Conditional Balance Tests: Increasing Sensitivity and Specificity With Prognostic Covariates

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May 24, 2022

Abstract

Researchers often use covariate balance tests to assess whether a treatment variable is assigned “as-if” at random. However, standard tests may shed no light on a key condition for causal inference: the independence of treatment assignment and potential outcomes. We focus on a key factor that affects the sensitivity and specificity of balance tests: the extent to which covariates are prognostic, that is, predictive of potential outcomes. We propose a “conditional balance test” based on the weighted sum of covariate differences of means, where the weights are coefficients from a standardized regression of observed outcomes on covariates. Our theory and simulations show that this approach increases power relative to other global tests when potential outcomes are imbalanced, while limiting spurious rejections due to imbalance on irrelevant covariates.

Keywords: Balance tests, natural experiments, prognostic covariates, ignorability, weights

Acknowledgments: We are grateful to Lily Medina for valuable assistance.

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1 Introduction

Methodologists often urge researchers to test observable implications of assumptions needed for causal inference. In natural experiments, regression-discontinuity designs, and related methods, a common approach is to test for statistical balance on pre-treatment covariates across the treatment and control groups. The logic appears straightforward: if a coin flip determined treatment assignment, as assumed or as stipulated by design in such studies, we would expect equal distributions of all covariates in the two groups—since covariate values are determined prior to treatment assignment. Thus, up to chance error, we should find proportionately as many men as women, or as many young as old people, assigned to treatment and control conditions. A statistically insignificant association between treatment assignment and gender, age, or other covariates is consistent with “as-if” random assignment of a treatment—while the presence of such an association may suggest a flaw in the design.2

Unfortunately, such tests may shed no light on a key condition for causal inference: the independence of treatment assignment and potential outcomes, that is, the outcomes that would be realized under counterfactual assignment to different treatments.3 Self-selection processes that lead to failures of as-if random may imply imbalances on some covariates but not on others. In an observational study of the efficacy of a new drug, for example, sicker patients may select into the treatment group—but men may be as likely to do so as women. This leads to expected balance on gender across the treatment and control groups but substantial imbalance on prior health. These covariates differ, however, in their informativeness about potential outcomes: health status after an intervention is likely tightly related to prior health status while gender may be unrelated to potential outcomes. If we only have data on gender, we may fail to reject random assignment; and yet the potential outcomes—the potential health outcomes under assignment to different treatments—are very likely imbalanced. Even if we do have data on both gender and prior health, the asymmetry in the informativeness of these covariates is not well captured by standard procedures, such as covariate-by-covariate or (unweighted) multivariate balance tests. Such tests may reject spuriously, due to imbalances on irrelevant covariates unrelated to potential outcomes; or they may fail to reject when potential outcomes are in fact imbalanced, because they do not take sufficient account of the extent to which covariates are prognostic, that is, predictive of potential outcomes.

We show in this paper how to diagnose and increase the power of balance tests to detect the dependence of treatment assignment on potential outcomes. When covariates are sufficient for potential outcomes, in a sense we define, finding imbalance on covariates implies imbalance of potential outcomes. However, tests using irrelevant covariates—those unrelated to potential outcomes—can lead researchers to over-reject as-if random when it is true or under-reject it when it is false. Projecting potential outcomes onto covariates before conducting balance tests helps to avoid this problem. A key insight is that post-treatment data on outcomes are often available at the time researchers conduct balance tests. This implies that the extent to which covariates are predictive of potential outcomes can be assessed empirically, for example, by using control- or treatment-group samples to estimate a finite-population relation between covariates and potential outcomes. Our principal test statistic is based on the difference between the fitted value of the average $\hat{Y}_i(0)$, the covariate-adjusted average potential outcome under control, in the treatment and control group samples. This yields a “conditional balance test,” with which we assess the independence of

1 Covariates could also be defined to include “placebo outcomes,” i.e., post-treatment variables known or assumed to be unaffected by treatment assignment (Eggers et al. (2021), Caughey et al. (2017)).
2 See e.g. Freedman (1999), Rosenbaum (2002, 2010, 2012), Hansen and Bowers (2008), Sekhon (2009), Imai et al. (2010), Dunning (2012), Caughey et al. (2017); or Eggers et al. (2021), Neyman et al. (1923), Holland (1986), Rubin (1974).
treatment assignment and $Y_i(0)|X$. The statistic is equivalent to a weighted sum of the differences of means for each individual covariate, where the weights are the fitted coefficients from the standardized regression of potential outcomes on covariates in the control group sample. This omnibus test—“omnibus” because unlike covariate-by-covariate tests, it is based on the joint distribution of covariate differences—thus takes account of the “importance” of each covariate, or the extent to which different covariates are linearly prognostic. The test upweights covariates that we would expect to be imbalanced if treatment assignment were in fact dependent on potential outcomes, while downweighting irrelevant, non-prognostic covariates.

Our theoretical and simulation results suggest several key findings and advantages of our approach. First and most importantly, the test is both more sensitive and more specific than existing approaches in the following sense: it tends to fail to reject as-if random assignment due to imbalances on irrelevant covariates more often than existing approaches (and is thus more specific); but it will boost rejection probabilities relative to standard tests when prognostic covariates are imbalanced (and thus it is sensitive). Methodologists have rightly pointed out that balance tests are often poorly powered to reject false null hypotheses stipulating as-if random assignment (Cattaneo et al. 2015). Moreover, failing to reject a null hypothesis of as-if random assignment is not the same as accepting it, leading some methodologists to recommend alternatives to standard balance tests (Hartman and Hidalgo 2018; Hartman 2021). Our results suggest a subtle relationship between the prognostic value of covariates and the power of tests, however. Unweighted omnibus tests may overreject null hypotheses when treatment assignment is independent of potential outcomes, because they give too much weight to irrelevant covariates unrelated to potential outcomes. Our weighted procedure can reduce false positives or Type I error in this case. However, when treatment assignment does depend on potential outcomes and we have prognostic covariates available, our regression-weighted approach rejects as-if random more often than unweighted approaches, thus limiting false negatives or Type II error and increasing power. In other words, the test limits both false negatives and false positives.

Second, unlike covariate-by-covariate tests, our approach provides a clear rejection rule based on a combination of covariate differences. Researchers often appear to rely on an informal rule of thumb in assessing the results of covariate-by-covariate tests. For example, if a treatment is randomly assigned, we would expect significant covariate imbalances at the 0.05 level in only 1 out of 20 or 5% of independent tests. However, when making multiple statistical comparisons across different covariates and when tests are dependent—which occurs whenever covariates are correlated with each other, that is to say, almost always in practice—such a rule-of-thumb is not reliable. It can therefore be a matter of opinion whether the totality of the evidence from a set of covariate balance tests undercuts a claim of as-if random. We thus add to recent work that proposes the use of omnibus statistics or combinations of $p$-values, including those that allow for dependence among covariates.5

Finally, our approach provides a basis for assessing the evidentiary value of balance tests. Pretreatment (lagged) measures of outcome variables tend to be highly prognostic, leading methodologists to counsel their use in falsification tests. Yet such predictive covariates may or may not be available. It is also an empirical question whether a lagged outcome or any other covariate is in fact prognostic in any study, as our motivating example in the next section suggests. In some settings all available covariates may be only weakly related to potential outcomes. Our approach suggests diagnostics that can help researchers assess the strength of the test of as-if random. When none of the pre-treatment covariates

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4See also Imai et al. 2008, who develop critiques of balance tests that differ from the issues we raise in this article.

5See e.g. Hansen and Bowers (2008); Caughey et al. (2017); or Gagnon-Bartsch and Shem-Tov (2019).

6Imbens and Rubin 2015: 483-4.
to which researchers have access are prognostic, balance tests may have especially weak power over an alternative hypothesis that potential outcomes are imbalanced. Their results should then not be taken as strong evidence in favor of the key identifying assumption for causal inference.

We take inspiration from a large literature on multiple testing in statistics and epidemiology, which recommends upweighting tests for “important” hypotheses—or those that are most plausibly false—in p-value combinations. However, our approach gives specific content to which hypotheses are most likely to be false in balance tests, by upweighting covariates that are related to potential outcomes. We also recommend constructing an omnibus p-value by evaluating the weighted sum of differences of covariate means directly, rather than by combining p-values from separate covariate-by-covariate tests; this differs from e.g. Caughey et al. (2017), who develop a non-parametric combination approach to generating omnibus p-values for placebo tests, including balance tests, using a combination metric owing to Fisher (1935). However, the choice of combination metric can introduce discretion; and this choice is not required for balance tests, in which mean differences for different covariates can be readily combined. Our approach is related to papers by Hansen and Bowers (2008), who develop a procedure for conducting balance tests based on combining covariate differences of means in block- (and cluster-) randomized experiments, and Gagnon-Bartsch and Shem-Tov (2019), who provide a classification permutation test. None of these important papers, however, considers variation in the prognostic power of different covariates. While many works on causal inference mention the usefulness of conducting tests for balance on prognostic covariates, the rationale is not always explicit, nor are the potential gains of doing so in terms of increased statistical power against a clear null hypothesis. Hansen (2008) proposes a “prognostic score” that is akin to our measure of prognosis, but he develops this approach for purposes of covariate adjustment in an outcome model. Our major innovation is therefore our focus on the association between covariates and potential outcomes as the basis for balance testing.

In the next Section 2, we discuss examples and give intuition for why pre-treatment covariates are created unequal. Section 3 then develops statistical theory behind our approach. In Section 4, we present simulation evidence on the power of our approach under different assumptions about data-generating processes. We conclude by discussing several possible extensions, including conditional balance tests based on more flexible, non-linear regression fits.

2 Motivation: the varying prognostic power of covariates

In an important study, Caughey and Sekhon (2011) appraise the use of regression-discontinuity designs to study the effect of incumbency, taking data from close U.S. House elections (1942-2008). A priori, in a very close election, which party winds up with a slightly greater vote share at time \( t \) seems quite plausibly as-if random. If so, assignment of the treatment—party incumbency—is independent of potential outcomes, as well as pre-treatment covariates. For this reason, the close-election design has become extremely widespread. Concerningly, however, Caughey and Sekhon’s plot of p-values from balance tests (their Figure 2) suggests statistically significant imbalances in past incumbency, as well as the winning party’s past vote share, campaign spending, and measures of candidate quality, suggesting a possible failure of as-if random in very close elections. Still, districts barely won by Democrats at time \( t \) do not

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7 Examples include Holm (1979); Benjamini and Hochberg (1997); and Genovese et al. (2006). See also Fisher (1935); Kost and McDermott (2002); or Westfall (2005).

