left( V \left( \sum_{\ell=1}^{m(j)} N_{j\ell} \bar{Y}_{j\ell,1} \mid N \right) \right) + V \left( E \left( \sum_{\ell=1}^{m(j)} N_{j\ell} \bar{Y}_{j\ell,1} \mid N \right) \right)
$$

$$
= E \left( \sum_{\ell=1}^{m(j)} \frac{(N_{j\ell,1} + N_{j\ell,0})^2}{N_{j\ell,1}} \frac{\sigma_{j,1}^2}{N_{j\ell,1}} \right) + V \left( \sum_{\ell=1}^{m(j)} N_{j\ell} \mu_{j\ell,1} \right)
$$

$$
V \left( N_{j} \bar{Y}_{j,1} \right) = E \left( V \left( N_{j} \bar{Y}_{j,1} \mid N \right) \right) + V \left( E \left( N_{j} \bar{Y}_{j,1} \mid N \right) \right)
$$

$$
= E \left( \frac{(\sum_{\ell=1}^{m(j)} (N_{j\ell,1} + N_{j\ell,0}))^2}{\sum_{\ell=1}^{m(j)} N_{j\ell,1}} \frac{\sigma_{j,1}^2}{N_{j,1}} + \nu (\mu_{j,1}) \right) + V \left( \left( \sum_{\ell=1}^{m(j)} N_{j\ell,1} \right) \bar{\mu}_{j,1} \right)
$$

We can broaden this to entire set of observations:

$$
V \left( \bar{z}_{strat}^s \right) = V \left( \sum_{j=1}^{m(j)} \sum_{\ell=1}^{m(j)} \frac{N_{j\ell}}{n} (\bar{Y}_{j\ell,1} - \bar{Y}_{j,0}) \right)
$$

$$
= \frac{1}{n^2} \left[ E \left( \sum_{j=1}^{m(j)} \sum_{\ell=1}^{m(j)} (N_{j\ell,1} + N_{j\ell,0})^2 \left( \frac{\sigma_{j,1}^2}{N_{j\ell,1}} + \frac{\sigma_{j,0}^2}{N_{j\ell,0}} \right) \right) + V \left( \sum_{j=1}^{m(j)} \sum_{\ell=1}^{m(j)} N_{j\ell} (\mu_{j,1} - \mu_{j,0}) \right) \right]
$$

$$
V \left( \bar{z}_{strat}^\lambda \right) = V \left( \sum_{j=1}^{m(j)} \frac{N_{j}}{n} (\bar{Y}_{j,1} - \bar{Y}_{j,0}) \right)
$$

$$
= \frac{1}{n^2} E \left( \sum_{j=1}^{m(j)} \left( \sum_{\ell=1}^{m(j)} (N_{j\ell,1} + N_{j\ell,0})^2 \left( \frac{\sigma_{j,1}^2}{N_{j\ell,1}} + \frac{\sigma_{j,0}^2}{N_{j\ell,0}} \right) \right) + V \left( \sum_{j=1}^{m(j)} \sum_{\ell=1}^{m(j)} N_{j\ell} (\mu_{j,1} - \mu_{j,0}) \right) \right)
$$

$$
+ \frac{1}{n^2} V \left( \sum_{j=1}^{m(j)} \left( \sum_{\ell=1}^{m(j)} N_{j\ell} (\bar{\mu}_{j,1} - \bar{\mu}_{j,0}) \right) \right)
$$
A.1 Case I: equal sub-strata means and variances

If \( \sigma^2_{j,\ell} = \bar{\sigma}^2_{j,\ell} \) and \( \mu_{j,\ell,i} = \bar{\mu}_{j,\ell,i} \) for all \( \ell, i \in \{1, \ldots, m(j)\} \times \{0, 1\} \), then \( v(\mu_{j,\ell,i}) = 0 \) and the variance and expectation terms factor out of both expressions and we are left to compare the nonlinear sums of the strata cell sizes. Focusing on \( \lambda(X) = j \) and \( Z = 1 \), we show by induction that

\[
\sum_{\ell=1}^{m(j)} \frac{(N_{j,\ell,1} + N_{j,\ell,0})}{N_{j,\ell,1}} \geq \frac{\left(\sum_{\ell=1}^{m(j)} (N_{j,\ell,1} + N_{j,\ell,0})\right)^2}{\sum_{\ell=1}^{m(j)} N_{j,\ell,1}}
\]

for positive cell sizes \( N_{j,\ell,1} \).

For the base case, suppose that \( m(j) = 2 \) so that

\[
\sum_{\ell=1}^{m(j)} \frac{(N_{j,\ell,1} + N_{j,\ell,0})^2}{N_{j,\ell,1}} = \frac{(N_{j,1,1} + N_{j,1,0})^2}{N_{j,1,1}} + \frac{(N_{j,2,1} + N_{j,2,0})^2}{N_{j,2,1}}
\]

and we show by

\[
\sum_{\ell=1}^{m(j)} \frac{(N_{j,\ell,1} + N_{j,\ell,0})^2}{N_{j,\ell,1}} \leq \frac{\left(\sum_{\ell=1}^{m(j)} (N_{j,\ell,1} + N_{j,\ell,0})\right)^2}{\sum_{\ell=1}^{m(j)} N_{j,\ell,1}}
\]

