THE
QUARTERLY JOURNAL
OF ECONOMICS

CHANNELING FISHER: RANDOMIZATION TESTS AND THE STATISTICAL INSIGNIFICANCE OF SEEMINGLY SIGNIFICANT EXPERIMENTAL RESULTS[*]

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I follow R. A. Fisher's *The Design of Experiments* (1935), using randomization statistical inference to test the null hypothesis of no treatment effects in a comprehensive sample of 53 experimental papers drawn from the journals of the American Economic Association. In the average paper, randomization tests of the significance of individual treatment effects find 13% to 22% fewer significant results than are found using authors' methods. In joint tests of multiple treatment effects appearing together in tables, randomization tests yield 33% to 49% fewer statistically significant results than conventional tests. Bootstrap and jackknife methods support and confirm the randomization results. *JEL* Codes: C12, C90.

I. Introduction

In contemporary economics, randomized experiments are seen as solving the problem of endogeneity, allowing for the iden-

tification and estimation of causal effects. Randomization has an additional strength: it allows for the construction of tests that are exact, that is, with a distribution that is known no matter what the sample size or characteristics of the errors. Randomized experiments, however, rarely make use of such methods, largely only presenting conventional econometric tests using asymptotically accurate clustered/robust covariance estimates. In this article, I apply randomization tests to 53 randomized experiments, using them to construct counterparts to conventional tests of the significance of individual treatment effects, as well as tests of the combined significance of multiple treatment effects appearing together within regressions or in tables presented by authors. In tests of individual treatment effects, on average randomization tests reduce the number of significant results relative to those found by authors by 22% and 13% at the .01 and .05 levels, respectively. The reduction in rates of statistical significance is greater in higher dimensional tests. In joint tests of all treatment effects appearing together in tables, for example, on average randomization inference produces 49% and 33% fewer .01 and .05 significant results, respectively, than comparable conventional tests based on clustered/robust covariance estimates. Bootstrap and jackknife methods validate randomization results, producing substantial reductions in rates of statistical significance relative to authors' methods.

The discrepancy between the results reported in journals and those found in this article can be traced to leverage, a measure of the degree to which individual observations on right-hand-side variables take on extreme values and are influential. A concentration of leverage in a few observations makes coefficients and standard errors extremely volatile, as their value becomes dependent on the realization of a small number of residuals, generating t-statistic distributions with much larger tail probabilities than recognized by putative degrees of freedom and producing sizable size distortions. I find that the discrepancy between authors' results and those based on randomization, bootstrap, or jackknife inference are largely limited to the papers and regressions with concentrated leverage. The results presented by most authors in the first table of their main analysis are generally robust to the use of alternative inference procedures, but as the data are explored, through the subdivision of the sample or the interaction of treatment measures with nontreatment covariates, leverage becomes concentrated in a few observations

and large discrepancies appear between authors' results and those found using alternative methods. In sum, regression design is systematically worse in some papers, and systematically deteriorates within papers as authors explore their data, producing less reliable inference using conventional procedures.

Joint and multiple testing is not a prominent feature of experimental papers (or any other field in economics), but arguably it should be. In the average paper in my sample, 60% of regressions contain more than one reported treatment effect.[1] When a .01 significant result is found, on average there are 4.0 reported treatment effects (and additional unreported coefficients on treatment measures), but only 1.6 of these are significant. Despite this, only two papers report any *F*-tests of the joint significance of all treatment effects within a regression. Similarly, when a table reports a .01 significant result, on average there are 21.2 reported treatment effects and only 5.0 of these are significant, but no paper provides combined tests of significance at the table level. Authors explore their data, independently and at the urging of seminar participants and referees, interacting treatment with participant covariates within regressions and varying specifications and samples across columns in tables. Readers need assistance in evaluating the evidence presented to them in its entirety. Increases in dimensionality, however, magnify the woes brought on by concentrated leverage, as inaccuracies in the estimation of high-dimensional covariance matrices and extreme tail probabilities translate into much greater size distortions. One of the central arguments of this article is that randomization provides virtually the only reliable approach to accurate inference in high-dimensional joint- and multiple-testing procedures, as even other computationally intensive methods, such as the bootstrap, perform poorly in such situations.

Randomization tests have what some consider a major weakness: they provide exact tests but only of sharp nulls, that is, nulls that specify a precise treatment effect for each participant. Thus, in testing the null of no treatment effects, randomization inference does not test whether the average treatment effect is zero, but rather whether the treatment effect is zero for each and every participant. This null is not unreasonable, despite its

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1. In this article, I use the term *regression* to refer broadly to an estimation procedure involving dependent and independent variables that produces coefficients and standard error estimates. Most of these are ordinary least squares (OLS).

apparent stringency, as it merely states that the experimental treatment is irrelevant, a benchmark arguably worth examining and (hopefully) rejecting. The problem is that randomization tests are not necessarily robust to deviations away from sharp nulls, as in the presence of unaccounted-for heterogeneity in treatment effects they can have substantial size distortions (Chung and Romano 2013; Bugni, Canay, and Shaikh 2017). This is an important concern, but not one that necessarily invalidates this article's results or its advocacy of randomization methods. First, confirmation from bootstrap and jackknife results, which test average treatment effects, and the systematic concentration of differences in high-leverage settings support the interpretation that the discrepancies between randomization results and authors' methods have more to do with size distortions in the latter than in the former. Second, average treatment effects intrinsically generate treatment-dependent heteroskedasticity, which renders conventional tests inaccurate in finite samples as well. Although robust covariance estimates have asymptotically correct size, asymptotic accuracy in the face of average treatment effects is equally a feature of randomization inference, provided treatment is balanced or appropriately studentized statistics are used in the analysis (Janssen 1997; Chung and Romano 2013; Bugni, Canay, and Shaikh 2017). I provide simulations that suggest that in the face of heterogeneous treatment effects, t-statistic-based randomization tests provide size that is much more accurate than clustered/robust methods. Moreover, in high-dimensional tests randomization tests appear to provide the only basis for accurate inference, if only of sharp nulls.

This article takes well-known issues and explores them in a broad practical sample. Consideration of whether randomization inference yields different results than conventional inference is not new. Lehmann (1959) showed that in a simple test of binary treatment, a randomization t-test has an asymptotic distribution equal to the conventional t-test; Imbens and Wooldridge (2009) found little difference between randomization and conventional tests for binary treatment in a sample of eight program evaluations. The tendency of White's (1980) robust covariance matrix to produce rejection rates higher than nominal size was quickly recognized by MacKinnon and White (1985), while Chesher and Jewitt (1987) and Chesher (1989) traced the bias and volatility of these standard error estimates to leverage. This article links these literatures, finding that randomization and conventional

results are very similar in the low-leverage situations examined in earlier papers but differ substantially both in individual results and average rejection rates in high-leverage conditions, where clustered/robust procedures produce large size distortions. Several recent papers (Anderson 2008; Heckman et al. 2010; Lee and Shaikh 2014; List, Shaikh, and Xu 2016) have explored the robustness of individually significant results to step-down randomization multiple-testing procedures in a few experiments. This article, in contrast, emphasizes the differences between randomization and conventional results in joint and multiple testing and shows how increases in dimensionality multiply the problems and inaccuracies of inexact inferential procedures, making randomization inference a nearly essential tool in these methods. It also highlights the practical value of joint tests as an alternative approach with different power properties than multiple-testing procedures.

The article proceeds as follows. Section II explains the criteria used to select the 53-paper sample, which uses every paper revealed by a keyword search on the American Economic Association (AEA) website that provides data and code and allows for randomization inference. Section III provides background information in the form of a thumbnail review of the role of leverage in generating volatile coefficients and standard error estimates, the logic and methods of randomization inference, and the different emphasis of joint- and multiple-testing procedures. Section IV uses Monte Carlos to illustrate how unbalanced leverage produces size distortions using clustered/robust techniques, the comparative robustness of t-statistic-based randomization tests to deviations away from sharp nulls, and the expansion of inaccuracies in high-dimensional testing. Section V provides the analysis of the sample, producing the results mentioned above, and Section VI concludes with some suggestions for improved practice.

The results of this research are anonymized, as the objective of this article is to improve methods, not target individual results. Thus, no information can be provided in the article, public use files, or private discussion regarding the results for particular papers. For the sake of transparency, I provide code that shows how each paper was analyzed, but the reader eager to know how a particular paper fared will have to execute this code themselves. A Stata ado file, available on my website, calculates randomization p-values for most Stata estimation com-

mands, allowing users to call for randomization tests in their own research.

II. The Sample

My sample is based on a search on www.aeaweb.org using the abstract and title keywords "random" and "experiment" restricted to the *American Economic Review* (AER), *American Economic Journal* (AEJ): *Applied Economics* and AEJ: *Microeconomics* which yielded papers up through the March 2014 issue of the AER. I then dropped papers that:

- did not provide public use data files and Stata compatible do-file code;
- were not randomized experiments;
- did not have data on participant characteristics; or
- had no regressions that could be analyzed using randomization inference.

Public use data files are necessary to perform any analysis and I had prior experience with Stata, which is by far the most popular program in this literature. My definition of a randomized experiment excluded natural experiments (e.g., based on an administrative legal change) but included laboratory experiments (i.e., experiments taking place in universities or research centres or recruiting their subjects from such populations). The sessional treatment of laboratory experiments is not generally explicitly randomized, but when queried laboratory experimenters indicated that they believed treatment was implicitly randomized through the random arrival of participants to different sessions. The requirement that the experiment contain data on participant characteristics was designed to filter out a sample that used mainstream multivariate regression techniques with estimated coefficients and standard errors.