8 Caughey and Sekhon (2011) also present evidence of “sorting” at the 0% vote margin: the incumbent party wins the closest elections more than twice as often as it loses (see their Figure 1).
differ from those barely won by Republicans on several other political and demographic variables, e.g. whether the state has a Democratic governor or secretary of state, the margin of victory in the presidential race, voter turnout, whether the seat is open (i.e., the incumbent candidate is not running), or the percentage of urban, Black, or foreign-born residents. Thus, we see imbalances on some covariates but lack of imbalance on several others.

How should one interpret the totality of the evidence in a balance plot such as Caughey and Sekhon’s? These researchers (rightly, in our view) attribute particular importance to the imbalance on the winning party’s past incumbency and vote share at time $t-1$, since these variables are presumably highly correlated with future incumbency and vote share. However, there is no formal procedure that takes into account the extent to which covariates are prognostic; and the covariates included in the balance tests are correlated, so simply comparing the number of rejections to the number of tests (e.g. to see if the ratio is greater than 1 out of 20) is not informative. In a subsequent study, Eggers et al. (2015) confirm that lagged incumbency seems to be the major driver of imbalances in Caughey and Sekhon’s data: in a procedure conceptually related to one we propose in Section 3, they show that Democratic near-winners and near-losers are not significantly different on pre-treatment covariates other than lagged incumbency, once the latter is controlled in a regression. They then extend the Caughey and Sekhon study to a broad range of majoritarian elections around the world, comparing close election winners and losers only on a measure of lagged incumbency. They find balance on this covariate in every other setting they examine, leading them to argue that the observed imbalance in U.S. House elections in the latter part of the twentieth century is unusual and may reflect special features of that context or may simply be due to chance.

The extent to which lagged incumbency is prognostic thus appears critical to adjudicating this debate about whether close elections are as-if random. The same is true of pre-treatment covariates in many other natural experiments or discontinuity designs. However, these studies and many others do not take prognosis into account formally or empirically. Eggers et al. (2015) are right to assess covariate balance across a wide range of elections. Yet they effectively assert that lagged incumbency is the only important covariate on which to test for balance across these contexts. In fact, the prognostic value of lagged incumbency varies across countries and types of elections. As we show in Appendix Tables 1-2, in their data, the correlation between the vote share of the incumbent party at time $t-1$ and time $t$ is 0.79 across all countries and election types but varies from a low of 0.09 in Brazilian mayoral elections to a high of 0.91 in the German Bundestag (full data set); in close elections (defined by a bandwidth of 0.5, i.e., the margin between the winning and runner-up party is less than 1 percentage point), it varies from a high of 0.32 in New Zealand’s post-war parliament to a low of −0.16 is the Canadian House of Commons (1867-1911).

Perhaps most importantly, the average correlation is just 0.02 across all close elections studied by Eggers et al., while it is substantially higher in the post-war U.S. House elections studied by Caughey and Sekhon (0.83 in the full data and 0.24 in close elections).

The prognosis of covariates is rarely considered systematically in balance testing, however. We coded a random sample of 150 articles using randomized experiments, natural experiments, and regression-discontinuity designs in three top journals in political science (the American Political Science Review, the

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Eggers et al. (2015: 262-3) argue that (a) the variety of characteristics on which winners and losers of close elections may vary can all be viewed as proxies for (are highly correlated with) incumbency; (b) testing for other covariates introduces multiple testing concerns; and (c) incumbency “confers electoral benefits in a variety of electoral settings around the world.”

This comports also with findings on the varied causal effects of incumbency across contexts; see e.g. Schiumerini (2015).

Restricting the analysis to close elections attenuates the correlations by truncating the range of variation on incumbent vote share at time $t$; yet this is arguably the relevant subset of the data in which to assess prognosis, since this is the set in which balance tests are typically conducted.
Overall, 52 percent of the sampled articles presented covariate balance tests in the body or appendix of the paper. The majority (56 percent) of those use only covariate-by-covariate tests, rather than some omnibus test statistic (such as the \( p \)-value for the \( F \)-statistic from the regression of a treatment indicator on all covariates). Only 18 percent of tests used a lagged dependent variable as a covariate. And we found no examples of systematic efforts to account for the prognostic importance of covariates in balance tests, for instance, using a weighted procedure like that we propose in this paper. The situation does not appear dissimilar in economics or other social science disciplines; in particular, procedures that take account of covariates’ degree of prognosis appear absent in the applied literature.

In practice, the covariates used in balance tests vary substantially in the extent to which they predict potential outcomes. We took a small further random sample from the 150 studies we coded, excluding the randomized experiments, stratifying by natural experiment versus discontinuity and on the presence of a lagged dependent variable or explicit discussion of prognosis in the paper. We then used replication data, where available, to calculate two measures for each study: the multiple \( R^2 \) from the regression of observed potential outcomes in the control group on all available covariates (“Prognosis”) and the multiple \( R^2 \) from the regression of a treatment assignment indicator on all available covariates (“Imbalance”). We note that we do not intend these measures as providing formal tests of prognosis or imbalance; we elaborate approaches to testing in the next section.

Figure[1] which plots these measures for our smaller sample of studies, suggests several insights. While a great deal of attention in balance testing focuses on the extent of imbalance, there is in fact considerable variation across studies in prognosis. Close to the vertical axis, covariates are non-prognostic and thus bear little apparent relationship to potential outcomes. For reasons we develop in the next section, such studies therefore provide less powerful tests of as-if random. Moreover, studies located in the upper-left quadrant may be prone to spurious rejection with standard procedures—because covariates unrelated to potential outcomes are imbalanced. In the lower left quadrant, the important concern may be that none of the measured covariates are prognostic of potential outcomes but we find balance on treatment assignment—leading to a form of Type I error in which we fail to reject, yet potential outcomes themselves may be imbalanced.

Moving towards the right side of the figure, we have studies in which covariates are more predictive of potential outcomes. These are studies in which a rejection may be most persuasive of a failure of as-if random—and a failure to reject most reassuring. We note that we would expect empirically to find relatively little imbalance in published natural experiments and discontinuity designs, due to a form of publication bias: studies that find substantial are unlikely to be published as natural experiments we may not expect heavy concentration. Indeed, our sampled studies bunch in the bottom part of the plot, where the \( R^2 \) for the regression of treatment assignment on covariates is low. The exception may be critiques, such as Caughey and Sekhon’s. Yet for those studies, the degree of prognosis suggests the power of the critique, with cases in the top-right quadrant—i.e. with prognostic and imbalanced covariates—the most damning. We develop the reasons why in the next section.

For code used in the sampling, see https://github.com/lilymedina/JSTOR_query.
The figure plots a random sample of natural experiments and regression-discontinuity (RD) designs drawn from all those published in the *American Political Science Review*, *American Journal of Political Science*, and *Journal of Politics*, 2000-2019; Caughey and Sekhon (2011) is added. Prognosis is the $R^2$ from a regression of potential outcomes under control on all available covariates (control group only). Imbalance is the $R^2$ from a regression of treatment assignment on all available covariates.

### 3 Are potential outcomes balanced? Why prognosis matters

There are at least two reasons that prognosis of covariates matters for testing as-if random—and also thus why covariates with differing degrees of prognosis should not be “treated equal.”

First, the most direct test of the key identifying condition for causal inference would assess balance in *potential outcomes* across the treatment and control groups. A direct test of this assumption is impossible, however, due to the fact that once treatment has occurred, we do not observe potential outcomes under control in the treatment group or potential outcomes under treatment in the control group. A covariate that is strongly associated with potential outcomes may nonetheless give us substantial information about this realized balance. Conversely, covariates unrelated to potential outcomes are uninformative about as-if random. This may be the reason that methodologists advise researchers to conduct balance tests on lagged dependent variables (e.g. Imbens and Rubin [2015] section 21.3), but the rationale for doing so is not always stated explicitly. We formalize this claim in this section as a statement about the sufficiency of covariates for potential outcomes. We show that imbalance in sufficient covariates implies imbalance in potential outcomes. Covariates that are highly prognostic—in the limit, that predict potential outcomes perfectly—thus allow for a direct test of the key identifying condition.
Second and relatedly, theories of self-selection bias suggest that covariates strongly related to potential outcomes are the most likely to be imbalanced across treatment and control groups, if as-if random assignment fails. If subjects have the opportunity to select into treatment and control groups, as in many observational studies, then—contrary to the assumption of as-if random—they may do so in a way that reflects the outcomes they would experience either under treatment and control. Sicker patients may select into the group that receives a new treatment if given the chance, since they need the treatment. This leads us to expect imbalance on prior health if as-if random fails—but not necessarily other covariates. As we outline here, prioritizing such prognostic covariates when assessing balance allows more sensitive and more powerful tests of as-if random.

In this section, we develop theory behind our approach, using a design-based, finite-population framework. After describing the set-up, we clarify conditions under which balance tests can be used to test as-if random. We then describe our conditional balance test, which consists of a regression-weighted test statistic. We compare its large-sample distribution to the exact distribution of an unweighted sum of covariate differences. In practice, we recommend permutation approaches for hypothesis testing.

### 3.1 A finite-population framework

Consider a study with a completely enumerated finite population of \( N \) units indexed by \( i = 1, \ldots, N \). Using standard notation in the tradition of Neyman (1923) and Rubin (1978), let \( Y_i(1) \) and \( Y_i(0) \) be potential outcomes—that is, the outcomes for unit \( i \) that would be realized under assignment to treatment or control groups, respectively.

The causal effect for each unit is \( \tau_i = Y_i(1) - Y_i(0) \). The Average Treatment Effect (ATE) is \( \tau = \mathbb{E}[Y_i(1) - Y_i(0)] \), where the expectation is taken over the draw of a single unit at random from the finite population (or “study group”) of \( N \) units. The random variable \( Z_i \in \{0, 1\} \) denotes treatment assignment, with 0 corresponding to a control group and 1 to a treatment group; an \( N \times 1 \) random vector \( Z \) collects the \( Z_i \). (We use boldface font for vectors and matrices). The sizes of the treatment and control groups are fixed at \( n_1 \) and \( n_0 \), respectively, with \( n_1 + n_0 = N \).