For ease of exposition, we let

\[
a = N_{j,1,1} \quad b = N_{j,1,0} \\
c = N_{j,2,1} \quad d = N_{j,2,0}
\]

and we thus compare \( \frac{(a+b)^2}{a} + \frac{(c+d)^2}{c} \) with \( \frac{(a+b+c+d)^2}{a+c} \) where \( a, b, c, d > 0 \)

\[
0 \leq [c(a+b) - a(c+d)]^2 \\
0 \leq c^2(a+b)^2 + a^2(c+d)^2 - 2ac(a+b)(c+d)
\]

\[
2ac(a+b)(c+d) \leq c^2(a+b)^2 + a^2(c+d)^2
\]

\[
ac(a+b)^2 + 2ac(a+b)(c+d) + ac(c+d)^2 \leq ac(a+b)^2 + c^2(a+b)^2 + a^2(c+d)^2 + ac(c+d)^2
\]

\[
ac[(a+b)+(c+d)]^2 \leq [c(a+b)^2 + a(c+d)^2] (a+c)
\]

\[
\frac{[(a+b)+(c+d)]^2}{a+c} \leq \frac{(a+b)^2}{a} + \frac{(c+d)^2}{c}
\]

Now, we proceed to the induction case. Assume that

\[
\sum_{\ell=1}^{m(j)} \frac{(N_{j,\ell,1} + N_{j,\ell,0})^2}{N_{j,\ell,1}} \geq \frac{\left(\sum_{\ell=1}^{m(j)} (N_{j,\ell,1} + N_{j,\ell,0})\right)^2}{\sum_{\ell=1}^{m(j)} N_{j,\ell,1}}
\]

We consider a new stratum, indexed \( m(j) + 1 \) and we see that

\[
\sum_{\ell=1}^{m(j) + 1} \frac{(N_{j,\ell,1} + N_{j,\ell,0})^2}{N_{j,\ell,1}} \geq \frac{\left(\sum_{\ell=1}^{m(j)+1} (N_{j,\ell,1} + N_{j,\ell,0})\right)^2}{\sum_{\ell=1}^{m(j)+1} N_{j,\ell,1}}
\]

Letting

\[
a = \sum_{\ell=1}^{m(j)} N_{j,\ell,1} \quad b = \sum_{\ell=1}^{m(j)} N_{j,\ell,0} \\
c = N_{m(j)+1,1} \quad d = N_{m(j)+1,0}
\]

we see that

\[
\frac{\left(\sum_{\ell=1}^{m(j)} (N_{j,\ell,1} + N_{j,\ell,0})\right)^2}{\sum_{\ell=1}^{m(j)} N_{j,\ell,1}} + \frac{(N_{m(j)+1,1} + N_{m(j)+1,0})^2}{N_{m(j)+1,1}} \geq \frac{(a+b)^2}{a} + \frac{(c+d)^2}{c}
\]

\[
= \frac{\left(\sum_{\ell=1}^{m(j)+1} (N_{j,\ell,1} + N_{j,\ell,0})\right)^2}{\sum_{\ell=1}^{m(j)+1} N_{j,\ell,1}}
\]
and the relationship follows by induction.

Thus, when \( \mathbb{E}(Y \mid s(X) = j \ell, Z = 1) \) and \( \mathbb{V}(Y \mid s(X) = j \ell, Z = 1) \) are constant for all \( j \in \lambda(X) \) and \( \ell \in \{1, \ldots, m(j)\} \), it follows that \( \mathbb{V}(\hat{\tau}_{\text{strat}}^\lambda) \leq \mathbb{V}(\hat{\tau}_{\text{strat}}^s) \).

We let

\[
\alpha_1 = \frac{1}{n^2} \mathbb{E} \left( \sum_{j \in \lambda(X)} \bar{\sigma}^2_{j,1} \left( \frac{\sum_{\ell=1}^{m(j)} (N_{j,\ell,1} + N_{j,\ell,0})^2}{N_{j,\ell,1}} \right) - \left( \frac{\sum_{\ell=1}^{m(j)} (N_{j,\ell,1} + N_{j,\ell,0})^2}{\sum_{\ell=1}^{m(j)} N_{j,\ell,1}} \right)^2 \right) \\
\alpha_0 = \frac{1}{n^2} \mathbb{E} \left( \sum_{j \in \lambda(X)} \bar{\sigma}^2_{j,0} \left( \frac{\sum_{\ell=1}^{m(j)} (N_{j,\ell,1} + N_{j,\ell,0})^2}{N_{j,\ell,0}} \right) - \left( \frac{\sum_{\ell=1}^{m(j)} (N_{j,\ell,1} + N_{j,\ell,0})^2}{\sum_{\ell=1}^{m(j)} N_{j,\ell,0}} \right)^2 \right)
\]

so that \( \alpha = \alpha_1 + \alpha_0 \) is the degree to which \( \mathbb{V}(\hat{\tau}_{\text{strat}}^\lambda) \leq \mathbb{V}(\hat{\tau}_{\text{strat}}^s) \) when all substrata of \( s(X) \) are constant. We see that this depends on \( \bar{\sigma}^2_{j,1}, \bar{\sigma}^2_{j,0} \), and the distribution of \( N_{j,\ell,i} \) for each \( j \) and \( i \).