Not every regression presented in papers based on randomized experiments can be analyzed using randomization inference. To allow for randomization inference, the regression must contain a common outcome observed under different treatment conditions. This is often not the case. For example, if participants are randomly given different roles and the potential action space differs for the two roles (e.g., in the dictator-recipient game), there is no common outcome between the two groups that can be examined. In other cases, participants under different treatment

regimes do have common outcomes, but authors evaluate each treatment regime using a separate regression, without using any explicit inferential procedure to compare differences. One could, of course, develop appropriate conventional and randomization tests by stacking the regressions, but this involves an interpretation of the authors' intent in presenting the side-by-side regressions, which could lead to disputes. I make it a point to adhere to the precise specification presented in tables.

Within papers, regressions were selected if they allow for and do not already use randomization inference and:[2]

- appear in a table and involve a coefficient estimate and standard error;
- pertain to treatment effects and not to an analysis of randomization balance, sample attrition, or nonexperimental cohorts;

while tests were done on the null that:

- randomized treatment has no effect, but participant characteristics or other nonrandomized treatment conditions might have an influence.

In many tables, means are presented, without standard errors or *p*-values, that is, without any attempt at statistical inference. I do not test these. Variations on regressions presented in tables are often discussed in surrounding text, but interpreting the specification correctly without the aid of the supplementary information presented in tables is extremely difficult because there are often substantial do-file inaccuracies. Consequently, I limited myself to specifications presented in tables. Papers often include tables devoted to an analysis of randomization balance or sample attrition, with the intent of showing that treatment was uncorrelated with either. I do not include any of these in my analysis. Similarly, I drop regressions projecting the behavior of nontreatment cohorts on treatment measures, which are typically used by authors to reinforce the internal validity of the experiment. In difference-in-difference equations, I only test the treatment coefficients associated with differences during the treatment period. I test, universally, the null of no randomized treatment effect, including

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2. One paper used randomization inference throughout and was dropped, and five others present some randomization-based exact (e.g., Wilcoxon rank sum) tests.

treatment interactions with other covariates, while allowing participant characteristics or nonrandomized experimental treatment to influence behavior. For example, in the regression

$$y = \alpha + \beta_T T + \beta_{\text{age}}\text{age} + \beta_{T^*\text{age}}T^*\text{age} + \beta_{\text{convex}}\text{convex}$$

$$(1) \qquad + \beta_{T^*\text{convex}}T^*\text{convex} + \varepsilon,$$

where T is a randomized treatment measure, age is participant age, and convex is a nonrandomized payment scheme introduced in later rounds of an experiment, I rerandomize the allocation of T, repeatedly recalculating T^*age and T^*convex, and use the distribution of test statistics to test the null that T, T^*age, and T^*convex have 0 effects on all participants.

I was able to analyze almost all papers and regressions that met the sample selection guidelines described above. The do files of papers are often inaccurate, producing regressions that are different from those reported in the published paper, but an analysis of the public use data files generally allows one to arrive at a specification that reproduces the published results within a small margin of error on coefficients and standard errors. There are only a handful of regressions, in four papers, that could not be reproduced and included in the sample. To permute the randomization outcomes of a paper, one needs information on stratification (if any was used) and the code and methods that produced complicated treatment measures distributed across different data files. I have called on a large number of authors who have generously answered questions and provided code to identify strata, create treatment measures, and link data files. Knowing no more than that I was working on a paper on experiments, these authors displayed an extraordinary degree of scientific openness and integrity. Only two papers, and an additional segment from another, were dropped from my sample because authors could not provide the information necessary to rerandomize treatment outcomes.

Table I summarizes the characteristics of my final sample, after reduction based on the criteria described above. I examine 53 papers, 14 of which are laboratory experiments and 39 of which are field experiments. Twenty-seven of the papers appeared in the AER, 21 in the AEJ: *Applied Economics*, and 5 in the AEJ: *Microeconomics*. The number of tables reporting estimates and standard errors for treatment effects that I am able to analyze using randomization inference varies substantially across papers,

TABLE I

CHARACTERISTICS OF THE SAMPLE

53 Papers					1,780 Regressions	
			Treatment Coefficients			
Location	Journal	Tables	Reported	Unreported	Method	Covariance
39 Field	27 AER	17 1–2	17 2–30	41 0	0.67 OLS	0.25 default
14 Lab	26 AEJ	17 3–4	18 32–80	7 1–48	0.22 MLE	0.70 cl/robust
		19 5–8	18 90–260	5 76–744	0.11 other	0.04 bootstrap
						0.02 other

Notes. For papers, numbers reported are number of papers by characteristic. For regressions, numbers reported are the average across papers of the share of regressions within each paper with the noted characteristic.

with 17 papers having only one or two such tables and 19 presenting five to eight. The number of coefficients reported in these tables varies even more, with one paper reporting 260 treatment coefficients and another only 2. I deal with the heterogeneity in the number of treatment results by adopting the convention of always reporting the average across papers of the within-paper average measure, so each paper, regardless of the number of coefficients, regressions, or tables, carries an equal weight in summary statistics. Although most papers report all of the treatment effects in their estimating equations, some do not, and the number of such unreported auxiliary coefficients ranges from 1 to 48 in seven papers to 76 to 744 in five papers. To avoid the distracting charge that I tested irrelevant treatment characteristics, I restrict the analysis that follows to reported coefficients. Results that include unreported treatment effects, in the Online Appendix, exhibit very much the same patterns.

My sample contains 1,780 regressions, broadly defined as a self-contained estimation procedure with dependent and independent variables that produces coefficient estimates and standard errors. In the average paper, 67% of these are OLS regressions (including weighted), 22% involve maximum likelihood estimation (MLE; mostly discrete choice models), and the remaining 11% include handfuls of quantile regressions, two-step Heckman models, and other methods. In the typical paper, 25% of regressions make use of Stata's default (i.e., homoskedastic) covariance matrix calculation, 70% avail themselves of clustered/robust estimates of covariance, 4% use the bootstrap, and the remaining 2% use hc2/hc3 type corrections of clustered/robust
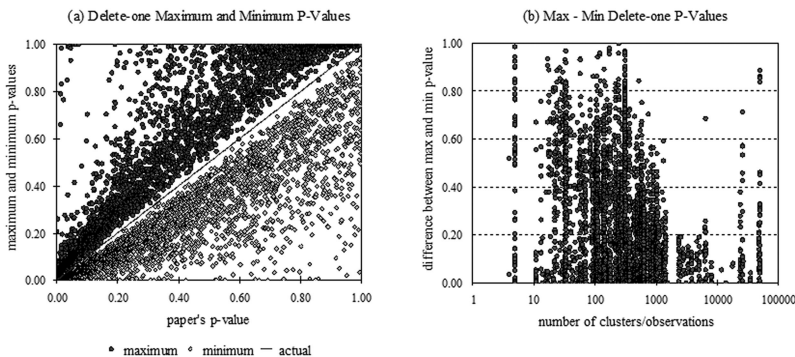
FIGURE I

Sensitivity to Outliers

covariance estimates. In 171 regressions in 12 papers (8 lab, 4 field), treatment is applied to groups, but the authors do not cluster or systematically cluster at a lower level of aggregation. This is not best practice, as correlations between the residuals for individuals playing games together in a lab or living in the same geographical region are quite likely. By clustering at below treatment level, these authors treat the grouping of observations in laboratory sessions or geographical areas as nominal. In implementing randomization, bootstrap, and jackknife inference in this article, I defer to this judgment, randomizing and sampling at the level at which they clustered (or didn't), treating the actual treatment grouping as irrelevant. Results with randomization and sampling at the treatment level, reported in the Online Appendix, find far fewer significant treatment effects.[3]

III. ISSUES AND METHODS

III.A. *Problems of Conventional Inference in Practical Applications*

One of the central characteristics of my sample is its remarkable sensitivity to outliers. Figure I, Panel A plots the maximum

3. In another three papers, the authors generally cluster at treatment level but fail to cluster some regressions. I randomize these at the treatment level so as to calculate the joint distribution of coefficients across equations. In three papers where the authors cluster across treatment groupings, I rerandomize at the treatment level.

and minimum coefficient p-values, using authors' methods, found when one deletes one cluster or observation at a time from each regression in my sample against the p-value found with the full sample.[4] With the removal of just one observation, 35% of .01-significant reported results in the average paper can be rendered insignificant at that level. Conversely, 16% of .01-insignificant reported results can be found to be significant at that level. Figure I, Panel B graphs the difference between these maximum and minimum p-values against the number of clusters/observations in the regression. In the average paper the mean difference between the maximum and minimum delete-one p-values is .23. To be sure, the problem is more acute in smaller samples, but surprising sensitivity can be found in samples with 1,000 clusters or observations and even in those with more than 50,000 observations.

A few simple formulas identify the sources of delete-one sensitivity. In OLS regressions, which make up much of my sample, the coefficient estimate with observation i removed ($\hat{\beta}_{-i}$) is related to the coefficient estimate from the full sample ($\hat{\beta}$) through the formula:

$$(2) \qquad \hat{\beta}_{-i} = \hat{\beta} - \frac{\tilde{x}_i}{\sum_i \tilde{x}_i^2} \frac{\hat{\varepsilon}_i}{(1 - h_{ii})},$$

where $\tilde{x}_i$ denotes the ith residual from the projection of independent variable $\mathbf{x}$ on the other regressors in the $n \times k$ matrix of regressors $\mathbf{X}$, $\hat{\varepsilon}_i$ the ith residual of the full regression, and h_{ii}, commonly known as leverage, is the ith diagonal element of the hat matrix $\mathbf{H} = \mathbf{X}(\mathbf{X}'\mathbf{X})^{-1}\mathbf{X}'$.[5] The robust variance estimate can be expressed as:

$$(3) \qquad \frac{1}{\sum \tilde{x}_i^2} \sum \tilde{h}_{ii} \left(\hat{\varepsilon}_i^2 \frac{n}{n-k} \right), \text{ where } \tilde{h}_{ii} = \frac{\tilde{x}_i^2}{\sum_i \tilde{x}_i^2}.$$

4. Where authors cluster, I delete clusters; otherwise I delete individual observations.

5. So called because $\hat{\mathbf{y}} = \mathbf{H}\mathbf{y}$. The formula for the deletion of vector $\mathbf{i}$ of clustered observations is $\hat{\beta}_{-\mathbf{i}} = \hat{\beta} - \frac{\tilde{\mathbf{x}}_{\mathbf{i}}'(\mathbf{I}_{\mathbf{i}} - \mathbf{H}_{\mathbf{ii}})^{-1}\hat{\varepsilon}_{\mathbf{i}}}{(\tilde{\mathbf{x}}'\tilde{\mathbf{x}})}$. When the coefficient on a variable is determined by an individual observation or cluster, h_{ii} equals 1 or (in the cluster case) $\mathbf{I}_{\mathbf{i}} - \mathbf{H}_{\mathbf{ii}}$ is singular. In this case, the delete-i formula for the remaining coefficients calculates $\mathbf{H}_{\mathbf{ii}}$ using the residuals of the remaining regressors projected on the variable in question.