Suppose there is a set of \( p \) pre-treatment covariates that could be collected in an \( N \times p \) matrix \( X \), with the value \( X_{ij} \) denoting the value for unit \( i \) on covariate \( j = 1, \ldots, p \). Covariate values are realized prior to treatment assignment and are assumed to be unaffected by the treatment. In some settings, we may be missing columns of \( X \)—perhaps because some covariates are difficult to observe or measure.

### 3.2 Testing random assignment

A key condition sufficient for identification of the ATE is

**Assumption 1. (As-if Random Assignment)*** \( Z \perp \perp \{Y(1), Y(0)\} \)

where \( \perp \perp \) denotes “is independent of.”

According to Assumption[1] treatment is assigned independently of potential outcomes. This condition ensures, for example, that sicker patients do not go systematically to the treatment group in a drug trial studying health outcomes, or that those less prone to vote do not disproportionately receive a vote-mobilizing intervention.
If this condition holds, the true treatment effect is estimable: we can form an unbiased estimator such that $\mathbb{E}[\hat{Y}|Z = 1] - \mathbb{E}[\hat{Y}|Z = 0] = \tau$. The assumption of “strong ignorability” given in Assumption 1 can be contrasted with “weak ignorability,” or $Z \perp \{Y(1), Y(0)|X\}$, which we consider later. In many natural experiments, analysts seek to assess the stronger, more useful Assumption 1, which obviates the possibility of confounding from unobserved variables (e.g., unmeasured or mismeasured elements of $X$) and thus allows simple, transparent estimation of the ATE, such as by taking a difference of mean outcomes in the treatment and control groups.

Assumption 1 cannot be directly verified, however, since $\{Y_i(1), Y_i(0)\}$ is not completely observed for any unit. This is sometimes called ‘the fundamental problem of causal inference’ (Holland, 1986). In a randomized experiment, the statistical independence of treatment assignment and potential outcomes is an implication of the physical process of randomization (Fisher 1933, Zhao and Ding 2021). In the context of natural experiments, “as-if” random assignment is held to be an implication of some concrete process of treatment assignment that produces a haphazard allocation, in particular, one that does not depend on the units’ potential outcomes (Freedman 1991, Dunning 2012).

With additional assumptions, however, it is possible to make Assumption 1 falsifiable.

### 3.3 Standard practice, and two counterexamples

Standard practice involves testing the claim that $Z \perp X$. The reasoning appears to be that:

**Claim 1. (Standard Practice: Balance tests)**

\[
Z \perp X \text{ and } X \not\perp Y(0), Y(1) \text{ jointly imply that } Z \perp Y(0), Y(1) \iff Z \perp X.
\]

Hence $Z \not\perp X \implies Z \not\perp Y(0), Y(1)$.

Claim 1 is not correct, however.

**Example 1. (Counterexample to Claim 1: False positives)**

Suppose $X = X^S \cup X^N$ (‘signal’ and ‘noise’, respectively), such that $X^S \not\perp Y(1), Y(0)$, while $X^N \perp Y(1), Y(0)$. Suppose further, that Nature has adversarially chosen $Z$ so that $Z \not\perp X^N$ and $Z \perp X^S$.

Then, we have that $Z \not\perp X$ but it does not follow that $Z \not\perp Y(1), Y(0)$.

To make this concrete, suppose that each individual observation is assigned an observed random number $X^N$. Suppose $Z$ is chosen in a way that depends on $X^N$. It is no longer the case that $Z$ is independent of observed covariates $X$. However, this is irrelevant, since the dependence between $Z$ and $X^N$ does not imply dependence between $Z$ and $Y(0), Y(1)$.

A researcher who believed Claim 1 might perform a balance test, observe imbalance between treatment and control groups on some subset of covariates, and conclude that treatment was not randomly assigned. However, if the imbalanced covariates are unrelated to potential outcomes, then their imbalance does not constitute evidence that Assumption 1 fails.

Conversely, the researcher might find balance on the subset of spurious covariates. But their balance also does not constitute evidence that potential outcomes are balanced, per the next counterexample.

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$^{13}$It also implies that the observed difference in means evaluated at each level of the covariate converges to the true conditional average treatment effect.
Example 2. (Counterexample to Claim 1: False negatives)

Suppose we measure only $X^N$. By $Z \perp \perp X^N$, we do not expect to observe imbalance, because we only observe irrelevant covariates: we will fail to reject the independence of covariates and treatment assignment with probability $1 - \alpha$, where $\alpha$ is the level of the test. But this does not provide evidence that $Z \perp \perp Y(1), Y(0)$, since we have $Z \not\perp \perp X^S$, and $X^S \not\perp \perp Y(0), Y(1)$.

In sum, if we only measure noise covariates—those unrelated to potential outcomes—then finding balance or imbalance on those covariates does little to verify Assumption (1).

3.4 The informativeness of covariates

The above discussion suggests we should consider the informativeness of covariates when constructing balance tests.

For this, we need the following assumption from [Dawid (1979)]:

**Definition 1. (Sufficiency of Covariates)**

A set of variables $X$ is sufficient for a random variable $\phi$ if $\forall X' \neq X$:

$$\phi \perp \perp X'|X$$

Equivalently, if $\sigma(X), \sigma(\phi)$ are the $\sigma$-algebras generated by $\phi, X$, then $\sigma(\phi) = \sigma(X)$, and $\sigma(X' \cap \phi) \subseteq \sigma(X)$. In other words, $X'$ conveys no additional information about $\phi$ that $X$ does not. This is also equivalent to [Pearl (1988)]'s notion of a Markov Blanket.

When sufficiency holds, standard balance tests may allow us to infer whether or not $Z$ is as-if random, i.e., whether Assumption 1 holds.

**Theorem 1. (The Logic of Balance Tests when $X$ is Sufficient)**

Assume $X$ is sufficient for $\{Y(1), Y(0)\}$. Then, $Z \not\perp \perp X \implies Z \not\perp \perp \{Y(1), Y(0)\}$. **Proof:** See Appendix.

The intuition is that, if $X$ is sufficient for the potential outcomes, then it must contain all the information contained in $X^S$—that is, covariates that are not independent of the potential outcomes. Hence, an association between $X$ and $Z$ is an association between the potential outcomes and $Z$.

We show below (see Theorem 4) that when covariates are sufficient, there is an important implication of as-if random assignment: rejecting the independence of $Z$ and $Y(0)|X$ or $Y(1)|X$ allows us to reject the independence of $Z$ and $Y(0)$ or $Y(1)$. Hence, though both approaches must assume sufficiency to test Assumption 1, conditional balance testing—to which we turn next—offers an advantage to researchers over standard balance tests.

Assuming sufficiency sets a high bar in practice, but Definition 1 clarifies what is needed to interpret balance tests as an assessment of Assumption 1. In practice, we can evaluate sufficiency empirically using an F-test, sensitivity analysis, or nonparametric trimming bounds. In some settings, researchers have access to a lagged (pre-treatment) measure of the response variable, which may be highly prognostic—and even sufficient for $Y(0)$, in the sense of Definition 1. For example, it might even be plausible that the pre-intervention value is identically equal to the potential outcome under control—that is, $X_i = Y_{i,t-1} \equiv Y_i(0)$.
This may be why methodologists counsel their use in falsification tests (Imbens and Rubin 2015: 483-4), though the reasons are rarely directly articulated. However, this requires an assertion of *temporal stability*, which would be violated if, for instance, there are time trends in the outcome. As our examples in section (2) suggest, it is also an empirical question whether a given covariate, including a lagged outcome, is in fact prognostic (or sufficient).

Sufficiency of $X$ for $\{Y(0), Y(1)\}$ implies $Z \perp \perp \{Y(0), Y(1)|X\}$. This, of course, is equivalent to “weak ignorability” of treatment assignment. One might ask, if we have at our disposal covariates $X$ such that sufficiency (weak ignorability) holds, why we would not simply adjust for those covariates when estimating the ATE? One answer is that this requires analysts to choose an adjustment strategy, which can introduce discretion and dependence on the adjustment model. Strong ignorability, when plausible, instead allows simpler and more transparent estimation of the ATE. Our goal here is therefore to provide a more sensitive and specific test of Assumption 1.

### 3.5 A conditional balance test

We now turn to our conditional balance test—“conditional” because it is formed as a function of potential outcomes given or conditional on $X$.

Consider conditional expectation functions $E(Y_i(0)|X_i)$ and $E(Y_i(1)|X_i)$ that give the average value of potential outcomes at each value of $X$ in the finite population. The expectation is taken over a unit sampled at random from this finite population, or from each population stratum defined by a particular value $X = x$. These can also be called population regression functions, since they give the average of $Y(0)$ or $Y(1)$ at each value of $X$.

If we observed $Y_i(0)$ or $Y_i(1)$ for all units in the study group, we could form a linear or a more flexible non-linear approximation to these conditional expectation functions. We begin with the linear approximation because it provides clear intuition and motivates the regression-weighted test statistic we present in sub-section (3.6.2). Also, we focus first on the regression of $Y_i(0)$ rather then $Y_i(1)$ on covariates; one reason is that covariates such as the lagged (pre-intervention) outcome may be most prognostic for $Y_i(0)$, especially when unit treatment effects are not the same for all $i$ (see Hansen 2008). In the conclusion, we consider extensions of our procedure to more flexible regression fits and those that make use of information on $Y_i(1)$.

We can denote the fitted value of $Y(0)$ given $X$ for each unit in the finite population as

$$Y_i(0)_lr = X_i\beta$$

where “lr” denotes the linear regression and $X_i$ is a $p \times 1$ vector with elements $X_{i,j}$ for $j = 1, \ldots, p$. Here, $\beta$ is the $p \times 1$ vector of coefficients from the finite-population regression. The associated population

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14It might sometimes happen that all pre-intervention units are exposed to a treatment, and a randomized intervention removes some of them from treatment status; in that case, the logic would be parallel but reversed, and $Y_i,t-1 = Y_i(1)$ in that case.

15This can equivalently be stated as $\{Y(0), Y(1)\} \perp \perp Z|X$; see Dawid (1979).

16We can define either potential outcome regression function and form consistent estimators of both from sample data.

17Adapting notation from Angrist and Pischke (2009: Section 3.1), the regression in the finite population produces the $p \times 1$ vector

$$\beta = (\sum_{i=1}^N X_iX_i)^{-1}\sum_{i=1}^N X_iY_i(0),$$

where $X_i$ is a $p \times 1$ vector with elements $X_{i,j}$ for unit $i$. We write $\beta$ in this way to emphasize that this is the mechanical
residuals are \( e_i = Y_i(0) - \hat{X}_i \beta \) for \( i = 1, \ldots, N \). As is well known, the regression minimizes the sum of squared residuals in the finite population and \( e \perp X \) \citep{Cochran1977}: Ch. 7), where “\( \perp \)” is read “is orthogonal to” and \( e \) is the vector collecting the \( e_i \)'s. This stems simply from the mechanics of a linear regression in a completely enumerated study group of \( N \) units. The fitted value of \( Y(1) \) given \( X \), denoted \( Y_i(1)_{\text{lr}} \), can be analogously defined.