**A.2 Case II: unequal strata means or variances**

We partition \( \lambda(X) \) into three sets:

- \( A \): \( m(a) = 1 \) for all \( a \in A \)
- \( B \): for all \( b \in B \), either
  - \( m(b) > 1 \)
  - \( \sigma^2_{b,\ell,i} \) and \( \mu_{b,\ell,i} \) are constant for all \( \ell \in m(b), i \in \{0, 1\} \)
- \( C \): For all \( c \in C \)
  - \( m(c) > 1 \), and
  - \( \sigma^2_{c,\ell,i} \) or \( \mu_{c,\ell,i} \) is non-constant for some \( i \in \{0, 1\} \)

In the previous section \( C = \emptyset \), so that the variance comparison is uncomplicated: \( s(X) \) was “overstratified” relative to \( \lambda(X) \) and as a result \( \mathbb{V}(\hat{\tau}_{\text{strat}}^\lambda) \leq \mathbb{V}(\hat{\tau}_{\text{strat}}^s) \).

In this case, \( C \neq \emptyset \) so that there may be variance reduction to stratification (see Lohr (2019) for one reference). We define several terms

\[
\beta_1 = \frac{1}{n^2} \mathbb{E} \left( \sum_{b \in B} \bar{\sigma}^2_{b,1} \left( \frac{\sum_{\ell=1}^{m(b)} (N_{b,\ell,1} + N_{b,\ell,0})^2}{N_{b,\ell,1}} \right) - \left( \frac{\sum_{\ell=1}^{m(b)} (N_{b,\ell,1} + N_{b,\ell,0})^2}{\sum_{\ell=1}^{m(b)} N_{b,\ell,1}} \right)^2 \right) \\
\beta_0 = \frac{1}{n^2} \mathbb{E} \left( \sum_{b \in B} \bar{\sigma}^2_{b,0} \left( \frac{\sum_{\ell=1}^{m(b)} (N_{b,\ell,1} + N_{b,\ell,0})^2}{N_{b,\ell,0}} \right) - \left( \frac{\sum_{\ell=1}^{m(b)} (N_{b,\ell,1} + N_{b,\ell,0})^2}{\sum_{\ell=1}^{m(b)} N_{b,\ell,0}} \right)^2 \right)
\]

\[
c_1 = \frac{1}{n^2} \mathbb{E} \left( \sum_{c \in C} \left( \sum_{\ell=1}^{m(c)} (N_{c,\ell,1} + N_{c,\ell,0}) \right)^2 \left( \frac{\sigma^2_{c,\ell,1} + \mathbb{V}(\mu_{c,\ell,1})}{N_{c,\ell,1}} \right) \right) + \frac{1}{n^2} \mathbb{V} \left( \sum_{c \in C} \sum_{\ell=1}^{m(c)} N_{c,\ell} \left( \bar{\mu}_{c,1} \right) \right) \\
c_0 = \frac{1}{n^2} \mathbb{E} \left( \sum_{c \in C} \left( \sum_{\ell=1}^{m(c)} (N_{c,\ell,1} + N_{c,\ell,0}) \right)^2 \left( \frac{\sigma^2_{c,\ell,0} + \mathbb{V}(\mu_{c,\ell,0})}{N_{c,\ell,0}} \right) \right) + \frac{1}{n^2} \mathbb{V} \left( \sum_{c \in C} \sum_{\ell=1}^{m(c)} N_{c,\ell} \left( \bar{\mu}_{c,0} \right) \right) \\
c_2 = \frac{1}{n^2} \mathbb{E} \left( \sum_{c \in C} \sum_{\ell=1}^{m(c)} (N_{c,\ell,1} + N_{c,\ell,0})^2 \left( \frac{\sigma^2_{c,\ell,1}}{N_{c,\ell,1}} + \frac{\sigma^2_{c,\ell,0}}{N_{c,\ell,0}} \right) \right) + \mathbb{V} \left( \sum_{c \in C} \sum_{\ell=1}^{m(c)} N_{c,\ell} \left( \mu_{c,1} - \mu_{c,0} \right) \right)
\]

\[ \eta = c_1 + c_0 - c_2 \]

\[ \nu = \beta_1 + \beta_0 \]
We see that $\nabla (\hat{\tau}_{strat}^\lambda) > \nabla (\hat{\tau}_{strat}^s)$ if $\eta > \nu$, where $\eta$ refers to the reduction in variance by stratifying on $s(X)$ within $C$ and $\nu$ refers to the increase in variance by “over-stratifying” on $B$.

APPENDIX B: PROOF OF THEOREM 3

We consider a sample of $n$ i.i.d. observations of $(X, Y, Z)$ from the data generating process in Equation 12. Let $\pi(x) = \mathbb{P}(Z = 1 | X = x)$ refer to the “true propensity function” and $\hat{\pi}(x) = N_{x,1}/N_x$ refer to the “empirical propensity function.”

We define two stratification estimators of the ATE as follows:

$$\hat{\tau}_{strat}^\pi = \sum_{x \in \mathcal{X}} \frac{N_x}{n} (\bar{Y}_x,1 - \bar{Y}_x,0) \quad \hat{\tau}_{strat}^\pi = \sum_{x \in \mathcal{X}} \frac{N_x}{n} \left( \frac{\hat{\pi}(x)}{\pi(x)} \bar{Y}_x,1 - \frac{1 - \hat{\pi}(x)}{1 - \pi(x)} \bar{Y}_x,0 \right)$$

where $N_x, N_{x,1}, N_{x,0}, \bar{Y}_x,1$, and $\bar{Y}_x,0$ are defined similar to Appendix A:

$$N_x = \sum_{i=1}^{n} 1(X_i = x)$$

$$N_{x,1} = \sum_{i=1}^{n} 1(X_i = x) 1(Z_i = 1)$$

$$N_{x,0} = \sum_{i=1}^{n} 1(X_i = x) 1(Z_i = 0)$$

$$\bar{Y}_x,1 = \frac{1}{N_{x,1}} \sum_{i=1}^{n} 1Y_i(X_i = x) 1(Z_i = 1)$$

$$\bar{Y}_x,0 = \frac{1}{N_{x,0}} \sum_{i=1}^{n} 1Y_i(X_i = x) 1(Z_i = 0)$$

Now, we consider an arbitrary $x \in \mathcal{X}$ with $N_{x,1} > 0$ and $N_{x,0} > 0$. We introduce some notation.