$\tilde{h}_{ii}$ might be called coefficient leverage, because it is the ith diagonal element of the hat matrix $\tilde{\mathbf{H}} = \tilde{\mathbf{x}}(\tilde{\mathbf{x}}'\tilde{\mathbf{x}})^{-1}\tilde{\mathbf{x}}'$ for the partitioned regression. As seen in expressions (2) and (3), when coefficient leverage is concentrated in a few observations, coefficient and standard error estimates, depending on the realization of residuals, are potentially sensitive to the deletion of those observations.

Sensitivity to a change in the sample is an indication that results are dependent on the realizations of a small set of disturbances. In non-i.i.d. settings, this translates into inaccurate inference for a *given* sample, the object of interest in this article. The summation in expression (3) is a weighted average as $\tilde{h}_{ii}$ varies between 0 and 1 and sums to 1 across all observations. With concentrated leverage, robust standard error estimates depend heavily on a small set of stochastic disturbances and become intrinsically more volatile, producing t-statistic distributions that are more dispersed than recognized by nominal degrees of freedom. When the effects of right-hand-side variables are heterogeneous, the residuals have a heteroskedastic variance that is increasing in the magnitude of the regressor. This makes the robust standard error even more volatile, as it now places a disproportionate weight on disproportionately volatile residuals. Concentrated leverage also shrinks estimated residuals, as coefficient estimates respond to the realization of the disturbance, so a heavy weight is placed on residuals that are biased toward 0, biasing the standard error estimate in the same direction.[6] Thus, the estimates of the volatility of coefficients and of the volatility of the standard error are both biased downward, producing t-statistic distributions with underappreciated tail probabilities.

Table II reports the total coefficient leverage accounted for by the clusters or observations with the largest leverage in my sample. I calculate the observation-level shares $\tilde{h}_{ii}$, sum across observations within clusters if the regression is clustered, and then report the average across papers of the within-paper mean share of the cluster/observation with the largest coefficient leverage ("max") and the total leverage share accounted for by the largest 1st, 5th, and 10th percentiles of the distribution. I include measures for non-OLS regressions in these averages as well, because all of these contain a linear $\mathbf{x}_i'\boldsymbol{\beta}$ term and leverage plays a similar

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6. As an extreme example, when coefficient leverage for observation i is 1, $\hat{y}_i = y_i$, the estimated residual for i is always 0 and the robust standard error estimate for the coefficient is 0 as well.

TABLE II

SHARES OF COEFFICIENT LEVERAGE FOR REPORTED TREATMENT EFFECTS

	With authors' covariates				Without covariates			
	Max	1%	5%	10%	Max	1%	5%	10%
All 53 papers	0.058	0.091	0.216	0.338	0.057	0.089	0.217	0.345
Low leverage (18 papers)	0.008	0.043	0.167	0.290	0.007	0.038	0.159	0.281
Medium leverage (17 papers)	0.030	0.070	0.200	0.322	0.029	0.070	0.207	0.338
High leverage (18 papers)	0.134	0.158	0.279	0.402	0.132	0.157	0.283	0.414
First table (45 papers)	0.031	0.064	0.183	0.302	0.027	0.058	0.173	0.293
Other tables (45 papers)	0.049	0.085	0.215	0.341	0.050	0.084	0.214	0.342
With interactions (29 papers)	0.054	0.109	0.268	0.411	0.062	0.120	0.303	0.462
Without interactions (29 papers)	0.036	0.065	0.176	0.297	0.028	0.053	0.150	0.264

Notes. Figures are the average across papers of the within-paper average measure for reported coefficients. Max, 1%, 5%, and 10% = cumulative leverage share of clusters/observations with the largest leverage, ranging from the observation with the maximum through the 1st, 5th, and 10th percentiles of the distribution; "without covariates" = leverage shares with nontreatment covariates other than the constant term excluded from the regression.

role in their standard error estimates.[7] As shown in the table, in the mean paper the largest cluster/observation has a leverage of 0.058, while the top 1st and 10th percentiles account for 0.091 and 0.338 of total leverage, respectively. These shares vary substantially by paper. Dividing the sample into thirds based on the average within-paper share of the maximal cluster/observation, one sees that in the low-leverage third the average share of this cluster/observation is 0.008, whereas in the high-leverage third it is 0.134 (with a mean as high as 0.335 in one paper).

Table II also compares the concentration of leverage in the first table where authors present their main results against later tables, in papers that have more than one table reporting treatment effects.[8] Leverage is more concentrated in later tables, as authors examine subsets of the sample or interact treatment with nontreatment covariates. Specifically comparing coefficients appearing in regressions where treatment is interacted with covariates against those where it is not, in papers that contain both types of regressions, we see that regressions with covariates have

7. Thus, the often used robust covariance for matrix maximum likelihood models can be re-expressed as $\mathbf{ARA}'$, where, with $\mathbf{D}_1$ and $\mathbf{D}_2$ denoting diagonal matrices of the observational-level 1st and 2nd derivatives of the ln-likelihood with respect to $\mathbf{x}_i{}'\boldsymbol{\beta}$, $\mathbf{R} = (\mathbf{X}'\mathbf{X})^{-1}(\mathbf{X}'\mathbf{D}_1\mathbf{D}_1\mathbf{X})(\mathbf{X}'\mathbf{X})^{-1}$ and $\mathbf{A} = (-\mathbf{X}'\mathbf{D}_2\mathbf{X})^{-1}\mathbf{X}'\mathbf{X}$. With $\mathbf{D}_1$ serving as the residual, leverage plays the same role in determining the elements of $\mathbf{R}$ as it does in the OLS covariance estimate.

8. Main results are identified as the first section with this title (a frequent feature) or a title describing an outcome of the experiment (e.g., "Does treatment-name affect interesting-outcome?").

more concentrated leverage. The presence of nontreatment covariates in the regression per se, however, does not have a very strong effect on coefficient leverage, as the table shows by recalculating treatment coefficient leverage shares with nontreatment covariates excluded from the regression (but treatment interactions with covariates retained). This is to be expected if covariates are largely orthogonal to treatment.

A few examples illustrate how regression design can lead to concentrated leverage. Binary treatment applied 50/50 to the whole sample, with otherwise only a constant term in the regression, produces uniform leverage. Apply three binary treatments and control each to one-quarter of the population, and in a joint regression with a constant term each treatment arm concentrates the entirety of leverage in one-half of the observations. The clustered/robust covariance estimate is now based on only half of the residuals and consequently has a volatility (degrees of freedom) consistent with half the sample size. As is often done, run the regression using only one of the three treatment measures as a right-hand-side variable, so that binary treatment in the regression is applied in 25/75 proportions, and one-quarter of observations account for three-quarters of leverage. Apply 50/50 binary treatment, and create a second treatment measure by interacting it with a participant characteristic that rises uniformly in even discrete increments within treatment and control, and one-fifth of observations account for about three-fifths of coefficient leverage for the binary treatment measure (even without the nontreatment characteristic in the regression). Seemingly innocuous adjustments in regression design away from the binary 50/50 baseline generate substantially unbalanced leverage, producing clustered/robust covariance estimates and t-statistics that are much more dispersed than recognized.

III.B. Randomization Statistical Inference

Randomization inference provides exact tests of sharp (i.e., precise) hypotheses no matter what the sample size, regression design, or characteristics of the disturbance term. The typical experimental regression can be described as $\mathbf{y}_E = \mathbf{T}_E \boldsymbol{\beta_t} + \mathbf{X} \boldsymbol{\beta_x} + \boldsymbol{\varepsilon}$, where $\mathbf{y}_E$ is the $n \times 1$ vector of experimental outcomes, $\mathbf{T}_E$ an $n \times t$ matrix of treatment variables (including possibly interactions with nontreatment covariates), and $\mathbf{X}$ an $n \times k$ matrix of nonrandomized covariates. Conventional econometrics describes

the statistical distribution of the estimated βs as coming from the stochastic draw of the disturbance term ε, and possibly the regressors, from a population distribution. In contrast, in randomization inference the motivating thought experiment is that, given the sample of experimental participants, the only stochastic element determining the realization of outcomes is the randomized allocation of treatment. For each participant, the observed outcome y_i is conceived as a determinate function of the treatment $\mathbf{t}_i$ allocated to that participant, $y_i(\mathbf{t}_i)$. Consequently, the known universe of potential treatment allocations determines the statistical distribution of the estimated βs and can be used to test sharp hypotheses that precisely specify the treatment effect for each participant, because sharp hypotheses of this sort allow the calculation of what outcomes would have been for any potential random allocation of treatment. Consider, for example, the null hypothesis that the treatment effects in the equation above equal β_0 for all participants. Under this null, the outcome vector that would have been observed had the treatment allocation been $\mathbf{T}_S$ rather $\mathbf{T}_E$ is given by $\mathbf{y}_S = \mathbf{y}_E - \mathbf{T}_E\beta_0 + \mathbf{T}_S\beta_0$ and this value, along with $\mathbf{T}_S$ and the invariant characteristics $\mathbf{X}$ can be used to calculate estimation outcomes under treatment allocation $\mathbf{T}_S$.[9]