Each element \( \beta_j \) of \( \beta \) is a measure of the extent to which the associated covariate is linearly prognostic. That is, it gives the “informativeness” of covariate \( X_j \), relative also to the other covariates. Indeed, it can be represented as the coefficient from the bivariate regression

\[
\beta_j = \frac{\text{Cov}(Y(0), \tilde{X}_j)}{\text{Var}(\tilde{X}_j)} \quad (2)
\]

where \( \tilde{X}_j \) is the residual from the finite-population regression of \( X_j \) on the other \( p - 1 \) covariates.\(^{18}\) When \( Y(0) \) and \( X_j \) are standardized, each \( \beta_j \) is the standardized multiple regression coefficient relating \( \tilde{X}_j \) to \( Y(0) \).\(^{19}\) More prognostic covariates will have larger absolute values of standardized \( \beta_j \). Conversely, the coefficient \( \beta_j \) vanishes when the partial correlation between \( Y(0) \) and \( X_j \) is zero.

It is important to emphasize that \( \beta \) has no causal interpretation: the regression simply provides the best linear approximation of the potential outcomes \( Y(0) \) given \( X \) in the finite population. Covariates are fixed features of units that are not here considered amenable to manipulation; even if they were, there is no expectation or requirement that manipulation would lead to expected changes in the value of the outcome variable. Nor is the correlation between \( Y(0) \) and a given \( X_j \) secured as a feature of a design, such as the randomization of a treatment. There is not a general rule about the partial correlation (degree of informativeness) we should anticipate for \( X_j \). However, for many features of study units that are irrelevant to the outcomes under consideration, we might expect only a weak correlation, while for more informative or relevant covariates, the strength of the linear association with potential outcomes may be greater.

We can now define our key testable assumption in terms of the fitted potential outcomes. First, randomization implies

\[\text{Assumption 2. (Implication of As-If Random Assignment) } Z_i \equiv \{Y_i(0)_{\text{lr}}, Y_i(1)_{\text{lr}}\} \forall i.\]

Note the subscript “\( \text{lr} \)” as in equation (1), in contrast to Assumption (1). \( Y_i(0)_{\text{lr}} \) and \( Y_i(1)_{\text{lr}} \) are the fitted values in the finite population based on the projection of potential outcomes onto covariates. If treatment is assigned independently of potential outcomes and of \( Y(0) | X \) and \( Y(1) | X \). Since covariates are fixed features of units invariant to treatment assignment, Assumption (2) is an implication of Assumption (1).

However, unlike Assumption (1), Assumption (2) is directly testable, because it involves quantities that are observable or for which, as we show, we have consistent estimators in both the treatment and control groups.

\(^{18}\) This is a property of what is called the Frisch-Waugh-Lovell theorem or “regression anatomy” in economics \citep{AngristPischke2009:3.1.2; Freedman2009: Ex. 3.17}.

\(^{19}\) The standardized values of \( Y(0) \) and \( X_j \) are \( Y_i(0) - \bar{Y}(0)/\sigma_{Y(0)} \) and \( X_{ij} - \bar{X}_j/\sigma_{X_j} \), respectively, where \( \bar{Y}(0) \) and \( \bar{X}_j \) are the respective averages and \( \sigma_{Y(0)} \) and \( \sigma_{X_j} \) are the standard deviations in the finite population.
Assumption (2) therefore motivates the following null and alternative hypotheses:

\[ H_0 : E[Z'Y(0)_{tr} - (1 - Z)'Y(0)_{tr}] = 0 \]
\[ H_A : E[Z'Y(0)_{tr} - (1 - Z)'Y(0)_{tr}] \neq 0, \]

where the \( N \times 1 \) fixed vector \( Y(0)_{tr} = (Y_1(0), \ldots, Y_N(0)) \) collects the \( Y_i(0)_{tr} \). We can define analogous null and alternative hypotheses for \( Y(1)_{tr} \).

3.6 Estimation and hypothesis testing

This formulation of the null and alternative hypotheses implies that we may form tests of \( H_0 \) based on comparison of linear combinations of covariates in the treatment and control groups. In particular, we may form a test based on the difference

\[ Z'\hat{Y}(0)_{tr} - (1 - Z)'\hat{Y}(0)_{tr}, \]

where \( \hat{Y}(0)_{tr} \) estimates the finite-population regression \( Y(0)_{tr} \) given by equation (1). We can test the analogous null hypothesis based on \( Y(1)_{tr} \) using an estimator \( \hat{Y}(1)_{tr} \).

To be sure, \( Y_i(0)_{tr} \) and \( Y_i(1)_{tr} \) are each incompletely known because we do not observe \( Y_i(0) \) or \( Y_i(1) \) for all units \( i \). However, \( Y_i(0)_{tr} \) and \( Y_i(1)_{tr} \) are each linear combinations of the covariates \( X_i \), per equation (1); and \( X_i \) is fully observed for all \( i \), since covariates are fixed attributes invariant to treatment assignment. Finally, the treatment and control groups are exchangeable under random assignment. Viewed from another perspective, the control group is a simple random sample from the finite population. As we show later, this implies that we can form a consistent estimator \( \hat{Y}_i(0)_{tr} \) for \( Y_i(0)_{tr} \) using the control group sample (and analogously we could form \( \hat{Y}_i(1)_{tr} \) as an estimator for \( Y_i(1)_{tr} \) using the treatment group sample).

3.6.1 A baseline case: an unweighted sum of covariate differences of means

Before turning to our main estimator—a regression-weighted sum of covariate differences of means—we first turn briefly to a useful baseline case. Consider the unweighted sum of covariate differences of means:

\[ \delta_{UW} = \sum_{j=1}^{p} \delta_j, \]

where each \( \delta_j \) is the difference of means for covariate \( X_j \), \( j = 1, \ldots, p \), across the treatment and control groups and “UW” stands for “unweighted.” Note that each \( \delta_j \) is a random variable, with randomness induced solely by treatment assignment \( Z \), and thus the sum \( \delta_{UW} \) is a random variable as well.\footnote{To make the dependence on \( Z \) explicit, each random variable \( \delta_j \) could be written \( (1/n_1)Z'X_j + (1/n_0)(1 - Z)'X_j = X^T_j - X^C_j \), where \( X^T_j \) and \( X^C_j \) are the means of covariate \( j \) in the treatment and control groups, respectively.} Under random assignment, (5) gives a sample analogue of \( H_0 \) in (3), with the restriction that \( \beta_j = 1 \) for all \( j \).

This baseline case is closely related to standard approaches to balance tests. Instead of conducting a separate hypothesis test for each covariate, however, in (5) we sum the covariate-by-covariate differences-of-means in order to conduct a single (“omnibus”) test on the sum. Here, each covariate difference receives the same weight. We may expect the power of tests formed using \( \delta_{UW} \) against violations of Assumption \( 1 \)
to be limited: the statistic does not take into account the prognosis of the covariates but treats all covariates equally. Let \( \sigma^2_{X_j} \) denote the variance of the covariate \( X_j \) calculated over all \( N \) units in the finite population and \( \sigma_{X_j,X_k} \) be the finite-population covariance between covariate \( X_j \) and covariate \( X_k \). 

Then the following theorem gives the distribution of the random variable \( \delta_{UW} \).

**Theorem 2.** *(Distribution of the unweighted sum of covariate differences of means, \( \delta_{UW} \)) When treatment assignment is randomized, \( Xe(\delta_{UW}) = 0 \), and the sum has an exact and fully observable variance

\[
\text{Var}(\delta_{UW}) = \frac{N^2}{N-1} \frac{1}{n_0(n_1)} \left[ \sum_{j=1}^{p} \sigma^2_{X_j} + 2 \sum_{j<k}^{p} \sigma_{X_j,X_k} \right].
\]

Also, \( \delta_{UW} \) is asymptotically normal. When each covariate is standardized, so that the finite-population variance of each covariate is equal to 1, we have \( \sum_{j=1}^{p} \sigma^2_{X_j} = p \) and each covariance \( \sigma_{X_j,X_k} \) is the coefficient of correlation between \( X_j \) and \( X_k \). **Proof:** See Appendix.

Note that the variance in (2) is exact and fully observable—not estimated from sample data—because we can observe covariate values for every unit in the finite population. Thus, the variances and covariances \( \sigma^2_{X_j} \) and \( \sigma_{X_j,X_k} \) can be calculated exactly for all \( j \) and all \( k \) in a given data set. In brief, when treatment is assigned at random, the treatment and control groups can both be viewed as simple random samples from the finite population of \( N \) units. The expected mean of each covariate \( X_j \) is then the same in the treatment and control samples—namely, the average \( \overline{X_j} \) in the finite population. Moreover, the variance of the difference of means for each covariate is given by a formula that reflects both sampling without replacement from the finite population and the dependence between the treatment and control group means.

This is similar to the variance of an estimator of an average treatment effect under the Neyman approach; but unlike in that case, here there are no unobservable sample covariances because \( X_i \) is invariant to treatment assignment.

**3.6.2 A regression-weighted sum of covariate differences of means**

Now consider a more general weighted case. The test we propose has a ready interpretation that is easy to relate to standard unweighted balance tests: whereas \( \delta_{UW} \) is the unweighted sum of the differences of covariate means, here we propose a regression-weighted sum—i.e., the weighted sum of differences of covariate means, where the weights are standardized regression coefficients.