$$\mu_{x,1} = \mathbb{E}(Y | X = x, Z = 1)$$

$$\mu_{x,0} = \mathbb{E}(Y | X = x, Z = 0)$$

$$\sigma_{x,1}^2 = \nabla(Y | X = x, Z = 1)$$

$$\sigma_{x,0}^2 = \nabla(Y | X = x, Z = 0)$$

Conditioning on $N = \{N_{x,1}, N_{x,0} : x \in \mathcal{X}\}$, we see

$$\nabla \left( \sum_{x \in \mathcal{X}} N_x \bar{Y}_x,1 | N \right) = \sum_{x \in \mathcal{X}} N_x^2 \nabla (\bar{Y}_x,1) = \sum_{x \in \mathcal{X}} N_x^2 \sigma_{x,1}^2 N_{x,1} = \sum_{x \in \mathcal{X}} \frac{(N_{x,1} + N_{x,0})^2}{N_{x,1}} \sigma_{x,1}^2$$

$$\nabla \left( \sum_{x \in \mathcal{X}} \frac{\hat{\pi}(x)}{\pi(x)} \bar{Y}_x,1 | N \right) = \sum_{x \in \mathcal{X}} N_x^2 \left( \frac{\hat{\pi}(x)}{\pi(x)} \right)^2 \nabla (\bar{Y}_x,1) = \sum_{x \in \mathcal{X}} N_x^2 \left( \frac{\hat{\pi}(x)}{\pi(x)} \right)^2 \sigma_{x,1}^2 N_{x,1}$$

$$= \sum_{x \in \mathcal{X}} \left( \frac{\hat{\pi}(x)}{\pi(x)} \right)^2 \frac{(N_{x,1} + N_{x,0})^2}{N_{x,1}} \sigma_{x,1}^2$$

and
\[
\mathbb{E}\left(\sum_{x \in \mathcal{X}} N_x \bar{Y}_{x,1} \mid N\right) = \sum_{x \in \mathcal{X}} N_x \mathbb{E}\left(\bar{Y}_{x,1}\right) = \sum_{x \in \mathcal{X}} N_x \mathbb{E}\left(Y \mid X = x, Z = 1\right) = \sum_{x \in \mathcal{X}} N_x \mu_{x,1},
\]

\[
\mathbb{E}\left(\sum_{x \in \mathcal{X}} N_x \frac{\hat{\pi}(x)}{\pi(x)} \bar{Y}_{x,1} \mid N\right) = \sum_{x \in \mathcal{X}} N_x \frac{\hat{\pi}(x)}{\pi(x)} \mathbb{E}\left(\bar{Y}_{x,1}\right)
= \sum_{x \in \mathcal{X}} \frac{\hat{\pi}(x)}{\pi(x)} N_x \mathbb{E}\left(Y \mid X = x, Z = 1\right)
= \sum_{x \in \mathcal{X}} \frac{\hat{\pi}(x)}{\pi(x)} N_x \mu_{x,1}.
\]

Thus, we have that

\[
\mathbb{V}\left(\sum_{x \in \mathcal{X}} N_x \bar{Y}_{x,1}\right) = \mathbb{E}\left(\mathbb{V}\left(\sum_{x \in \mathcal{X}} N_x \bar{Y}_{x,1} \mid N\right)\right) + \mathbb{V}\left(\mathbb{E}\left(\sum_{x \in \mathcal{X}} N_x \bar{Y}_{x,1} \mid N\right)\right)
= \mathbb{E}\left(\sum_{x \in \mathcal{X}} \left(\frac{N_{x,1} + N_{x,0}}{N_x}\right)^2 \sigma_{x,1}^2\right) + \mathbb{V}\left(\sum_{x \in \mathcal{X}} N_x \mu_{x,1}\right)
\]

\[
\mathbb{V}\left(\sum_{x \in \mathcal{X}} N_x \frac{\hat{\pi}(x)}{\pi(x)} \bar{Y}_{x,1}\right) = \mathbb{E}\left(\mathbb{V}\left(\sum_{x \in \mathcal{X}} N_x \frac{\hat{\pi}(x)}{\pi(x)} \bar{Y}_{x,1} \mid N\right)\right) + \mathbb{V}\left(\mathbb{E}\left(\sum_{x \in \mathcal{X}} N_x \frac{\hat{\pi}(x)}{\pi(x)} \bar{Y}_{x,1} \mid N\right)\right)
= \mathbb{E}\left(\sum_{x \in \mathcal{X}} \left(\frac{\hat{\pi}(x)}{\pi(x)}\right)^2 \left(\frac{N_{x,1} + N_{x,0}}{N_x}\right)^2 \sigma_{x,1}^2\right) + \mathbb{V}\left(\sum_{x \in \mathcal{X}} \frac{\hat{\pi}(x)}{\pi(x)} N_x \mu_{x,1}\right)
\]