An exact test of a sharp null is constructed by calculating possible realizations of a test statistic and rejecting if the observed realization in the experiment itself is extreme enough. In the typical experiment there is a finite set $\mathbf{\Omega}$ of equally probable potential treatment allocations $\mathbf{T}_S$. Let $f(\mathbf{T}_E)$ denote a test statistic calculated using the treatment applied in the experiment and $f(\mathbf{T}_S)$ the known (under the sharp null) value the statistic would have taken if the treatment allocation had been $\mathbf{T}_S$. If the total number of potential treatment allocations in $\mathbf{\Omega}$ is M, the p-value of the experiment's test statistic is given by:

$$\text{randomization } p\text{-value} = \frac{1}{M} \sum_{S=1}^{M} I_S\left(> \mathbf{T}_E\right)$$

$$(4) \qquad\qquad + U * \frac{1}{M} \sum_{S=1}^{M} I_S\left(= \mathbf{T}_E\right),$$

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9. Imbens and Rubin (2015) provide a thorough presentation of inference using randomized experiments, contrasting and exploring the Fisherian potential outcomes and Neymanian population sampling approaches.

where $I_S(>\mathbf{T}_E)$ and $I_S(=\mathbf{T}_E)$ are indicator functions for $f(\mathbf{T}_S) > f(\mathbf{T}_E)$ and $f(\mathbf{T}_S) = f(\mathbf{T}_E)$, respectively, and U is a random variable drawn from the uniform distribution. In words, the *p*-value of the randomization test equals the fraction of potential outcomes that have a more extreme test statistic added to the fraction that have an equal test statistic times a uniformly distributed random number. In the Online Appendix, I prove that this *p*-value is uniformly distributed, that is, the test is exact with a rejection probability equal to the nominal level of the test.

Calculating equation (4), evaluating $f(\mathbf{T}_S)$ for all possible treatment realizations in $\mathbf{\Omega}$, is generally impractical. However, under the null random sampling with replacement from $\mathbf{\Omega}$ allows the calculation of an equally exact *p*-value provided the original treatment result is automatically counted as a tie with itself. Specifically, with N additional draws (beyond the original treatment) from $\mathbf{\Omega}$, the *p*-value of the experimental result is given by:

$$\text{sampling randomization } p\text{-value} = \frac{1}{N+1} \sum_{S=1}^{N} I_S\left(> \mathbf{T}_E\right)$$

$$(5) \hspace{2cm} + U * \frac{1}{N+1} \left[1 + \sum_{S=1}^{N} I_S\left(= \mathbf{T}_E\right)\right].$$

In the Online Appendix, I show that this *p*-value is uniformly distributed regardless of the number of draws N used in its evaluation.[10] This establishes that size always equals nominal value, even though the full distribution of randomization outcomes is not calculated. However, provided it is a concave function of the nominal size of the test, power is increasing in N (Jockel 1986). Intuitively, as the number of draws increases, the procedure is better able to identify what constitutes an outlier outcome in the distribution of the test statistic $f()$. In my analysis, I use 10,000 draws to evaluate equation (5). When compared with results calculated with fewer draws, I find no appreciable change in rejection rates beyond 2,000 draws.

One drawback of randomization inference, easily missed in the short presentation above, is that in equations with multiple

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10. The proof is a simple extension of Jockel's (1986) result for nominal size equal to a multiple of $\frac{1}{N+1}$. It generalizes to treatment allocations that are not equally probable by simply duplicating each treatment outcome in $\mathbf{\Omega}$ according to its relative frequency, so that each element in $\mathbf{\Omega}$ becomes equally likely.

treatment measures the *p*-value of the null for one coefficient generally depends on the null assumed for other treatment measures, as these nulls influence the outcome $\mathbf{y}_S$ that would have been observed for treatment allocation $\mathbf{T}_S$. It is possible in some multitreatment cases to calculate *p*-values for individual treatment measures that do not depend on the null for other treatments by considering a subset of the universe of potential randomization allocations that holds other treatments constant.[11] Such calculations, however, must be undertaken with care, as there are many environments where it is not possible to conceive of holding one treatment measure constant while varying another.[12] In results reported in this article, I always test the null that all treatment effects are 0 and all reported *p*-values for joint *or* individual test statistics are under that joint null. In the Online Appendix I calculate, where possible, alternative *p*-values for individual treatment effects in multitreatment equations that do not depend on the null for other treatment measures. On average, the results are less favorable to my sample (i.e., reject less often and produce bigger *p*-value changes).

I make use of two randomization-based test statistics, which find counterparts in commonly used bootstrap tests. The first is based on a comparison of the Wald statistics of the conventional tests of the significance of treatment effects, as given by $\hat{\boldsymbol{\beta}}_{\mathbf{t}}'(\mathbf{T}_S)\mathbf{V}(\hat{\boldsymbol{\beta}}_{\mathbf{t}}(\mathbf{T}_S))^{-1}\hat{\boldsymbol{\beta}}_{\mathbf{t}}(\mathbf{T}_S)$, where $\hat{\boldsymbol{\beta}}_{\mathbf{t}}$ and $\mathbf{V}(\hat{\boldsymbol{\beta}}_{\mathbf{t}})$ are the regression's treatment coefficients and the estimated covariance matrix of those coefficients. This method in effect calculates the probability

$$(6) \qquad \hat{\boldsymbol{\beta}}_{\mathbf{t}}'(\mathbf{T}_S)\mathbf{V}(\hat{\boldsymbol{\beta}}_{\mathbf{t}}(\mathbf{T}_S))^{-1}\hat{\boldsymbol{\beta}}_{\mathbf{t}}(\mathbf{T}_S) \geqslant \hat{\boldsymbol{\beta}}_{\mathbf{t}}'(\mathbf{T}_E)\mathbf{V}(\hat{\boldsymbol{\beta}}_{\mathbf{t}}(\mathbf{T}_E))^{-1}\hat{\boldsymbol{\beta}}_{\mathbf{t}}(\mathbf{T}_E)$$

I use the notation ($\mathbf{T}_S$) to emphasize that both the coefficients and covariance matrix are calculated for each realization of the ran-

11. Consider the case with control and two mutually exclusive treatment regimes denoted by the dummy variables T_1 and T_2. Holding the allocation of T_2 constant (for example), one can rerandomize T_1 across those who received T_1 or control, modifying $\mathbf{y}$ for the hypothesized effects of T_1 only, and calculate a *p*-value for the effect of T_1 that does not depend on the null for T_2.

12. Consider, for example, the case of treatment interactions with covariates (which arises frequently in my sample), as in the equation $y = \alpha + \beta_T T + \beta_{age}age + \beta_{T^*age}T^*age + \varepsilon$. It is not possible to rerandomize T holding T^*age constant, or to change T^*age while holding T constant, so there is no way to calculate a *p*-value for either effect without taking a stand on the null for the other.

dom draw $\mathbf{T}_S$ from $\mathbf{\Omega}$. This test might be called the randomization-t, as in the univariate case it reduces to a comparison of squared t-statistics. It corresponds to bootstrap tests based on the percentiles of Wald statistics.

An alternative test of no treatment effects is to compare the relative values of $\hat{\boldsymbol{\beta}}_t'(\mathbf{T}_S)\mathbf{V}(\hat{\boldsymbol{\beta}}_t(\mathbf{\Omega}))^{-1}\hat{\boldsymbol{\beta}}_t(\mathbf{T}_S)$, where $\mathbf{V}(\hat{\boldsymbol{\beta}}_t(\mathbf{\Omega}))$ is the covariance of $\hat{\boldsymbol{\beta}}_t$ across the universe of potential treatment draws in $\mathbf{\Omega}$. In this case, a fixed covariance matrix is used to evaluate the coefficients produced by each randomized draw $\mathbf{T}_S$ from $\mathbf{\Omega}$, calculating the probability

$$(7) \qquad \hat{\boldsymbol{\beta}}_t'(\mathbf{T}_S)\mathbf{V}(\hat{\boldsymbol{\beta}}_t(\mathbf{\Omega}))^{-1}\hat{\boldsymbol{\beta}}_t(\mathbf{T}_S) \geqslant \hat{\boldsymbol{\beta}}_t'(\mathbf{T}_E)\mathbf{V}(\hat{\boldsymbol{\beta}}_t(\mathbf{\Omega}))^{-1}\hat{\boldsymbol{\beta}}_t(\mathbf{T}_E)$$

In the univariate case, this reduces to the square of the coefficients divided by a common variance and, after eliminating the common denominator, a simple comparison of squared coefficients. Hence, I refer to this as the randomization-c. It corresponds to bootstrap tests which use the distribution of bootstrapped coefficients to calculate the covariance matrix. In the analysis of my sample, I use 10,000 randomization draws to approximate $\mathbf{V}(\hat{\boldsymbol{\beta}}_t(\mathbf{\Omega}))$.

Although in principle all randomization test statistics are equally valid, in practice I find the randomization-t to be superior to the -c. First, when jointly testing more than one treatment effect, the -c relies on a sampling approximation of the coefficient covariance matrix. Consequently, the comparison in inequality (7) is not strictly speaking a draw-by-draw comparison of $f(\mathbf{T}_S)$ to $f(\mathbf{T}_E)$, and the assumptions underlying the proof that equation (5) is exact do not hold. In fact, in simulations (further below) I find statistically significant deviations from nominal size of -c in joint tests of true sharp nulls. Second, when the sharp null is false and heterogeneity in treatment effects exists, the randomization-c performs very poorly, even in tests of individual treatment effects, but the randomization-t does quite well, as shown later. The greater robustness of the randomization-t to an error in the underlying assumptions is clearly a desirable feature. That said, in the actual analysis of my sample results using the randomization-c and -t are very similar.