The weights are constructed from a sample analogue to the finite-population regression in (1). The average of the potential outcomes in the control group sample is

\[
\overline{Y(0)}^C = \overline{X}^C \hat{\beta}^C,
\]

where \( \overline{X}^C \) is the average value of the covariates in the control group and ‘ indicates the transpose (a \( 1 \times p \) vector), and the \( p \times 1 \) vector \( \hat{\beta}^C \) gives the coefficients of the regression fit in the control group sample (thus

\[
\text{That is, } \sigma^2_{X_j} = \frac{1}{N} \sum_{i=1}^{N} (X_{ij} - \overline{X_j})^2, \text{ and } \sigma_{X_j,X_k} = \frac{1}{N} \sum_{i=1}^{N} (X_{ij} - \overline{X_j})(X_{ik} - \overline{X_k}).
\]

\[
\text{See Neyman (1923); Freedman (2007: A32-A34); Samii and Aronow (2011, Theorem 2); Gerber and Green (2012: 57); Dunning (2012: 193).
\]

\[
\text{This is similar however to the variance of an estimator of an average treatment effect under a strict null hypothesis, i.e., } Y_i(1) = Y_i(0) \text{ for all } i.
\]

14
The left-hand side of (6) can be viewed as a regression-weighted estimator for the average potential outcome under control in the finite population, using well-known approaches from the theory of sampling from a finite population (Cochran (1977), Chapter 7). We cannot fit the analogous regression in the treatment group sample, since in the treatment group we do not observe potential outcomes under control. However, the treatment and control groups are exchangeable under random assignment, justifying the use of $\hat{\beta}^c$ in place of the coefficients from the analogous treatment group regression. Thus, we can estimate the average of the potential outcomes under control in the treatment group as

$$\hat{Y}(0)^T = X^T \cdot \hat{\beta}^c,$$

(7)

where the superscript “T” indicates treatment. We then subtract (6) from (7), giving

$$\hat{Y}(0)^T - \hat{Y}(0)^C = (X^T - X^C) \cdot \hat{\beta}^c.$$  

(8)

Equation (8) can equivalently be written as the regression-weighted (“RW”) sum of the covariate differences-of-means,

$$\delta_{RW} = \sum_{j=1}^{p} \hat{\beta}_j^c \delta_j,$$

(9)

where as before each $\delta_j$ is the difference of covariate means across the treatment and control groups, and $\hat{\beta}^c$ is as given in footnote 24. In contrast to $\delta_{UW}$ in (5), with $\delta_{RW}$ in (9) we weight each covariate difference of means by a measure of the covariate’s importance—i.e. the extent to which it is linearly prognostic.

**Theorem 3.** *(Distribution of the regression-weighted sum of covariate differences of means, $\delta_{RW}$)* When treatment is randomly assigned, $\text{plim}(\delta_{RW}) = 0.$ The large-sample variance of $\delta_{RW}$, conditional on the weights, is proportional to $\text{Var}(\delta_{UW})$ as given in Theorem 2. **Proof:** See Appendix.

In sum, our key test statistic $\delta_{RW}$ can be described as a weighted sum of individual covariate differences of means, where the weights are formed from the regression of potential outcomes on covariates in the control group sample. Thus, it has the form of the difference in equation (4). Imbalances in the multivariate distribution of $X$ in the treatment and control group may cause us to reject $H_0$; but unlike standard balance tests, in expectation irrelevant covariates (those for which $\beta_j$ is zero) will not figure in the sum of covariate differences.

Tests based on $\delta_{RW}$ should thus be more *specific* than standard balance tests: they should fail to reject when imbalances are due to irrelevant (non-prognostic) covariates. In contrast, covariates contribute more greatly to the weighted sum to the extent that they are more informative about potential outcomes. The regression-weighted tests we propose are therefore also responsive to imbalances on covariates that are important—i.e., prognostic. The test should therefore also be *sensitive*: it should reject null hypotheses in the sense of $H_0$ in (3) that are false.

---

24Thus,

$$\hat{\beta}^c = \left( \sum_{i=1}^{n_0} X_iX_i^\prime \right)^{-1} \sum_{i=1}^{n_0} X_iY_i(0).$$

is a $p \times 1$ vector with elements $\hat{\beta}_j$ for $j = 1, \ldots, p$. Here we index by $i = 1, \ldots, n_0$ the random subset of $n_0$ units sampled into the control group from the $N$ units in the finite population.
When $X$ is sufficient for $\{Y(0), Y(1)\}$ in the sense of Definition 1, the following observable implication holds.

**Theorem 4. (Observable Implications of As-If Randomization)**

Suppose that $X$ is sufficient for $\{Y(1), Y(0)\}$. Then:

$$Z \not\perp \perp \widehat{Y(Z)} | X \implies Z \not\perp \perp Y(Z)$$

where $\widehat{Y(Z)}$ is based on the sample analogue to the finite-population regression in (1). **Proof**: see Appendix.

This provides an empirical or observable corollary to Theorem (1): when $X$ is sufficient, we can use the regression-weighted statistic to test as-if random.

### 3.6.3 A permutation hypothesis test

Theorem 3 gives a large-sample result that may not be reliable in small samples, due to ratio-estimator bias (the sample regression estimator can be viewed as a ratio of random variables, since covariate values in the control group are random). To conduct hypothesis tests, we could form a $t$- or $z$-test based on $\delta/\sqrt{\text{Var}(\delta)}$, where $\delta$ is one of $\delta_{UW}$ or $\delta_{RW}$. However, we recommend a permutation test, which compares each observed $\delta$ with its randomization distribution. These design-based tests may be most reliable, especially given that large-sample approximations to the distribution of $\delta_{RW}$ may be unreliable in small studies.

To implement a permutation test, we take an observed (weighted or unweighted) sum of covariate differences of means, $\delta_{\text{obs}}$, in a given data set; randomly permute the treatment assignment labels $Z_i$, calculating $\delta^{*b}$ in each of $b \in \{1, \ldots, B\}$ permutations (where we can make $B$ arbitrarily large); and calculate a two-sided permutation-based $p$-value as

$$p^* = \frac{1}{B} \sum_{b=1}^{B} \mathbb{1}(|\delta^{*b}| \geq |\delta_{\text{obs}}|). \quad (10)$$

For the regression-weighted sum of covariate differences, for example, we would compare $\delta_{\text{obs}}^{RW}$ to its permutation distribution and reject the null hypothesis if a regression-weighted sum as extreme in absolute value as the observed $\delta_{\text{obs}}^{RW}$ would arise in fewer than 5% of random assignments.

### 4 Simulations: the power and specificity of the conditional balance test

We now turn to simulations to assess the performance of our weighted and unweighted measures of covariate balance under different degrees of prognosis and covariate imbalance.

**Step 1:** We start by simulating $N = 500$ observations. For each observation we generate a set of $p = 3$ covariates, $X_1$, $X_2$, and $X_3$, a treatment assignment vector $Z$—where half the units are assigned to either treatment or control—and outcomes $Y$. The variables are drawn from a multivariate standard normal distribution and we vary the degree of expected correlation between covariates and treatment.
assignment and between covariates and the potential outcomes. That is, when \( \text{corr}(X_p, Z) \neq 0 \), we have expected imbalance on \( X_p \); when \( \text{corr}(X_p, Y) \neq 0 \), then \( X_p \) is prognostic, with the correlation reflecting the degree of prognosis. Prognosis and imbalance induce correlation between \( Z \) and potential outcomes—and thus also bias in the treatment effect estimator \( \hat{ATE} \). We use this data set to calculate the observed \( \delta_{UW} \), \( \delta_{RW} \), as well as another well-known (unweighted) statistic used to test for multivariate equivalence, Hotelling’s \( T \).

**Step 2:** We calculate p-values for each observed test statistic by randomly permuting the labels of \( Z \) \( k = 500 \) times. A p-value is given by the analogue to [10]. We reject the null hypothesis when \( p^* < 0.05 \).

**Step 3:** We repeat Steps 1-2 above 1000 times for each simulated data generating process (i.e., each time producing a set of \( N = 500 \) observations with a given expected correlation structure). This allows us to calculate the rejection rate for a given degree of imbalance and prognosis on \( X_1 \). We also run simulations in which \( X_2 \) is imbalanced (but non-prognostic)\(^{25} \).

Here, an analogue to Type I error is given by the proportion of rejections when the draws are set with expected (a) \( \text{corr}(X_1, Z) = 0 \) and/or (b) \( \text{corr}(X_1, Y) = 0 \). In our framework, rejections in both cases constitute false negatives, because \( X_1 \) is not imbalanced in expectation or because it is not prognostic—in which case we do not wish to reject. Type II error, on the other hand, constitutes failure to reject when (a) and (b) are both false. Thus, our statistical power is given by the proportion of rejection in the latter cases.

Figure 2 plots results of a set of simulations where we vary (a) the expected correlation between \( X_1 \) and \( Z \)—\( \text{corr}(X_1, Z) \) (X1 imbalance)—and between \( X_1 \) and \( Y \)—\( \text{corr}(X_1, Y) \) (X1 prognosis), while \( X_2 \) and \( X_3 \) are random noise (independent of other variables), and (b) the expected correlation between \( X_2 \) and \( Z \)—\( \text{corr}(X_2, Z) \) (X2 imbalance)—and between \( X_1 \) and \( Y \)—\( \text{corr}(X_1, Y) \) (X1 prognosis), while \( X_3 \) is random noise (independent of other variables). The facets show increasing imbalance—i.e., the expected correlation between \( X_1 \) and \( Z \)—as we move from left to right and top to bottom in the figure. The horizontal axis of each facet plots prognosis—the expected correlation between \( X_1 \) and \( Y(0) \). Thus, in each facet, the origin corresponds to zero prognosis (no correlation between \( X_1/X_2 \) and \( Y(0) \)), and increases in increments of 0.05. Each facet then depicts, for a given dataset with a particular degree of prognosis (and given the degree of imbalance implied by the facet label), the proportion of rejections across the 1000 datasets generated with a particular correlation structure (where for each of the 1000 realizations of the data-generating process, we classify rejection according to the p-value from 500 permutations of treatment assignment). Thus, we plot the rejection rate associated with each of three estimators—our unweighted sum \( \delta_{UW} \), our regression-weighted sum \( \delta_{RW} \), and Hotelling’s \( T \) (solid and dashed curves). When there is imbalance and prognosis is non-zero, potential outcomes and treatment assignment are correlated in expectation and as-if random (i.e., Assumption 1) fails. Were we to fail to reject as-if random and calculate \( \hat{ATE} \) as the difference in mean outcomes across the treatment and control groups, we would expect bias. Thus, for each level of prognosis in each facet, we plot the standardized bias of \( \hat{ATE} \) in the simulation (black points).