We can broaden this to entire set of observations:

\[
\mathbb{V}\left(\frac{\hat{\pi}}{\pi}_{\text{strat}}\right) = \mathbb{V}\left(\sum_{x \in \mathcal{X}} \frac{N_x}{n} \left(\bar{Y}_{x,1} - \bar{Y}_{x,0}\right)\right)
= \frac{1}{n^2} \left[ \mathbb{E}\left(\sum_{x \in \mathcal{X}} \left(\frac{N_{x,1} + N_{x,0}}{N_x}\right)^2 \left(\frac{\sigma_{x,1}^2}{N_{x,1}} + \frac{\sigma_{x,0}^2}{N_{x,0}}\right)\right) + \mathbb{V}\left(\sum_{x \in \mathcal{X}} N_x \left(\mu_{x,1} - \mu_{x,0}\right)\right)\right]
\]

\[
\mathbb{V}\left(\frac{\hat{\pi}}{\pi}_{\text{strat}}\right) = \mathbb{V}\left(\sum_{x \in \mathcal{X}} \frac{N_x}{n} \left(\frac{\hat{\pi}(x)}{\pi(x)} \bar{Y}_{x,1} - \frac{1 - \hat{\pi}(x)}{1 - \pi(x)} \bar{Y}_{x,0}\right)\right)
= \frac{1}{n^2} \mathbb{E}\left(\sum_{x \in \mathcal{X}} \left(\frac{N_{x,1} + N_{x,0}}{N_x}\right)^2 \left(\frac{(\hat{\pi}(x))^2 \sigma_{x,1}^2}{\pi(x)} + \frac{(1 - \hat{\pi}(x))^2 \sigma_{x,0}^2}{1 - \pi(x)}\right)\right)
+ \frac{1}{n^2} \mathbb{V}\left(\sum_{x \in \mathcal{X}} N_x \left(\frac{\hat{\pi}(x)}{\pi(x)} \mu_{x,1} - \frac{1 - \hat{\pi}(x)}{1 - \pi(x)} \mu_{x,0}\right)\right)
\]

\[
\mathbb{V}\left(\frac{\hat{\pi}}{\pi}_{\text{strat}}\right) = \mathbb{E}\left(\sum_{x \in \mathcal{X}} \frac{N_{x,1}}{(\pi(x))^2} \sigma_{x,1}^2 + \frac{N_{x,0}}{(1 - \pi(x))^2} \sigma_{x,0}^2\right)
+ \frac{1}{n^2} \mathbb{V}\left(\sum_{x \in \mathcal{X}} N_x \left(\frac{\hat{\pi}(x)}{\pi(x)} \mu_{x,1} - \frac{1 - \hat{\pi}(x)}{1 - \pi(x)} \mu_{x,0}\right)\right)
\]
B.1 Case I: $\mu(x) = \tau(x) = 0$

First, consider the degenerate case in which $\mu(x) = \tau(x) = 0$ for all $x \in \mathcal{X}$. In this case, the second term vanishes and we are left to compare $\mathbb{E}\left(\sum_{x \in \mathcal{X}} \left( \frac{N_{x,1}}{(\bar{\pi}(x))^2} \sigma_{x,1}^2 + \frac{N_{x,0}}{(1-\bar{\pi}(x))^2} \sigma_{x,0}^2 \right) \right)$ with $\mathbb{E}\left(\sum_{x \in \mathcal{X}} \frac{N_{x,1}}{\bar{\pi}(x)} \pi(x) \right)^2 + \frac{N_{x,0}}{(1-\bar{\pi}(x))} \pi(x) \right)^2 \sigma_{x,0}^2 \right)$. Since we have defined $\hat{\pi}$ empirically, we have that $\mathbb{E}(\hat{\pi}(x)) = \pi(x)$. We also have, by iterated expectations that $\mathbb{E}(N_{x,1}) = \mathbb{E}(\mathbb{E}(N_{x,1} | N_x)) = \mathbb{E}(N_x \pi(x)) = \pi(x) \mathbb{E}(N_x)$.

Now, define $g(x, y) = y^2 / x$, so that $g(N_{x,1}, N_x) = N_x^2 / N_{x,1} = N_{x,1} / \pi(x)^2$. $g(N_{x,1}, N_x)$ can be shown to be a convex function as its Hessian is positive semidefinite (Boyd and Vandenberghe (2004)). Thus, by Jensen’s inequality,

$$\mathbb{E}(g(N_{x,1}, N_x)) \geq g(\mathbb{E}(N_{x,1}, N_x))$$

$$\mathbb{E}\left(\frac{N_x^2}{N_{x,1}}\right) \geq g(\pi(x) N_x, N_x)$$

$$\mathbb{E}\left(\frac{N_{x,1}}{\pi(x)^2}\right) \geq \frac{N_x \pi(x)}{\pi(x)^2} = \frac{N_{x,1} \pi(x)}{\pi(x)^2} = \mathbb{E}\left(\frac{N_{x,1}}{\pi(x)^2}\right)$$

Intuitively, this result shows that when there is “nothing to estimate” in that $y$ is degenerate, the variance of the $\hat{\pi}_{strat}$ estimator is greater than that of the $\hat{\pi}_\pi$ estimator.