III.C. *Joint- versus Multiple-Hypothesis Testing*

I use joint- and multiple-testing procedures to test the null that all treatment effects reported together in regressions
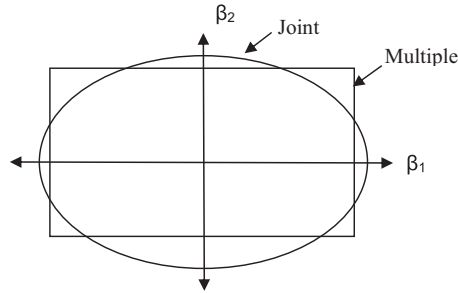
FIGURE II

Acceptance Regions for Joint and Multiple Testing with Independent Estimates

or tables are 0. The two approaches provide power against different alternatives, as illustrated in Figure II, which considers the case of testing the significance of two coefficients whose distribution is known to be normal and independent of each other.[13] The rectangle in the figure is the acceptance region for the two coefficients tested individually with a multiple-testing adjustment to critical values, whereas the oval is the Wald acceptance region for the joint significance of the two coefficients. In the multiple-testing framework, to keep the probability of one or more Type I errors across the two tests at level α, one could select a size η for each test such that $1 - (1 - \eta)^2 = \alpha$. The probability of no rejections, under the null, given by the integral of the probability density inside the rectangle, then equals $1 - \alpha$. The integral of the probability density inside the Wald ellipse is also $1 - \alpha$. The Wald ellipse, however, is the translation-invariant procedure that minimizes the area such that the probability of falling in the acceptance region is $1 - \alpha$.[14] It achieves this, relative to the multiple-testing rectangle, by dropping corners where the probability of two extreme outcomes is low and increasing the acceptance region along the axes. Consequently, the joint test has greater power to reject in favor of alternatives within quadrants, whereas multiple testing has greater power to reject when alternatives lie on axes. In the analysis of the experimental

13. A version of this diagram can be found in Savin (1984).

14. A procedure is translation invariant if, after adding a constant to both the point estimate and the null, one remains in the confidence region. Stein (1962) provides examples of procedures that do not satisfy this requirement but produce smaller confidence regions.

sample further below, I find that joint testing produces rejection rates that are generally slightly greater than those found using multiple-testing procedures, that is, while articles emphasize the extreme effects of individual statistically significant treatment measures, evidence in favor of the relevance of treatment is at least as often found in the modest effects of multiple aspects of treatment.

Multiple testing is an evolving literature. The classical Bonferroni method evaluates each test at the $\frac{\alpha}{N}$ level, which, based on Boole's inequality, ensures that the probability of a Type I error in N tests is less than or equal to α for any correlation between the test statistics. For values of α such as 0.01 or 0.05, the gap between $\frac{\alpha}{N}$ and the p-value cutoff $\eta = 1 - (1-\alpha)^{\frac{1}{N}}$ that would be appropriate if the test statistics were known to be independent, as in the example above, is minuscule. Nevertheless, because Bonferroni's method does not make use of information on the covariance of p-values, it can be quite conservative. For example, if the p-values of individual tests are perfectly correlated under the null, then α is the αth percentile of their minimum and hence provides an α probability of a Type I error when applied to all tests. In recognition of this, Westfall and Young (1993) suggested using bootstrap or randomization inference to calculate the joint distribution of p-values and then using the αth percentile of the minimum as the cutoff value. In the analysis of the sample below, I find that Westfall and Young's procedure yields substantially higher rejection rates than Bonferroni's method in table-level tests of treatment effects, as coefficients appearing in different columns of tables are often highly (if not perfectly) correlated.

While joint testing produces a single 0/1 decision, multiple testing allows for further tests, as following an initial rejection one can step-down through the remaining tests using less demanding cutoffs (e.g., Holm 1979; Westfall and Young 1993). Step-down procedures of this sort require either "subset pivotality" (Westfall and Young 1993), that is, that the multivariate distribution of p-values for subsets of hypotheses does not depend on the truth of other hypotheses, or more generally, that critical values are weakly monotonic in subsets of hypotheses (Romano and Wolf 2005). Both conditions trivially hold when authors kindly project a different dependent variable on a single treatment measure in each column of a table. This rarely occurs. Within

equations, treatment measures are interacted with covariates, making the calculation of a randomization distribution without an operational null on each treatment measure impossible, as noted earlier. Across columns of tables the same dependent variable is usually projected on slightly different specifications or subsamples, making the existence of nonzero effects in one specification and a sharp null in another logically impossible.[15] However, the null that every aspect of treatment has zero effects everywhere on everyone can always be tested.

I use joint- and multiple-testing procedures in this article to highlight the relevance of randomization inference in these, as the size distortions of inexact methods are much larger in higher-dimensional joint tests and in evaluating extreme tail probabilities. In multiple testing I restrict attention to the existence of any rejection, as this initial test can be applied to any group of results in my sample. Alternative multiple-testing procedures start with the same initial Bonferroni or Westfall–Young cutoff, and hence their initial decisions are subsumed in those results.[16] The existence of any rejection in multiple testing also produces a result equivalent to the joint test, that is, a statement that the combined null is false, allowing a comparison of the two methods and of the evidentiary value of traditionally emphasized treatment effects on axes against that provided by the combinations of treatment effects found within quadrants.

IV. MONTE CARLOS

In this section, I use simulations with balanced and unbalanced regression design and fixed and heterogeneous treatment effects to compare rejection rates of true nulls using clustered/robust covariance estimates to results obtained using randomization inference, as well as those found using the bootstrap and jackknife. For randomization inference and the bootstrap I use the randomized and bootstrapped distribution

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15. As examples: (i) having rejected the null of zero effects for women, it is not possible to consider a sharp null of zero in an equation that combines men and women; (ii) having rejected the null of zero effects in the projection of an outcome on treatment and covariates, it is not possible to then consider a sharp null of zero in the projection of the outcome on treatment alone.

16. Thus, for example, control of the false discovery rate at rate α using Benjamini and Hochberg's (1995) step-down procedure imposes a rejection criterion of $\frac{\alpha}{N}$ for the first step.

of coefficients and robust t-statistics to evaluate the p-value, that is, the -c and -t methods described earlier in inequalities (6) and (7). The bootstrap-t is generally considered superior to the -c because its rejection probabilities converge more rapidly asymptotically to nominal size (Hall 1992). For OLS regressions, the jackknife substitutes $\varepsilon_{\sim i}$, the residual for observation i when the delete-i coefficient estimates are used to calculate predicted values for that observation, for the $[\frac{n}{n-k}]^{\frac{1}{2}}$ adjusted estimated residual used in the clustered/robust formula (3) earlier, which has the disadvantage of being biased toward 0 in high-leverage observations. It is equivalent to the hc3 finite-sample correction of clustered/robust covariance estimates, which appears to provide better inference in finite samples (MacKinnon and White 1985).

Table III reports size at the .05 level of the different methods in tests of individual coefficients. Panel A uses the data generating process $y_i = \alpha + t_i \beta_i + \varepsilon_i$, with ε_i distributed i.i.d. standard normal and t_i a 0/1 treatment measure administered in a balanced (50/50) or unbalanced (10/90) fashion. For β_i, I consider both fixed treatment effects, with $\beta_i = \beta$ for all observations, and heterogeneous treatment effects, with β_i distributed i.i.d. standard normal or i.i.d. chi^2. Panel B uses the data-generating process $y_i = \alpha + t_i^* \beta_i + t_i^* x_i^* \gamma_i + x_i + \varepsilon_i$, where ε_i is again distributed i.i.d. normal and t_i is a 0/1 treatment measure administered in a balanced (50/50) fashion. Treatment interacts with a participant characteristic x_i, which is distributed i.i.d. exponential with mean 1. Once again, the parameters β_i and γ_i are either fixed or distributed i.i.d. normal or chi^2. Sample sizes range from 20 to 2,000. In each case, I use OLS to estimate average treatment effects in a specification that follows the data-generating process. With 10,000 simulations there is a 0.99 probability of estimated size lying between 0.044 and 0.056 if the rejection probability is actually 0.05.

Two patterns emerge in Table III. First, with evenly distributed leverage, all methods, with the exception perhaps of the bootstrap-c, do reasonably well. This is apparent in the left side of Panel A, where leverage is evenly distributed in all samples, but also in the rapid convergence of size to nominal value with unbalanced regression design in the right side of Panel A, where the maximal leverage of a single observation falls from 0.45 to 0.045 to 0.0045 with the increase in the sample size. Things proceed much less smoothly in Panel B, where the exponentially distributed covariate ensures that the maximal observation leverage share re-

TABLE III
SIZE AT THE 0.05 LEVEL IN 10,000 SIMULATIONS
(REJECTION RATES IN TESTS OF THE TRUE MEAN OF THE DATA-GENERATING PROCESS)