The findings highlights several interesting insights. When imbalance is zero (top-left facet), the measures perform similarly; the regression-weighted sum rejects at a rate of approximately 0.05, indicating appropriate control over Type I error. We see greater divergence across the estimators as imbalance increases. Consider, for example, the facet with imbalance (expected correlation between \( X_1 \) and \( Z \) ) set to 0.2. At

\(^{25}\text{Simulations were run on UC Berkeley’s Savio cluster. The entire process runs in parallel on 24 CPU and takes on average 40 hours.}\)
Figure 2: We simulate an $N = 500$ data set specifying expected correlations between covariates $X_1$, $X_2$, and $X_3$ and a treatment vector $Z$ (covariate imbalance) and between each covariate and $Y(0)$ (covariate prognosis). We calculate the observed unweighted and regression-weighted sums of differences of covariate means and Hotelling’s $T$-statistic and obtain randomization inference $p$-values for each statistic by permuting treatment assignment $k = 500$ times. We repeat this process 1000 times to obtain rejection rates for each set of expected correlations. The figure displays rejection rates with imbalances on $X_1$ or $X_2$, across different values of $X_1$ prognosis ($X_2$ and $X_3$ are non-prognostic), along with the standardized bias of $ATE$. The pink regions correspond to $Z \perp \{Y(0), Y(1)\}$. 

Estimator --- Unweighted --- Regression-weighted --- Hotelling
the origin, as-if random holds, but the Hotelling $T$-statistic rejects with probability 1, while $\delta_{UW}$ rejects at rate 0.75. The regression-weighted sum $\delta_{RW}$ is substantially more specific, i.e. it results in a substantially lower false-positive rate of 50%. The regression-weighted estimator appears sensitive to imbalance on a non-prognostic covariate, but to a much lesser degree than the unweighted measures. Finally, note that as expected both of the unweighted measures are essentially invariant to the degree of prognosis—and thus to the failure of Assumption [1] and the presence of bias—while the regression-weighted estimator’s rejection rate increases with prognosis. In contrast, the regression-weighted estimator is sensitive to failures of as-if random.

In sum, the results suggest that the conditional balance test is both more sensitive and more specific. Compared to unweighted tests, it fails to reject when as-if random holds. With prognostic covariates, however, it rejects with higher probability when as-if random fails.

5 Discussion and conclusion

We propose that balance tests should be weighted by the prognostic power of the covariates being tested. We do this using a conditional balance test, in which potential outcomes are projected onto covariates. We propose as test statistic a weighted sum of the individual covariate differences of means across the treatment and control groups, where the weights are the standardized regression coefficients. Our theory and simulations suggest that the conditional balance test reduces false positive rejections at low levels of prognosis and increases power at low levels of imbalance.

The extent to which covariates are prognostic is therefore a critical consideration for balance testing. Absent prognosis, balance tests may be uninformative about failures of as-if random. Treatment assignment may depend on potential outcomes, yet balance tests with irrelevant covariates will fail to detect it. Conversely, imbalances on irrelevant covariates could lead to spurious rejects even when as-if random holds.

Some researchers conduct balance tests on lagged dependent variables, presuming perhaps—though this is often left implicit—that they are prognostic. This is generally an excellent practice; our theory provides a rationale for doing this in terms of the sufficiency of covariates. Yet, as our motivating example in Section 2 suggested, a lagged outcome may not in fact be prognostic, depending on the setting. More generally, the extent to which any covariate or set of covariates is in fact predictive of potential outcomes is an empirical question. Analysts should check and report diagnostics, such as the multiple $R^2$ from a regression of potential outcomes on covariates.

Our conditional balance test test produces a single $p$-value and is thus a global or omnibus test. This limits problems of discretion that can arise in the choice of metric for combining $p$-values from different tests. However, we suggest reporting separate covariate-by-covariate tests, as in often current standard practice, in addition to the global $p$-value from a conditional balance test. Like reporting the underlying components of an index, this will allow readers to continue to assess informally the overall weight of the evidence in support or against as-if random and boosts transparency, even as readers may perhaps give priority to the more powerful and specific global measure.

The research we present here also suggests several possible extensions. One mentioned in Section 3 is the use of non-linear approximations to $E(Y_i|X)$ in the finite population, such as lowess regressions. Our approach also has important connection to sensitivity analysis: variables that are related to potential outcomes—i.e. that have larger standardized $\beta_j$—are potentially confounders when estimating treatment effects, if they are also imbalanced across the treatment and control groups. Indeed, we can think about the
focus of the article in a slightly different way as well. Confounding is endemic in observational studies—but confounders must be associated with both a putative cause and an outcome. Existing balance tests look only at the association with treatment (the putative cause) but not with the outcome. They conclude from evidence of balance that confounding is not a problem (i.e., as-if random holds); or from evidence of imbalance that as-if random should be rejected. Yet as we show, this reasoning is misleading. We could find imbalance on irrelevant ("noise") covariates and yet as-if random could fail; or we could find balance on those covariates and yet treatment assignment could depend on potential outcomes. For these reasons, finding statistical balance on prognostic ("signal") covariates is encouraging—while imbalances suggest possible failures of the design and confounding selection biases. This line of thinking also provides a connection between our approach and various forms of sensitivity analysis, which can be further explored in future work.

For natural experiments and discontinuity designs, assessing as-if random prior to the estimation of treatment effects and thus of any statistical adjustment for confounders or any formal sensitivity analysis. Results from our routine can provide guidance regarding which covariates may need to be adjusted in estimating treatment effects, should the key identifying assumption fail. If powerful balance tests such as that we propose here provide evidence consistent with the assumption of independent of treatment assignment and potential outcomes, however, statistical adjustment and sensitivity tests become less relevant and the advantages of design-based analysis more powerful.
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# Appendix

## 6.1 Prognosis of lagged incumbency (Eggers et al.)

Table 1: Eggers et al.: Correlation between party vote shares at times $t$ and $t-1$ across election types (all)

| Country         | Office                                         | Corr.Y0_X |
|-----------------|------------------------------------------------|-----------|
| USA             | HOUSE OF REPRESENTATIVES, 1880-2010            | 0.852     |
| USA             | HOUSE OF REPRESENTATIVES, 1880-1944            | 0.8712    |
| USA             | HOUSE OF REPRESENTATIVES, 1946-2010            | 0.8339    |
| USA             | STATEWIDE                                      | 0.7465    |
| USA             | STATE LEGISLATURE                              | 0.7681    |
| USA             | MAYOR                                          | 0.6029    |
| CANADA          | COMMONS, 1867-2011                             | 0.7457    |
| CANADA          | COMMONS, 1867-1911                             | 0.5383    |
| CANADA          | COMMONS, 1921-2011                             | 0.7569    |
| UK              | HOUSE OF COMMONS                               | 0.8434    |
| UK              | LOCAL COUNCIL                                  | 0.7883    |
| GERMANY         | BUNDESTAG                                      | 0.9139    |
| GERMANY         | BAVARIA, MAYOR                                 | 0.4255    |
| FRANCE          | NATIONAL ASSEMBLY                              | 0.7019    |
| FRANCE          | MUNICIPALITY                                   | 0.6667    |
| AUSTRALIA       | HOUSE OF REPS, 1987-2007                       | 0.904     |
| NEW ZEALAND     | PARLIAMENT, 1949-1987                          | 0.8043    |
| INDIA           | LOWER HOUSE, 1977–2004                         | 0.4316    |
| BRAZIL          | MAYORS, 2000-2008                              | 0.0847    |
| MEXICO          | MAYORS, 1970-2009                              | 0.7465    |
| All COUNTRIES   | ALL RACES                                      | 0.7907    |
Table 2: Eggers et al.: Correlation between party vote shares at times t and t-1 across election types (RD study group with bandwidth 0.5—close winners and close losers)

| Country   | Office                          | Corr.Y0.X |
|-----------|---------------------------------|-----------|
| USA       | HOUSE OF REPRESENTATIVES, 1880-2010 | 0.1417    |
| USA       | HOUSE OF REPRESENTATIVES, 1880-1944 | 0.0649    |
| USA       | HOUSE OF REPRESENTATIVES, 1946-2010 | 0.2389    |
| USA       | STATEWIDE                       | -0.1045   |
| USA       | STATE LEGISLATURE               | 4.0e-04   |
| USA       | MAYOR                           | 0.0173    |
| CANADA    | COMMONS, 1867-2011              | -0.064    |
| CANADA    | COMMONS, 1867-1911              | -0.1625   |
| CANADA    | COMMONS, 1921-2011              | -0.0383   |
| UK        | HOUSE OF COMMONS                | 0.1764    |
| UK        | LOCAL COUNCIL                   | 0.0513    |
| GERMANY   | BUNDESTAG                       | -0.052    |
| GERMANY   | BAVARIA, MAYOR                  | -0.1254   |
| FRANCE    | NATIONAL ASSEMBLY               | -0.0647   |
| FRANCE    | MUNICIPALITY                    | 0.1305    |
| AUSTRALIA | HOUSE OF REPS, 1987-2007        | 0.2946    |
| NEW ZEALAND | PARLIAMENT, 1949-1987       | 0.3236    |
| INDIA     | LOWER HOUSE, 1977–2004         | -0.073    |
| BRAZIL    | MAYORS, 2000-2008               | -0.0487   |
| MEXICO    | MAYORS, 1970-2009               | 0.0065    |
| ALL COUNTRIES | ALL RACES                 | 0.0221    |

6.2 Proof of Theorem 1 ( Sufficiency of Covariates)

Theorem 1 states: Assume \( X \) is sufficient for \{\( Y(1), Y(0) \)\}. Then, \( Z \not\perp \perp X \implies Z \not\perp \perp \{Y(1), Y(0)\} \).

**Proof.** By Definition 1 if \( X \) is sufficient for \{\( Y(1), Y(0) \)\} then \( Z \perp \perp \{Y(1), Y(0)\} \| X \).

If \( X \) is sufficient for \{\( Y(1), Y(0) \)\} and \( X^S \not\perp \perp \{Y(1), Y(0)\} \) then \( \{Y(1), Y(0)\} \perp \perp X^S \| X \iff \sigma(X^S \cap \{Y(1), Y(0)\}) \subseteq \sigma(X) \implies X^S \subseteq X \). Hence,

\[
\begin{align*}
Z \not\perp X & \implies \\
Z \not\perp X^S & \implies \quad \text{(Since } X^S \subseteq X \text{)} \\
Z \not\perp X^S \cup X^N & \implies \quad \text{(By definition of } X^N \text{)} \\
Z \not\perp X^S & \implies \quad \text{(Since } X^N \perp \perp Z \text{)} \\
Z \not\perp \{Y(1), Y(0)\} & \\
\end{align*}
\]
6.3 Proof of Theorem 2 (distribution of $\delta_{UW}$)

We consider in turn the three claims in the theorem—i.e., regarding 1. the expectation, 2. the variance, and 3. asymptotic normality of the random variable $\delta_{UW}$.