B.2 Case II: $|\mu(x)| + |\tau(x)| > \epsilon$ for some $x \in \mathcal{X}$ and for a data-dependent $\epsilon$

Setting aside this degenerate case, we first define

$$a = \mathbb{E}\left(\sum_{x \in \mathcal{X}} \left( \frac{N_{x,1}}{(\bar{\pi}(x))^2} \sigma_{x,1}^2 + \frac{N_{x,0}}{(1-\bar{\pi}(x))^2} \sigma_{x,0}^2 \right) \right) - \mathbb{E}\left(\sum_{x \in \mathcal{X}} \frac{N_{x,1}}{\bar{\pi}(x) \pi(x)} \pi(x) \right)^2 + \frac{N_{x,0}}{(1-\bar{\pi}(x))^2} \pi(x) \right)^2 \sigma_{x,0}^2 \right)$$

$a$ is nonnegative as demonstrated above. We assume that there exists at least one $x$ for which either or both of $\mu_{x,1} \neq 0$ and $\mu_{x,0} \neq 0$ is true. Without loss of generality, we focus on the case in which there is exactly one such $x$ (i.e., $\mu_{x,1} \neq 0$ and $l$ or $\mu_{x,0} \neq 0$, while $\mu_{x',1} = \mu_{x',0} = 0$ for all $x' \in \mathcal{X} \setminus x$)

$$\mathbb{V}\left(\sum_{x \in \mathcal{X}} N_x \left( \frac{\hat{\pi}(x)}{\pi(x)} \mu_{x,1} - \frac{1 - \hat{\pi}(x)}{1 - \pi(x)} \mu_{x,0} \right) \right) = \mathbb{V}\left(\sum_{x \in \mathcal{X}} N_x \left( \frac{\hat{\pi}(x)}{\pi(x)} \mu_{x,1} - \frac{1 - \hat{\pi}(x)}{1 - \pi(x)} \mu_{x,0} \right) \right)$$

$$= \mathbb{V}\left(\sum_{x \in \mathcal{X}} N_x \left( \frac{\hat{\pi}(x)}{\pi(x)} \mu_{x,1} - \frac{1 - \hat{\pi}(x)}{1 - \pi(x)} \mu_{x,0} \right) \right)$$

$$= \mathbb{V}\left(\sum_{x \in \mathcal{X}} N_x \left( \frac{\hat{\pi}(x)}{\pi(x)} \mu_{x,1} - \frac{1 - \hat{\pi}(x)}{1 - \pi(x)} \mu_{x,0} \right) \right)$$

$$= \mathbb{V}\left(\sum_{x \in \mathcal{X}} N_x \left( \frac{\hat{\pi}(x)}{\pi(x)} \mu_{x,1} - \frac{1 - \hat{\pi}(x)}{1 - \pi(x)} \mu_{x,0} \right) \right)$$

$$= \mathbb{V}\left(\sum_{x \in \mathcal{X}} N_x \left( \frac{\hat{\pi}(x)}{\pi(x)} \mu_{x,1} - \frac{1 - \hat{\pi}(x)}{1 - \pi(x)} \mu_{x,0} \right) \right)$$

$$= \mathbb{V}\left(\sum_{x \in \mathcal{X}} N_x \left( \frac{\hat{\pi}(x)}{\pi(x)} \mu_{x,1} - \frac{1 - \hat{\pi}(x)}{1 - \pi(x)} \mu_{x,0} \right) \right)$$

$$= \mathbb{V}\left(\sum_{x \in \mathcal{X}} N_x \left( \frac{\hat{\pi}(x)}{\pi(x)} \mu_{x,1} - \frac{1 - \hat{\pi}(x)}{1 - \pi(x)} \mu_{x,0} \right) \right)$$

$$= \mathbb{V}(\sum_{x \in \mathcal{X}} N_x (\mu_{x,1} - \mu_{x,0})) = \mathbb{V}(N_x (\mu_{x,1} - \mu_{x,0}))$$
We rewrite $\mu_{x,1} = (\mu_{x,1} - \mu_{x,0}) + \mu_{x,0}$ and see that
\[
\mathbb{V} \left( \sum_{x \in \mathcal{X}} N_x \left( \frac{\hat{\pi}(x)}{\pi(x)} \mu_{x,1} - \frac{1 - \hat{\pi}(x)}{1 - \pi(x)} \mu_{x,0} \right) \right) = \mathbb{V} \left( \frac{N_{x,1}}{\pi(x)} \mu_{x,1} - \frac{N_{x,0}}{(1 - \pi(x))} \mu_{x,0} \right)
\]
\[
= \mathbb{V} \left( \frac{N_{x,1}}{\pi(x)} [(\mu_{x,1} - \mu_{x,0}) + \mu_{x,0}] - \frac{N_{x,0}}{(1 - \pi(x))} \mu_{x,0} \right)
\]
\[
= \mathbb{V} \left( \frac{N_{x,1}}{\pi(x)} (\mu_{x,1} - \mu_{x,0}) + \mu_{x,0} \left( \frac{N_{x,1}}{\pi(x)} - \frac{N_{x,0}}{(1 - \pi(x))} \right) \right)
\]

We know that $\mathbb{V} \left( \frac{N_{x,1}}{\pi(x)} (\mu_{x,1} - \mu_{x,0}) + \mu_{x,0} \left( \frac{N_{x,1}}{\pi(x)} - \frac{N_{x,0}}{(1 - \pi(x))} \right) \right)$ is the sum of the variances of the two terms plus twice their covariance, so we focus on $\mathbb{V} \left( \frac{N_{x,1}}{\pi(x)} (\mu_{x,1} - \mu_{x,0}) \right)$.