	Robust (1)	Rand-t (2)	Rand-c (3)	Boot-t (4)	Boot-c (5)	J-knife (6)	Robust (1)	Rand-t (2)	Rand-c (3)	Boot-t (4)	Boot-c (5)	J-knife (6)
Panel A: Tests of effects of binary treatment (t_i) given the data-generating process $y_i = \alpha + t_i{}^*\beta_i + \varepsilon_i$												
			Balanced regression design							Unbalanced regression design		
Fixed treatment effects: $\beta_i = \beta$, $\varepsilon_i \sim$ standard normal												
20	0.048	0.048	0.048	0.039	0.068	0.044	0.241	0.046	0.048	0.000	0.108	0.140
200	0.048	0.048	0.048	0.050	0.051	0.048	0.067	0.051	0.050	0.049	0.063	0.057
2,000	0.049	0.049	0.049	0.050	0.050	0.049	0.053	0.052	0.052	0.051	0.053	0.052
Heterogeneous treatment effects: $\beta_i \sim$ standard normal, $\varepsilon_i \sim$ standard normal												
20	0.052	0.052	0.052	0.040	0.073	0.046	0.283	0.089	0.129	0.000	0.129	0.172
200	0.053	0.052	0.052	0.053	0.055	0.052	0.064	0.051	0.131	0.045	0.060	0.055
2,000	0.049	0.048	0.048	0.048	0.048	0.049	0.052	0.052	0.137	0.051	0.052	0.051
Heterogeneous treatment effects: $\beta_i \sim$ chi^2, $\varepsilon_i \sim$ standard normal												
20	0.060	0.062	0.062	0.046	0.082	0.055	0.290	0.091	0.144	0.000	0.131	0.174
200	0.054	0.055	0.055	0.051	0.055	0.053	0.083	0.065	0.189	0.056	0.079	0.071
2,000	0.045	0.045	0.045	0.045	0.045	0.045	0.054	0.052	0.195	0.051	0.054	0.053
Panel B: Tests given the data-generating process $y_i = \alpha + t_i{}^*\beta_i + t_i{}^*x_i{}^*\gamma_i + x_i + \varepsilon_i$												
			Of coefficient on binary treatment (t_i)							Of coefficient on interaction ($t_i{}^*x_i$)		
Fixed treatment effects: $\beta_i = \beta$, $\gamma_i = \gamma$, $\varepsilon_i \sim$ standard normal												
20	0.064	0.051	0.052	0.025	0.049	0.039	0.114	0.052	0.054	0.037	0.021	0.042
200	0.053	0.049	0.049	0.051	0.052	0.048	0.064	0.049	0.050	0.054	0.051	0.049
2,000	0.051	0.050	0.050	0.051	0.051	0.051	0.052	0.049	0.050	0.050	0.051	0.049
Heterogeneous treatment effects: β_i, γ_i, & $\varepsilon_i \sim$ standard normal												
20	0.077	0.055	0.056	0.028	0.052	0.038	0.205	0.074	0.067	0.068	0.042	0.065
200	0.071	0.055	0.056	0.052	0.066	0.053	0.101	0.066	0.072	0.058	0.087	0.072
2,000	0.054	0.053	0.054	0.047	0.056	0.051	0.058	0.053	0.056	0.048	0.058	0.053
Heterogeneous treatment effects: β_i & $\gamma_i \sim$ chi^2, $\varepsilon_i \sim$ standard normal												
20	0.072	0.057	0.055	0.022	0.046	0.034	0.205	0.076	0.071	0.073	0.038	0.074
200	0.069	0.059	0.060	0.052	0.066	0.054	0.134	0.105	0.107	0.100	0.117	0.107
2,000	0.064	0.061	0.062	0.058	0.065	0.061	0.077	0.074	0.072	0.063	0.077	0.074

Note. 20, 200, 2,000 = number of observations in the regression; randomization and bootstrap test statistics evaluated using 1,000 draws with *-t* versions using robust standard error estimates; clustered/robust tests evaluated using conventional $n - k$ degrees of freedom; jackknife tests evaluated using $n - 1$ degrees of freedom.

mains above 0.029 (and as high as 0.11) more than one-quarter of the time even in samples with 2,000 observations. Asymptotic theorems rely on an averaging that is precluded when estimation places a heavy weight on a small number of observations, so regression design rather than the crude number of observations is probably a better guide to the quality of inference based on these theorems.

Second, Table III shows that the randomization-*t* is much more robust than the -*c* to deviations from the sharp null. When heterogeneous treatment effects not accounted for in the randomization test are introduced, rejection rates using the randomization-*c* rise well above nominal value, but the randomization-*t* continues to do well, with rejection probabilities that are closer to nominal size than any method other than the bootstrap-*t*. The intuition for this result is fairly simple. Heterogeneous treatment effects introduce heteroskedasticity correlated with extreme values of the regressors, making coefficient estimates more volatile. When treatment is rerandomized with a sharp null adjustment to the dependent variable equal to the mean treatment effect of the data-generating process, the average treatment effect is retained, but the correlation between the residual and the treatment regressor is broken, so the coefficient estimate becomes much less volatile. When the deviation of the original coefficient estimate from the null is compared to this randomized distribution of coefficients, it appears to be an outlier, generating a randomization-*c* rejection probability well in excess of nominal size, as shown in the table. In contrast, the randomization-*t* adjusts the initial coefficient deviation from the null using its large robust standard error estimate and all the later, less volatile, coefficient deviations from the null using the much smaller robust standard error estimates that arise when heteroskedasticity is no longer correlated with the regressors. By implicitly taking into account how rerandomization reduces the correlation between heteroskedastic residuals and the treatment regressor, the randomization-*t* adjusts for how rerandomization reduces the dispersion of coefficient estimates around the null.

Table IV evaluates rejection rates of the different methods in joint and multiple tests of the significance of treatment effects in 10 independent equations of the form used in Table III, Panel A. The top panel reports the frequency with which at least one true null is rejected using the Bonferroni multiple-testing critical value of $\frac{0.05}{10} = 0.005$. The bottom panel reports the frequency with which the joint null that all 10 coefficients equal the data-generating value are rejected, using White's (1982) extension of the robust covariance method to estimate the covariance of treatment coefficients across equations for the conventional Wald statistic and the randomization and bootstrap estimates of its distribution. The most notable difference in the pattern of results,

TABLE IV

SIZE AT THE 0.05 LEVEL IN 10,000 SIMULATIONS OF JOINT AND MULTIPLE TESTS
(10 INDEPENDENT EQUATIONS WITH THE
DATA-GENERATING PROCESS OF TABLE III, PANEL A)

	Balanced regression design						Unbalanced regression design					
	Robust	Rand-t	Rand-c	Boot-t	Boot-c	J-knife	Robust	Rand-t	Rand-c	Boot-t	Boot-c	J-knife
	(1)	(2)	(3)	(4)	(5)	(6)	(1)	(2)	(3)	(4)	(5)	(6)
Panel A: Bonferroni tests: probability of a rejection of any of the 10 true nulls												
Fixed treatment effects: $\beta_i = \beta$, $\varepsilon_i \sim$ standard normal												
20	0.056	0.055	0.055	0.026	0.128	0.049	0.682	0.051	0.045	0.000	0.065	0.489
200	0.047	0.047	0.047	0.046	0.053	0.046	0.098	0.051	0.053	0.039	0.081	0.080
2,000	0.052	0.052	0.052	0.051	0.051	0.052	0.052	0.048	0.048	0.047	0.052	0.051
Average treatment effects: $\beta_i \sim$ standard normal, $\varepsilon_i \sim$ standard normal												
20	0.053	0.052	0.052	0.023	0.126	0.046	0.813	0.143	0.193	0.000	0.094	0.611
200	0.052	0.051	0.051	0.050	0.056	0.051	0.106	0.056	0.277	0.041	0.090	0.085
2,000	0.047	0.047	0.047	0.045	0.044	0.046	0.051	0.049	0.275	0.048	0.050	0.050
Average treatment effects: $\beta_i \sim$ chi^2, $\varepsilon_i \sim$ standard normal												
20	0.078	0.078	0.078	0.037	0.164	0.068	0.821	0.157	0.230	0.000	0.093	0.621
200	0.057	0.059	0.059	0.051	0.061	0.057	0.173	0.106	0.436	0.078	0.143	0.143
2,000	0.051	0.051	0.051	0.051	0.052	0.051	0.073	0.068	0.478	0.060	0.071	0.071
Panel B: Joint tests: probability of rejecting the jointly true null												
Fixed treatment effects: $\beta_i = \beta$, $\gamma_i = \gamma$, $\varepsilon_i \sim$ standard normal												
20	0.594	0.053	0.060	0.000	0.595	0.378	0.998	0.051	0.058	0.000	0.000	0.995
200	0.071	0.049	0.054	0.045	0.078	0.060	0.386	0.048	0.055	0.007	0.346	0.324
2,000	0.053	0.048	0.054	0.049	0.057	0.052	0.069	0.048	0.054	0.046	0.071	0.065
Average treatment effects: β_i, γ_i, & $\varepsilon_i \sim$ standard normal												
20	0.615	0.064	0.074	0.000	0.617	0.404	1.00	0.217	0.217	0.000	0.000	1.00
200	0.076	0.050	0.056	0.046	0.084	0.064	0.457	0.087	0.385	0.002	0.411	0.392
2,000	0.050	0.047	0.051	0.048	0.054	0.049	0.068	0.051	0.392	0.047	0.070	0.064
Average treatment effects: β_i and $\gamma_i \sim$ chi^2, $\varepsilon_i \sim$ standard normal												
20	0.671	0.094	0.106	0.000	0.672	0.466	1.00	0.316	0.315	0.000	0.000	1.00
200	0.089	0.063	0.069	0.046	0.093	0.075	0.536	0.141	0.617	0.002	0.487	0.473
2,000	0.051	0.050	0.056	0.048	0.058	0.050	0.086	0.068	0.644	0.052	0.088	0.083

Notes. Covariance matrix for the joint Wald test using robust, randomization-t, and bootstrap-t methods calculated following White (1982). Robust standard error used for -t based distributions in multiple tests.

relative to Table III, is the magnitude of the size distortions. In small samples the robust and jackknife approaches have rejection probabilities approaching 1.0, particularly in joint tests, whereas bootstrap rejection probabilities range from 0 to well above 0.05 but are rarely near 0.05. Even with perfectly balanced leverage, in small samples joint and multiple tests often have rejection

probabilities that are well outside the 0.99 probability interval for an exact test.[17]

Size distortions increase with dimensionality in joint and multiple tests for different reasons. In the case of multiple tests, the problem is that a change in the thickness of the tails of a distribution generally results in a proportionally greater deviation at more extreme tail values. Thus, a test that has twice (or one-half) the nominal rejection probability at the .05 level will typically have more than twice (less than one-half) the nominal rejection probability at the .005 level. Consequently, as N increases and the $\frac{\alpha}{N}$ Bonferroni cutoff falls, the probability of a rejection across any of the N coefficients will deviate further from its nominal value, so small size distortions in the test of one coefficient become large size distortions in the test of N.