**Proof.** 1. Each random variable $\delta_j$ can be written $(1/n_1)Z'X_j + (1/n_0)(1 - Z)X_j = X_j^T - X_j^C$, where $X_j^T$ and $X_j^C$ are the means of covariate $j$ in the treatment and control groups, respectively. Random assignment of the treatment implies that $Z_i \perp u_i$ for any fixed variate $u_i$, including each of the $X_j$'s. Viewed differently, the treatment and control groups are both simple random samples from the same underlying population. The expectations of the two sample means therefore coincide: $E(\delta_j) = E(X_j^T) - E(X_j^C) = 0$ for $j = 1, \ldots, p$. Thus, after distributing expectations, $E(\delta_{UW}) = E(\delta_1) + E(\delta_2) + \ldots E(\delta_p) = 0$.

2. Next, for the variance, consider as a preliminary two arbitrary features $(u_i, v_i)$ in the finite population $i = 1, \ldots, N$. Define the population variances as

$$
\sigma^2_u = \frac{1}{N} \sum_{i=1}^{N} (u_i - \bar{u})^2
$$

and

$$
\sigma^2_v = \frac{1}{N} \sum_{i=1}^{N} (v_i - \bar{v})^2,
$$

where $\bar{u} = 1/N \sum_{i=1}^{N} u_i$ and $\bar{v} = 1/N \sum_{i=1}^{N} v_i$ are the population means. The population covariance between these features is

$$
\sigma_{u,v} = \frac{1}{N} \sum_{i=1}^{N} (u_i - \bar{u})(v_i - \bar{v}).
$$

Let $\bar{U}_z$ denote the sample average in the treatment ($z = 1$) or control ($z = 0$) group, and similarly for $\bar{V}_z$. Here, $\bar{U}_z$ and $\bar{V}_z$ are random variables, due to randomness in $Z$. If we observe both $u_i$ and $v_i$ in the treatment sample, then we have

$$
\text{Cov}(\bar{U}_1, \bar{V}_1) = \frac{N - n_1}{N - 1} \frac{\sigma_{u,v}}{n_1} = \frac{n_0}{N - 1} \frac{\sigma_{u,v}}{n_1},
$$

since the features are drawn without replacement from a finite population of size $N$ [Cochran 1977, Theorem 2.3]. If we observe $u_i$ and $v_i$ in the control sample, then

$$
\text{Cov}(\bar{U}_0, \bar{V}_0) = \frac{N - n_0}{N - 1} \frac{\sigma_{u,v}}{n_1} = \frac{n_1}{N - 1} \frac{\sigma_{u,v}}{n_1}.
$$

(If $n_1 \neq n_0$, these theoretical covariances must be figured separately for the two groups).

If we observe $u_i$ only for $i$ in the treatment sample and $v_i$ only for $i$ in the control sample, the variances of the samples averages are

$$
\text{Var}(\bar{U}_1) = \frac{N - n_1}{N - 1} \frac{\sigma^2_u}{n_1} = \frac{n_0}{N - 1} \frac{\sigma^2_u}{n_1},
$$

and

$$
\text{Var}(\bar{U}_0) = \frac{N - n_0}{N - 1} \frac{\sigma^2_u}{n_1} = \frac{n_1}{N - 1} \frac{\sigma^2_u}{n_1}.
$$

26 Thus, $\bar{U}_1$ can be written as $\frac{1}{n_1} Z' u$ and $\bar{U}_0$ as $\frac{1}{n_0} (1 - Z)' v$, where $u$ is an $N \times 1$ vector collecting the $N$ values of $u_i$; we can write $\bar{V}_z$ similarly. This notation clarifies that randomness in the sample means depends on treatment assignment $Z$. 

26
and
\[ \text{Var}(\bar{V}_0) = \frac{N - n_0 \sigma_v^2}{N - 1} \frac{n_1}{n_0} = \frac{n_1}{N - 1} \sigma_v^2. \quad (17) \]

Using combinatorial calculations (see Freedman et al. 2007, A32-34 or Neyman et al. 1923), the covariance of the sample averages is
\[ \text{Cov}(\bar{U}_1, \bar{V}_0) = -\frac{\sigma_{u,v}}{N - 1}. \quad (18) \]

The variance of the difference of the sample means \( \bar{U}_1 - \bar{V}_0 \) is then
\[
\text{Var}(\bar{U}_1 - \bar{V}_0) = \text{Var}(\bar{U}_1) + \text{Var}(\bar{V}_0) - 2\text{Cov}(\bar{U}_1, \bar{V}_0) \\
= \frac{1}{N - 1} \left[ \frac{n_0 \sigma_u^2}{n_1} + \frac{n_1 \sigma_v^2}{n_0} + 2 \sigma_{u,v} \right] \\
= \frac{1}{N - 1} \left[ \frac{n_0^2 \sigma_u^2 + n_1^2 \sigma_v^2 + 2n_1(n_0) \sigma_{u,v}}{n_1(n_0)} \right], \quad (19)
\]
using (16), (17), and (18) in the second step. (For related derivations, see Neyman et al. 1923; Freedman et al. 2007, A32-A34; Samii and Aronow 2012, Theorem 2; Gerber and Green 2012, 57; or Dunning 2012, 193.)

With these preliminaries, we can derive the variance of the random variable \( \delta_{UW} \). We have
\[
\text{Var}(\delta_{UW}) = \text{Var}\left( \sum_{j=1}^{p} \delta_j \right) \\
= \sum_{j=1}^{p} \text{Var}(\delta_j) + 2 \sum_{j<k} \text{Cov}(\delta_j, \delta_k). \quad (20)
\]
First, \( \text{Var}(\delta_j) \) has the same form as the variance of \( \bar{U}_1 - \bar{V}_0 \) when \( u_i = v_i \) for all \( i \) (since \( X_{ji} \) has the same value whether unit \( i \) is assigned to the treatment or the control group). Using equation (19), we find
\[
\text{Var}(\delta_j) = \text{Var}(\bar{X}_{j1} - \bar{X}_{j0}) \\
= \frac{1}{N - 1} \left[ \frac{n_1 \sigma_{X_j}^2}{n_0} + \frac{(n_0) \sigma_{X_j}^2}{n_1} + 2 \sigma_{X_j}^2 \right] \\
= \frac{1}{N - 1} \left[ \frac{n_0^2 \sigma_{X_j}^2 + n_1^2 \sigma_{X_j}^2 + 2n_1(n_0) \sigma_{X_j}^2}{n_0(n_1)} \right] \\
= \frac{1}{N - 1} \left[ \frac{(n_0 + n_1)^2 \sigma_{X_j}^2}{n_0(n_1)} \right] \\
= \frac{1}{N - 1} \left[ \frac{N^2 \sigma_{X_j}^2}{n_0(n_1)} \right]. \quad (21)
\]

\(^{27}\)Following the previous note, the difference of means \( \bar{U}_1 - \bar{V}_0 \) can be written as \( \delta_{u,v} = \frac{1}{n_1} Z'u + \frac{1}{n_0} (1 - Z)'v \), where \( u \) and \( v \) are the \( N \times 1 \) vectors collecting the \( N \) values of \( u_i \) and \( v_i \), respectively.
Here, $\bar{X}_j$ indicates the sample average of $X_j$ in the treatment group and $\bar{X}_{j0}$ indicates the sample average in the control group. Also, $\sigma^2_{X_j}$ is with $u_j = X_{ij}$; it denotes the variance of the covariate $X_j$ calculated over all $N$ units in the finite population, that is,

$$
\sigma^2_{X_j} = \frac{1}{N} \sum_{i=1}^{N} (X_{ij} - \bar{X}_j)^2,
$$

(22)

where $\bar{X}_j$ is the mean of $X_j$ over the $N$ study units. Also, since the covariance of a variable with itself is its variance, $\text{Cov}(X_j, X_j) = \sigma^2_{X_j}$.

Thus, we can calculate an exact, fully observable sampling variance for each $\delta_j$. As under a strict null hypothesis, where one “sees” potential outcomes for unit $i$ in both treatment and control conditions (by the stipulation that $Y_i(1) = Y_i(0)$ for all $i$), here we observe covariate values $X_i$ under both treatment and control conditions, whether unit $i$ is in fact assigned to the treatment or the control group—because covariates are fixed values invariant to treatment assignment. Note also that $\sigma^2_k$ is fully observed because we see values of each covariate for every study unit. In sum, there are no terms in (21) or (30) that would need to be estimated from sample data: this exact variance is fully observable.

As for $\text{Cov}(\delta_j, \delta_k)$, we have

$$
\text{Cov}(\delta_j, \delta_k) = \text{Cov}(\bar{X}_j - \bar{X}_{j0}, \bar{X}_k - \bar{X}_{k0})
$$

$$
= \text{Cov}(\bar{X}_j, \bar{X}_k) - \text{Cov}(\bar{X}_j, \bar{X}_{k0}) - \text{Cov}(\bar{X}_{j0}, \bar{X}_k) + \text{Cov}(\bar{X}_{j0}, \bar{X}_{k0}).
$$

(23)

The first and fourth terms in (23) are the covariances of the sample averages of two features, both sampled without replacement from a finite population of size $N$. Using (14) and (15), we have

$$
\text{Cov}(\bar{X}_j, \bar{X}_k) = \frac{N - n_j}{N - 1} \frac{\sigma_{X_j, X_k}}{n_j} = \frac{n_k}{N - 1} \frac{\sigma_{X_j, X_k}}{n_j}
$$

(24)

and

$$
\text{Cov}(\bar{X}_{j0}, \bar{X}_k) = \frac{N - n_0}{N - 1} \frac{\sigma_{X_j, X_k}}{n_0} = \frac{n_1}{N - 1} \frac{\sigma_{X_j, X_k}}{n_0}.
$$

(25)

The second and third terms in (23) are instead the covariances of the sample averages of two features, one assigned to the treatment group and one assigned to the control group. Using (18), we have

$$
\text{Cov}(\bar{X}_j, \bar{X}_{k0}) = \text{Cov}(\bar{X}_{j0}, \bar{X}_k) = -\frac{1}{N - 1} \sigma_{X_j, X_k},
$$

(26)

where $\sigma_{X_j, X_k}$ is the population covariance given in (13), with the covariates $X_j$ and $X_k$ in place of $u$ and $v$.