\[
\mathbb{V} \left( \frac{N_{x,1}}{\pi(x)} (\mu_{x,1} - \mu_{x,0}) \right) = \mathbb{E} \left( \mathbb{V} \left( \frac{N_{x,1}}{\pi(x)} (\mu_{x,1} - \mu_{x,0}) \mid N_x \right) \right) + \mathbb{V} \left( \mathbb{E} \left( \frac{N_{x,1}}{\pi(x)} (\mu_{x,1} - \mu_{x,0}) \mid N_x \right) \right)
\]
\[
= \mathbb{E} \left( N_x \left( \frac{\mu_{x,1} - \mu_{x,0}}{\pi(x)} \right)^2 \right) \geq 0
\]
\[
\mathbb{V} \left( \mu_{x,0} \left( \frac{N_{x,1}}{\pi(x)} - \frac{N_{x,0}}{(1 - \pi(x))} \right) \right) \geq 0
\]

Similarly,
\[
\mathbb{V} \left( \mu_{x,0} \left( \frac{N_{x,1}}{\pi(x)} - \frac{N_{x,0}}{(1 - \pi(x))} \right) \right) = \mathbb{E} \left( \mathbb{V} \left( \mu_{x,0} \left( \frac{N_{x,1}}{\pi(x)} - \frac{N_{x,0}}{(1 - \pi(x))} \right) \mid N_x \right) \right)
\]
\[
+ \mathbb{V} \left( \mathbb{E} \left( \mu_{x,0} \left( \frac{N_{x,1}}{\pi(x)} - \frac{N_{x,0}}{(1 - \pi(x))} \right) \mid N_x \right) \right)
\]
\[
= \mathbb{E} \left( \mathbb{V} \left( \mu_{x,0} \left( \frac{N_{x,1}}{\pi(x)} - \frac{N_{x,0}}{(1 - \pi(x))} \right) \mid N_x \right) \right)
\]
\[
= \mathbb{E} \left( \mathbb{V} \left( \mu_{x,0} \left( \frac{N_{x,1}}{\pi(x)} - \frac{N_{x,0}}{(1 - \pi(x))} \right) \mid N_x \right) \right)
\]
\[
= \mathbb{E} \left( \mathbb{V} \left( \mu_{x,0} \left( \frac{N_{x,1}}{\pi(x)} + \frac{1}{(1 - \pi(x))} - \frac{N_{x}}{(1 - \pi(x))} \right) \mid N_x \right) \right)
\]
\[
= \mathbb{E} \left( \mathbb{V} \left( \mu_{x,0} \left( \frac{N_{x,1}}{\pi(x)} + \frac{1}{(1 - \pi(x))} \right) \mid N_x \right) \right)
\]
\[
= \mathbb{E} \left( \mathbb{V} \left( \mu_{x,0} \left( \frac{N_{x,1}}{\pi(x)(1 - \pi(x))} \right) \mid N_x \right) \right)
\]
\[
= \mathbb{E} \left( \frac{\mu_{x,0}}{(\pi(x)(1 - \pi(x)))^2} \mathbb{V} \left( N_{x,1} \mid N_x \right) \right)
\]
\[
= \mathbb{E} \left( \frac{\mu_{x,0}^2}{\pi(x)(1 - \pi(x))^2} (\pi(x)(1 - \pi(x))(N_x)) \right)
\]
\[
= \mathbb{E} \left( \frac{\mu_{x,0}^2}{\pi(x)(1 - \pi(x))} N_x \right)
\]
so we are left to analyze

\[
\text{Cov}\left(\frac{N_{x,1}}{\pi(x)}, \frac{N_{x,1}}{\pi(x)} - \frac{N_{x,0}}{(1 - \pi(x))}\right)
\]

We can show that this is nonnegative using the law of total covariance (see for example Casella and Berger (2002))

\[
\text{Cov}\left(\frac{N_{x,1}}{\pi(x)}, \frac{N_{x,1}}{\pi(x)} - \frac{N_{x,0}}{(1 - \pi(x))}\right) = \mathbb{E}\left(\text{Cov}\left(\frac{N_{x,1}}{\pi(x)}, \frac{N_{x,1}}{\pi(x)} - \frac{N_{x,0}}{(1 - \pi(x))} \mid N_x\right)\right)
\]

and so we solve

\[
\mathbb{E}\left(\mathbb{E}\left(\frac{N_{x,1}^2}{\pi(x)^2} - \frac{N_{x,1}N_{x,0}}{\pi(x)(1 - \pi(x))} \mid N_x\right)\right) = \mathbb{E}\left(\frac{(\pi(x)N_x + \pi(x)^2N_x(\pi_x - 1)}{\pi(x)^2} - \mathbb{E}\left(\frac{N_{x,1}N_{x,0}}{\pi(x)(1 - \pi(x))} \mid N_x\right)\right)
\]
Now, consider the entire variance
\[
\mathbb{V}
\left(\frac{N_{x,1}}{\pi(x)} (\mu_{x,1} - \mu_{x,0}) + \mu_{x,0} \left( \frac{N_{x,1}}{\pi(x)} - \frac{N_{x,0}}{(1 - \pi(x))} \right) \right)
\]
\[= \mathbb{V}
\left( \frac{N_{x,1}}{\pi(x)} (\mu_{x,1} - \mu_{x,0}) \right) + \mathbb{V}
\left( \mu_{x,0} \left( \frac{N_{x,1}}{\pi(x)} - \frac{N_{x,0}}{(1 - \pi(x))} \right) \right)
\]
\[+ 2\mu_{x,0} (\mu_{x,1} - \mu_{x,0}) \text{Cov} \left( \frac{N_{x,1}}{\pi(x)}, \frac{N_{x,1}}{\pi(x)} - \frac{N_{x,0}}{(1 - \pi(x))} \right)
\]
\[= \mathbb{E}
\left( \frac{N_{x} (\mu_{x,1} - \mu_{x,0})^2 (1 - \pi(x))}{\pi(x)} \right) + \mathbb{V}
\left( \mu_{x,1} - \mu_{x,0} \right)
\]
\[+ \mathbb{E}
\left( \frac{\mu_{x,0}^2}{\pi(x)(1 - \pi(x))} \right) N_x
\]
\[+ 2\mu_{x,0} (\mu_{x,1} - \mu_{x,0}) \mathbb{E}
\left( \frac{N_{x}}{\pi(x)} \right)
\]
\[= \mathbb{V}
\left( \mu_{x,1} - \mu_{x,0} \right)
\]
\[+ (\mu_{x,1} - \mu_{x,0})^2 \left( 1 - \pi(x) \right) \mathbb{E}
\left( \frac{N_{x}}{\pi(x)} \right) + \mu_{x,0}^2 \mathbb{E}
\left( \frac{N_{x}}{\pi(x)} \right) + 2\mu_{x,0} (\mu_{x,1} - \mu_{x,0}) \mathbb{E}
\left( \frac{N_{x}}{\pi(x)} \right)
\]
\[= \mathbb{V}
\left( \mu_{x,1} - \mu_{x,0} \right) + \mathbb{E}
\left( \frac{N_{x}}{\pi(x)} \right) \left( (\mu_{x,1} - \mu_{x,0}) \sqrt{1 - \pi(x)} + \frac{\mu_{x,0}}{\sqrt{1 - \pi(x)}} \right)^2
\]