In the case of joint tests, intuition can be found by noting that the Wald statistic is actually the maximum squared t-statistic that can be found by searching over all possible linear combinations $\mathbf{w}$ of the estimated coefficients (Anderson 2003), that is:

$$(8) \qquad \hat{\boldsymbol{\beta}}'\hat{\mathbf{V}}^{-1}\hat{\boldsymbol{\beta}} = \underset{\mathbf{w}}{\mathrm{Max}}\, \frac{\left(\mathbf{w}'\hat{\boldsymbol{\beta}}\right)^2}{\mathbf{w}'\hat{\mathbf{V}}\mathbf{w}}.$$

When the covariance estimate equals the true covariance matrix $\mathbf{V}$ times a scalar error, that is, $\hat{\mathbf{V}} = \mathbf{V}\frac{\hat{\sigma}^2}{\sigma^2}$, as is the case with homoskedastic errors and covariance estimates, this search is actually very limited and produces a variable with a chi^2 or F distribution.[18] However, when $\hat{\mathbf{V}}$ is no simple scalar multiple of the true covariance $\mathbf{V}$, the search

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17. As noted earlier, in joint tests the randomization-c is no longer exact even in tests of sharp nulls, as the covariance matrix in the calculation of the distribution of test statistics in inequality (7) is only an approximation to the covariance matrix across all possible randomization draws. This is clearly seen in the rejection probabilities of 0.060 and 0.058 in samples of 20 in Panel B's analysis of joint tests of fixed treatment effects.

18. Employing the transformations $\tilde{\mathbf{w}} = \mathbf{V}^{1/2}\mathbf{w}$ and $\tilde{\boldsymbol{\beta}} = \mathbf{V}^{-1/2}\hat{\boldsymbol{\beta}}$, plus the normalization $\tilde{\mathbf{w}}'\tilde{\mathbf{w}} = 1$:

$$\underset{\mathbf{w}}{\mathrm{Max}}\, \frac{\left(\mathbf{w}'\hat{\boldsymbol{\beta}}\right)^2}{\mathbf{w}'\hat{\mathbf{V}}\mathbf{w}} = \underset{\tilde{\mathbf{w}}}{\mathrm{Max}}\, \frac{\left(\tilde{\mathbf{w}}'\tilde{\boldsymbol{\beta}}\right)^2}{\tilde{\mathbf{w}}'\mathbf{V}^{-1/2}\hat{\mathbf{V}}\mathbf{V}^{-1/2}\tilde{\mathbf{w}}} = \frac{\tilde{\boldsymbol{\beta}}'\tilde{\boldsymbol{\beta}}}{\hat{\sigma}^2/\sigma^2}$$

The last equality follows because the denominator reduces to $\frac{\hat{\sigma}^2}{\sigma^2}$ no matter what the $\tilde{\mathbf{w}}$ such that $\tilde{\mathbf{w}}'\tilde{\mathbf{w}} = 1$, while the maximum of the numerator across $\tilde{\mathbf{w}}$ equals $\tilde{\boldsymbol{\beta}}'\tilde{\boldsymbol{\beta}}$, which is typically an independent chi^2 variable with k degrees of freedom (dof). Thus, the maximum is either distributed chi^2 with k dof (when asymptotically $\hat{\sigma}^2 = \sigma^2$) or else equals k times an $F_{k,n\text{-}k}$ (when the denominator is
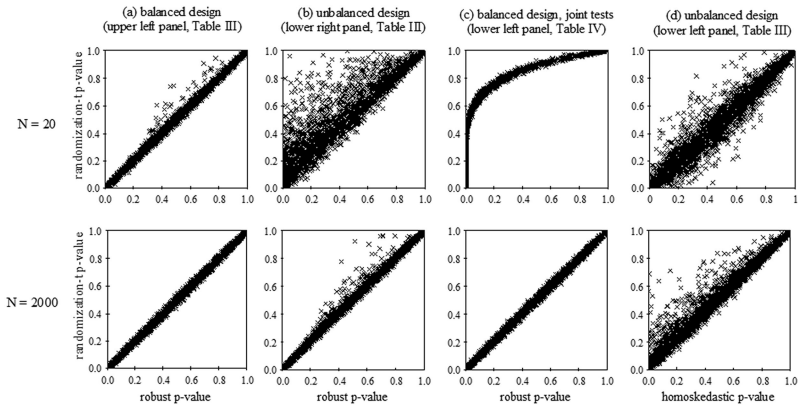
FIGURE III

Randomization-*t* versus Conventional *p*-Values

Each figure shows 3,000 paired *p*-values, 1,000 for each of the treatment effect data-generating processes (fixed, normal, and chi^2) in the indicated table panel with *N* observations.

possibilities expand, allowing for much larger tail outcomes. This systematically produces rejection probabilities much greater than size in clustered/robust joint tests.[19] At the same time, if the bootstrapped or randomized distribution of $\hat{\mathbf{V}}$ is even slightly misrepresentative of its true distribution, the two methods can greatly over- or understate the search possibilities in the original procedure, producing large size distortions of their own. Thinking of a joint test as a maximization problem, I believe, provides some intuition for why errors in approximating the distribution increase with the dimensionality of the test.

Figure III graphs randomization-*t* *p*-values against those found using conventional techniques. In each panel I take the first 1,000 results from each of the three data-generating processes for parameters (fixed, normal, and chi^2), comparing results with small ($N = 20$) and large ($N = 2,000$) samples. Panel A graphs *p*-values from the balanced regression design of the upper left panel of Table III, where robust *p*-values are nearly exact in both

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$(n - k)^{-1}$ times a chi^2 variable with $n - k$ dof). However, when $\hat{\mathbf{V}} \neq \mathbf{V}\frac{\hat{\sigma}^2}{\sigma^2}$ the search possibilities in the denominator clearly expand.

19. Young (2018a) provides further evidence of this for the case of *F*-tests of coefficients in a single regression.

small and large samples, showing that randomization and conventional *p*-values are almost identical in both cases. Panel B graphs the *p*-values of the lower right panel of Table III, where robust methods have positive size distortions in small samples. In small samples, randomization *p*-values are concentrated above conventional results, with particularly large gaps for statistically significant results, but in large samples the two types of results are once again almost identical. Panel C graphs the joint tests of the lower left panel of Table IV, where robust methods produce large size distortions in small samples but have accurate size in large samples. In small samples the pattern of randomization *p*-values lying above robust results, particularly for small conventional *p*-values, is accentuated, but once again differences all but disappear in large samples.[20]

Panels A–C of Figure III might lead to the conclusion that randomization and conventional *p*-values agree in large samples or when both *p*-values are nearly exact. Panel D shows this is not the case by examining conventional inference with the default homoskedastic covariance estimate for the highly leveraged coefficients tested in the lower left panel of Table III. With samples of 20 observations, despite the fact that errors are heteroskedastic in two-thirds of the simulations (β_i distributed chi^2 or normal), conventional and randomization-*t* inference using the homoskedastic covariance estimate produce rejection rates that are very close to nominal value (i.e., .048 and .051 at the .05 level, respectively). Nevertheless, randomization and conventional *p*-values are scattered above and below each other.[21] As the sample size increases, the default covariance estimate results in a growing rejection probability for the conventional test (0.080 at the .05 level), but no change in randomization rejection rates, so randomization *p*-values end up systematically above the conventional results. The pattern that does emerge from these simulations is that randomization and conventional *p*-values are quite close when maximal leverage, either through regression design or the effects of sample size, is relatively small and conventional and randomization inference are exact or very nearly so.

20. Figures for bootstrap-*t* and jackknife *p*-values compared with robust *p*-values show the same patterns.

21. This is not an artifact of the use of the homoskedastic covariance estimate under heteroskedastic conditions. The dispersion of *p*-values in the case of fixed treatment effects, where both methods are exact, is similar.

Beyond size, there is the question of power. In the Online Appendix I vary the mean treatment effect of the data-generating processes in the upper panel of Table III and calculate the frequency with which randomization-t and conventional robust inference reject the incorrect null of zero average or sharp treatment effects. When both methods have size near nominal value, their power is virtually identical. When conventional robust inference has large size distortions, that is, in small samples with unbalanced regression design, randomization inference has substantially lower power. This is to be expected, as a tendency to reject too often becomes a valuable feature when the null is actually false. However, from the point of view of Bayesian updating between nulls and alternatives, it is the ratio of power to size that matters, and here randomization inference dominates, with ratios of power to size that are above (and as much as two to three times) those offered by robust inference when the latter has positive size distortions.

To conclude, Tables III and IV show the clear advantages of randomization inference, particularly randomization inference using the randomization-t. When the sharp null is true, randomization inference is exact no matter what the characteristics of the regression. Moreover, the fact that randomization inference is superior to all other methods when the sharp null is true, does not imply the inverse, that is, that it is inferior to all other methods when the sharp null is false. When unrecognized heterogeneous treatment effects are present, the randomization-t test of the sharp fixed null produces rejection probabilities that are often quite close to nominal value, and in fact closer than most other testing procedures. In the case of high-dimensional multiple- and joint-testing problems, it is arguably the only basis to construct reliable tests in small samples, albeit only of sharp nulls.