---

28 We could write $\bar{X}_j = \frac{1}{n_j} Z_j^\prime X_j$ and $\bar{X}_{j0} = \frac{1}{n_0} (1 - Z_j^\prime) X_j$ to clarify dependence of the sample averages on the random assignment vector $Z$.

29 If $u_i = Y_i(1)$ is a potential outcome under treatment and $v_i = Y_i(0)$ is a potential outcome under control, then the random variable $\delta_{u,v}$ estimates the average treatment effect. Then $\text{Var}(\delta_{u,v})$ is the variance of $ATE$ under a strict null hypothesis of no unit-level effect.
Thus,

\[
\text{Cov}(\delta_j, \delta_k) = \frac{n_0 \sigma_{X_jX_k}}{N - 1} + \frac{2 \sigma_{X_jX_k}}{N - 1} + \frac{n_0 \sigma_{X_jX_k}}{n_0}
\]

\[
= \frac{\sigma_{X_jX_k}}{N - 1} \left[ \frac{n_0}{n_1} + 2 + \frac{n_1}{n_0} \right] \]

\[
= \frac{\sigma_{X_jX_k}}{N - 1} \left[ \frac{n_0^2}{n_1(n_0)} + \frac{2n_1(n_0)}{n_1(n_0)} + \frac{n_1^2}{n_1(n_0)} \right] \]

\[
= \frac{\sigma_{X_jX_k}}{N - 1} \left[ \frac{n_0^2}{n_1(n_0)} + 2n_1(n_0) + n_1^2 \right] \]

\[
= \frac{\sigma_{X_jX_k}}{N - 1} \left[ \frac{n_0(n_0 + n_1)^2}{n_1(n_0)} \right] \]

\[
= \frac{\sigma_{X_jX_k}}{N - 1} \left[ \frac{N_0^2}{n_1(n_0)} \right].
\]

Returning to (20) and substituting for \(\text{Var}(\delta_j)\) and \(\text{Cov}(\delta_j, \delta_k)\), we have

\[
\text{Var}(\delta_{UW}) = \text{Var} \left( \sum_{j=1}^{p} \delta_j \right)
\]

\[
= \sum_{j=1}^{p} \text{Var}(\delta_j) + 2 \sum_{j<k} \text{Cov}(\delta_j, \delta_k) \]

\[
= \sum_{j=1}^{p} \frac{1}{N - 1} \left[ \frac{N_0^2 \sigma_{X_j}^2}{n_0(n_1)} \right] + 2 \sum_{j<k} \frac{\sigma_{X_jX_k}}{N - 1} \left[ \frac{N_0^2}{n_1(n_0)} \right] \]

\[
= \frac{N_0^2}{N - 1} \left[ \frac{1}{n_0(n_1)} \sum_{j=1}^{p} \sigma_{X_j}^2 \right] + 2 \frac{N_0^2}{N - 1} \frac{1}{n_1(n_0)} \sum_{j<k} \sigma_{X_jX_k} \]

\[
= \frac{N_0^2}{N - 1} \left[ \frac{1}{n_0(n_1)} \sum_{j=1}^{p} \sigma_{X_j}^2 + 2 \sum_{j<k} \sigma_{X_jX_k} \right].
\]

Thus, data on \(p\) covariates for \(N\) units allows us to calculate the exact variance of the sum of the covariate differences of means. As with the variance of each \(\delta_j\), \(\text{Var}(\delta)\) is fully observable: it need not be estimated from sample data because \(\sigma_{X_j}^2\) and \(\sigma_{X_j} \sigma_{X_k}\) are both measurable from the covariate data for the \(N\) units in the population. Note also that here we assume that \(n_1\) and \(n_0\) are fixed, not random.\(^{30}\)

Note that when the covariate \(X_j\) is standardized as

\[
(X_{ij} - \bar{X}_j) / \sigma_j,
\]

\(^{30}\)This is standard in experimental analysis, where the group sizes are planned in advance of randomization; for a natural experiment, the assumption is more debatable. If the group sizes are random variables, ratio-estimator bias may arise for small samples, though with moderately large \(n_1\) and \(n_0\) the distinction should make little difference.
we find using (21) that
\[ \text{Var}(\delta_{j,\text{stand}}) = \frac{N^2}{N - 1} \left[ \frac{1}{n_0(n_1)} \right], \]  
(30)
and \( \sigma_{X_j,X_k} \) in (27) is \( \rho_{X_j,X_k} \), the correlation of \( X_j \) and \( X_k \). Then
\[ \text{Var}(\delta)_{UW,\text{stand}} = \frac{N^2}{N - 1} \frac{1}{n_0(n_1)} \left[ p + 2 \sum_{j<k} \rho_{X_j,X_k} \right], \]  
(31)

3. Finally, for the third claim in the theorem, note that under an appropriate central limit theorem (Erdős and Rényi 1959, Hájek 1960, Höglund 1978), the sampling distribution of each \( \delta_j \) and thus of \( \delta \) is asymptotically normal. It will be approximately normal in a finite study group if \( n_0 \) and \( n_1 \) are large or even moderately sized, and even more so if the variables \( X_j \) themselves have an approximately normal distribution. That each \( \delta_j \) is a difference of averages also helps foster approximate normality, even in small samples. In sum, \( \delta \sim N(0, \text{Var}(\delta)) \), where here \( \sim \) means “approximately distributed as,” which can aid hypothesis testing when justified.

### 6.4 Proof of Theorem 3 (distribution of \( \delta_{RW} \))

Consider the distribution of
\[ \delta_{RW} = (\bar{X}^T - \bar{X}^C)'\hat{\beta}^C, \]
that is,
\[ \hat{Y}(0)^T - \hat{Y}(0)^C, \]
the difference between the fitted average potential outcomes under control in the treatment and control groups.

By Slutsky’s theorem, it is immediate that \( \text{plim}(\delta_{RW}) = \text{plim}(\bar{X}^T - \bar{X}^C)'\hat{\beta}^C = \text{plim}(\bar{X}^T - \bar{X}^C)'\text{plim}(\hat{\beta}^C) = 0 \) when treatment is randomly assigned; in that case, the covariate means in the treatment and control groups are equal in expectation.

As for the conditional variance of \( \delta_{RW} \),
\[
\text{Var}(\delta_{RW} | \hat{\beta}) = \text{Var}(\sum_{j=1}^{p} \hat{\beta}_j^C \delta_j | \hat{\beta}) = \sum_{j=1}^{p} \text{Var}(\hat{\beta}_j^C \delta_j | \hat{\beta}) + 2 \sum_{j<k} \text{Cov}(\hat{\beta}_j^C \delta_j, \hat{\beta}_k^C \delta_k | \hat{\beta}) = \sum_{j=1}^{p} \hat{\beta}_j^C \text{Var}(\delta_j) + 2 \sum_{j<k} \hat{\beta}_j^C \hat{\beta}_k^C \text{Cov}(\delta_j, \delta_k) = \sum_{j=1}^{p} \hat{\beta}_j^C \text{Var}(\delta_j) + 2 \sum_{j<k} \hat{\beta}_j^C \hat{\beta}_k^C \text{Cov}(\delta_j, \delta_k),
\]
\[
= \sum_{j=1}^{p} \hat{\beta}_j^C \left[ \text{Var}(\delta_j) \right] + 2 \sum_{j<k} \hat{\beta}_j^C \hat{\beta}_k^C \text{Cov}(\delta_j, \delta_k)
\]
\[
= \sum_{j=1}^{p} \hat{\beta}_j^C \left[ \frac{N^2 \sigma_{X_j}^2}{N - 1} \frac{1}{n_0(n_1)} \right] + 2 \sum_{j<k} \hat{\beta}_j^C \hat{\beta}_k^C \sigma_{X_j,X_k} \frac{N^2}{N - 1} \frac{1}{n_0(n_1)},
\]
where in the final line we use (20) and (27). When the elements of $X$ are standardized, we have

$$\text{Var}(\delta_{RW,\text{stand}}\beta) = \sum_{j=1}^{p} \beta_{j}^2 \frac{1}{N-1} \left[ \frac{N^2}{n_0(n_1)} \right] + 2 \sum_{j<k} \beta_{j}^2 \beta_{k}^2 \rho_{X_j,X_k} \frac{N^2}{n_1(n_0)}$$

(32)

In sum, the conditional variance of $\delta_{RW}$ is proportional to the variance of $\delta_{UW}$, with constants of proportionality equal to the regression weights. With standardized regressions, terms for which the fitted regression coefficients approach zero tend to vanish.

6.5 Proof of Theorem 4 (conditional balance test)

The statement of Theorem 4 (Observable Implications of As-If Randomization): Suppose that $X$ is sufficient for $\{Y(1), Y(0)\}$. Then $Z \not\perp\!
\not\!
\perp \hat{Y}(Z)|X \implies Z \not\perp\!
\not\!
\perp Y(Z)$

Proof. Consider the estimator $\hat{Y}(Z)|X = \sum_j \beta_j^c X_j$, which is the sample analogue of $Y(Z)_l$, as defined in equation (1) for $Y(0)$; randomness in $\beta_j^c$ is induced by treatment assignment (see e.g. footnote 24). Define, for any $U \neq X$, a linear estimator $Y(Z)|X, U$ such as $\beta_j^c X_j + \gamma U$. By sufficiency, $\forall U \neq X, Y(Z) \perp\!
\perp U|X$. By the Frisch-Waugh-Lovell theorem (or “regression anatomy,” Angrist and Pischke (2009): 3.1.2), the coefficient on $U$ in the sample regression is

$$\gamma = \frac{\text{Cov}(Y(0), \tilde{U})}{\text{Var}(\tilde{U})},$$

(33)

where $\tilde{U}$ is the residual from the sample regression of $U$ on $X$ and we use $\tilde{}$ to denote the sample estimator.

By consistency of $\hat{Y}(Z)$ (from Theorem 3) and sufficiency, $E(\gamma) \to 0$, and

$$\lim_{n \to \infty} P\left( \left| \hat{Y}(Z)|X - Y(Z)|X, U \right| > \epsilon \right) \to 0$$

for arbitrarily small $\epsilon$.

Take $U$ to be $X^C$, the complement of $X$. Then we have

$$Z \not\perp\!
\not\!
\perp \hat{Y}(Z)|X \implies$$

$$Z \not\perp\!
\not\!
\perp \hat{Y}(Z)|X, X^C \implies$$

$$Z \not\perp\!
\not\!
\perp \hat{Y}(Z) \implies$$

$$Z \not\perp\!
\not\!
\perp Y(Z).$$