Thus we have that
\[
\mathbb{V}
\left( \sum_{x \in \mathcal{X}} N_x \left( \frac{\hat{\pi}(x)}{\pi(x)} \mu_{x,1} - \frac{1 - \hat{\pi}(x)}{1 - \pi(x)} \mu_{x,0} \right) \right) \geq \mathbb{V}
\left( \sum_{x \in \mathcal{X}} N_x \left( \mu_{x,1} - \mu_{x,0} \right) \right)
\]

Now, we define
\[
b = \mathbb{V}
\left( \sum_{x \in \mathcal{X}} N_x \left( \frac{\hat{\pi}(x)}{\pi(x)} \mu_{x,1} - \frac{1 - \hat{\pi}(x)}{1 - \pi(x)} \mu_{x,0} \right) \right) - \mathbb{V}
\left( \sum_{x \in \mathcal{X}} N_x \left( \mu_{x,1} - \mu_{x,0} \right) \right)
\]

and we see that \( \mathbb{V}(\hat{\pi}^{\text{strat}}_{\text{strat}}) \leq \mathbb{V}(\hat{\pi}^{\text{strat}}_{\text{strat}}) \) when \( b \geq a \). This is true when \( \mu_{x,1} \) and \( \mu_{x,0} \) are “far enough” from zero that the variance benefit of estimating propensity scores outweighs the numerical effect detailed in Appendix B.1. Formally, we may choose a data-dependent \( \epsilon \) such that if \( |\mu(x)| + |\tau(x)| > \epsilon \) for at least one \( x \in \mathcal{X} \), then \( b \geq a \).

**APPENDIX C: PROOF OF COROLLARY 1**

The proof follows directly from the proof outlined in Appendix A.1. To make the extension to Theorem 2 as clear as possible, we index level sets of \( \pi(\mathcal{X}) \) using \( j \). For each \( j \in \pi(\mathcal{X}) \), we let \( m(j) = \{ x : \pi(x) = j \} \). By the assumptions of Corollary 1, we have that for all \( j \in \pi(\mathcal{X}) \) and all \( \ell = \{1, \ldots, m(j)\} \),

- \( \sigma^2_{j \ell} = \sigma^2_j \)
- \( \mu_{j \ell,1} = \bar{\mu}_{j,1} \)
- \( \mu_{j \ell,0} = \bar{\mu}_{j,0} \)
where the second two points are true because both $\mu(x)$ and $\tau(x)$ are constant for each $x$ with $\pi(x) = j$. Thus, it follows that

$$
\mathbb{V} \left( \sum_{j \in s(X)} \hat{\tau}_{ipw}^j, s \right) \leq \mathbb{V} \left( \sum_{j \in s(X)} \left( \sum_{\pi: g(\pi) = j} \frac{N_\pi}{N_j} \hat{\tau}_{ipw}^j, \pi \right) \right)
$$

**APPENDIX D: PROOF OF COROLLARY 2**

As in Appendix C, the proof follows more or less directly from the proof of Theorem 2. By assumption, there exist $x, x' \in \mathcal{X}$ such that

- $x \neq x'$
- $\pi(x) \neq \pi(x')$
- $s(x) = s(x')$
- $\mu(x) = \mu(x')$
- $\tau(x) = \tau(x')$

We let $j = s(x) = s(x')$ denote a level set of $s$ for which the above conditions are true, and we define $m(j) = |\{x : \pi(x) = j\}|$. By the assumptions of Corollary 2, we have that for all $\ell = \{1, \ldots, m(j)\}$,

- $\sigma^2_{j, \ell} = \sigma^2_j$
- $\mu_{j, \ell, 1} = \mu_{j, 1}$
- $\mu_{j, \ell, 0} = \mu_{j, 0}$

Thus, it follows that

$$
\mathbb{V} \left( \sum_{j \in s(X)} \hat{\tau}_{ipw}^j, s \right) \leq \mathbb{V} \left( \sum_{j \in s(X)} \left( \sum_{\pi: g(\pi) = j} \frac{N_\pi}{N_j} \hat{\tau}_{ipw}^j, \pi \right) \right)
$$