V. Results

This section applies the testing procedures described above to the 53 papers in my sample. As the number of coefficients, regressions, and tables varies greatly by paper, reported results are the average across papers of within-paper rejection rates, so that each paper carries an equal weight in summary statistics. All randomization tests are based upon the distribution of t and Wald statistics, which, as noted above, are more robust to deviations away from sharp nulls in favor of heterogeneous treatment ef-

TABLE V

INDIVIDUAL STATISTICAL SIGNIFICANCE OF REPORTED TREATMENT EFFECTS

	.01	.05	.01	.05	.01	.05	.01	.05
	All papers (53 papers)		Low leverage (18 papers)		Medium leverage (17 papers)		High leverage (18 papers)	
Authors' p-value	0.216	0.354	0.199	0.310	0.164	0.313	0.283	0.437
Randomization-t	0.78	0.87	0.96	0.98	0.79	0.96	0.65	0.74
Bootstrap-t	0.79	0.84	0.99	0.98	0.87	0.89	0.60	0.70
Jackknife	0.78	0.83	0.95	0.89	0.87	0.91	0.61	0.73
	First table (45 papers)		Other tables (45 papers)		With interactions (29 papers)		No interactions (29 papers)	
Authors' p-value	0.303	0.446	0.188	0.338	0.148	0.292	0.310	0.450
Randomization-t	0.82	0.97	0.81	0.84	0.76	0.82	0.87	0.97
Bootstrap-t	0.85	0.91	0.90	0.80	0.86	0.80	0.87	0.88
Jackknife	0.91	0.94	0.81	0.79	0.80	0.83	0.93	0.89

Notes. Based on 4,044 reported treatment coefficients. .01, .05 = level of the test. Top row reports average across papers of within-paper fraction of significant results evaluated using authors' methods; values in lower rows are average fraction of significant results evaluated using indicated method divided by the top row. Randomization and bootstrap use 10,000 iterations to calculate p-values based on the distribution of squared t-statistics (calculated using authors' methods); interactions refers to coefficients in regressions that interact treatment with participant characteristics or other nontreatment covariates; first/other table and with/no interactions comparisons involve fewer than 53 papers because not all papers have both types of regressions.

fects. Corresponding tests based on the distribution of coefficients alone produce very similar results and are reported in the Online Appendix. Reported bootstrap tests are also based on the distribution of t and Wald statistics, which asymptotically and in simulation produce more accurate size. Results using the bootstrapped distribution of coefficients are reported in the Online Appendix and have systematically higher rejection rates. To avoid controversy, I restrict the tests to treatment effects authors report in tables, rather than the unreported and arguably less important coefficients on other treatment measures in the same regressions. Results based on all treatment measures are reported in the Online Appendix and, with a few noted exceptions, exhibit similar patterns. Details on the methods used to implement the randomization, bootstrap, and jackknife tests are given in the Online Appendix. Variations on these methods (also reported there) produce results that are less favorable to authors and conventional tests.

Table V tests the statistical significance of individual treatment effects. The top row in each panel reports the average across papers of the fraction of coefficients that are statistically significant using authors' methods (rounded to three decimal places), and lower rows report the ratio of the same measure calculated using alternative procedures to the figure in the top row (rounded to two decimal places for contrast). In the upper left panel we see

that using authors' methods in the typical paper 21.6% and 35.4% of reported coefficients on treatment measures are significant at the .01 and .05 levels, respectively, but that the average number of significant treatment effects found using the randomization distribution of the *t*-statistic is only 78% and 88% as large at the two levels. Jackknife and *t*-statistic-based bootstrap significance rates agree with the randomization results at the .01 level and find somewhat lower relative rates of significance (83% to 84%) at the .05 level.

Table V divides the sample into low, medium, and high leverage groups based on the average share of the cluster or observation with the greatest coefficient leverage, as described in Table II. As shown, the largest difference between the three methods and authors' results is found in high-leverage papers, where on average randomization techniques find only 65% and 74% as many significant results at the .01 and .05 levels, respectively. Differences in rejection rates in the one-third of papers with the lowest average leverage are minimal. Jackknife and bootstrap results follow this pattern as well. The lower panel of the table compares treatment effects appearing in first tables to those in other tables, and those in regressions with treatment interactions with covariates against those without, in papers that have both types of coefficients. Results in first tables and in regressions without interactions tend to be more robust to alternative methods, with randomization rejection rates at the .05 level, in particular, coming in close (97%) to those found using authors' methods. Regressions in the Online Appendix of conventional versus randomization significance differences on dummies for a first table regression or one without interactions, as well as the number of observations, find that the addition of maximal coefficient leverage to the regression generally moves the coefficients on these measures substantively toward 0, while leaving the coefficient on leverage largely unchanged. Regression design is systematically worse in some papers and deteriorates within papers as authors explore their data using subsamples and interactions with covariates and this, rather than being in a first table or regression without covariates per se, appears to be the determinant of differences between authors' results and those found using randomization methods.

Table VI tests the null that all reported treatment effects in regressions with more than one reported treatment coefficient are zero using joint- and multiple-testing methods. The average num-

TABLE VI

JOINT STATISTICAL SIGNIFICANCE OF REPORTED TREATMENT EFFECTS (REGRESSION LEVEL)
(REGRESSIONS WITH MORE THAN ONE REPORTED TREATMENT COEFFICIENT)

	All papers (47 papers)		Low leverage (16 papers)		Medium leverage (16 papers)		High leverage (15 papers)		First table (29 papers)		Other tables (29 papers)	
	.01	.05	.01	.05	.01	.05	.01	.05	.01	.05	.01	.05
Significant coef.	0.431	0.643	0.353	0.596	0.450	0.607	0.495	0.731	0.469	0.620	0.413	0.584
Panel A: Joint test based on F and Wald statistics												
Authors' method	0.438	0.546	0.435	0.508	0.392	0.539	0.490	0.595	0.383	0.528	0.400	0.473
Randomization-t	0.76	0.83	1.01	1.00	0.90	0.94	0.42	0.58	0.84	0.92	0.84	0.86
Bootstrap-t	0.72	0.81	0.96	0.96	0.84	0.91	0.39	0.57	0.93	0.84	0.74	0.81
Jackknife	0.90	0.88	0.98	0.96	0.97	0.91	0.76	0.79	0.98	0.96	0.90	0.92
Panel B: Presence of at least one significant measure in multiple testing based on Bonferroni (B) and Westfall–Young (WY) methods												
Authors' p-value (B)	0.335	0.494	0.274	0.426	0.322	0.526	0.415	0.533	0.340	0.501	0.306	0.442
Randomization-t (B)	0.73	0.85	0.99	1.02	0.59	0.88	0.66	0.67	0.81	0.96	0.78	0.82
Bootstrap-t (B)	0.76	0.88	1.06	1.03	0.80	0.87	0.51	0.76	1.01	0.90	0.86	0.85
Jackknife (B)	0.80	0.85	1.03	0.93	0.78	0.94	0.64	0.68	0.98	0.97	0.85	0.84
Randomization-t (WY)	0.76	0.89	1.00	1.08	0.68	0.92	0.66	0.70	0.78	0.96	0.85	0.89
Bootstrap-t (WY)	0.77	0.92	1.07	1.03	0.80	0.93	0.52	0.81	1.01	0.94	0.87	0.87

Notes. Unless otherwise noted, as in Table V. Based on 922 regressions with multiple reported treatment effects. Significant coef. = presence of any coefficient in the regression with an authors' p-value below the indicated level; top row in each panel reports average across papers of the fraction of tests within each paper rejecting the combined null using authors' methods; lower rows report the same number calculated using alternative methods divided by the top row. Because the sample is restricted to regressions with more than one reported treatment coefficient, the number of papers appearing in each group differs from Table V, as indicated. Randomization and bootstrap tests are based on the distribution of the Wald statistic.

ber of reported treatment effects tested in such regressions in a paper ranges from 2 to 17.5, with a median of 3.0 and mean of 3.7. The top row of the table records (using three decimal places) the average fraction of regressions which find at least one individually .01 or .05 significant treatment coefficient using authors' methods. Below this I report (also using three decimal places) the average fraction of regressions which, again using authors' covariance calculation methods, either reject the combined null of zero effects directly in a joint *F*/Wald test (Panel A) or implicitly by having a minimum coefficient *p*-value that lies below the Bonferroni multiple-testing-adjusted cutoff (Panel B). As expected, the Bonferroni adjustment reduces the average number of significant results, as the movement from an α to an $\frac{\alpha}{N}$ *p*-value cutoff raises the average critical value of the *t*- or *z*-statistic for .01 significance from ± 2.6 to ± 3.0 in the average paper. Joint tests expand the critical region on any given axis further than multiple-testing procedures; in the case of my sample to an average .01 *t*- or *z*-critical value of ± 3.5 in the average paper. Despite this, joint tests have systematically higher rejection rates, in the sample as a whole and in every subsample examined in the table, as evidence against the irrelevance of treatment is found not in extreme coefficient values along the axes but in a combination of moderate values within quadrants. Although Wald ellipses expand acceptance regions along the axes, the area that receives all of the attention in the published discussion of individually significant coefficients, they do so to tighten the rejection region within quadrants, and this may yield otherwise underappreciated evidence against the null that experimental treatment is irrelevant. In a similar vein, when these tests are expanded to all coefficients, not merely those reported, rejection rates in joint and multiple tests actually rise slightly, despite the increase in critical values, as evidence against the null is found in treatment measures authors did not emphasize (Online Appendix).

Panels A and B of Table VI also report (using two decimal places for contrast) the average rejection rates of tests based on randomization, bootstrap, and jackknife techniques expressed as a ratio of the average rejection rate of the corresponding test using authors' methods. The relative reduction in rejection rates using randomization techniques is slightly greater than in Table V's analysis of coefficients and is especially pronounced in high-leverage papers, where, in joint tests, randomization tests find only 42% and 58% as many significant results as authors'

methods. This may be a consequence of the greater size distortions of clustered/robust methods in higher-dimensional tests, especially in high-leverage situations, discussed earlier. In joint tests bootstrap and jackknife results are alternately somewhat more and less pessimistic than those based on randomization inference, but both show similar patterns, with differences with conventional results concentrated in higher-leverage subsamples. Westfall–Young randomization and bootstrap measures raise rejection rates relative to Bonferroni-based results, as should be expected, as they calculate the joint distribution of *p*-values avoiding the "worst-case scenario" assumptions of the Bonferroni cutoffs.[22] Levels and patterns of relative rejection rates are quite similar when the tests are expanded to include unreported treatment effects (Online Appendix).

Table VII reports results for joint tests of reported treatment effects appearing together in tables. The results presented in tables usually revolve around a theme, typically the exploration of alternative specifications in the projection of one or more related outcomes of interest on treatment, treatment interactions with covariates, and treatment subsamples. The presence of both significant and insignificant coefficients in these tables calls for some summary statistic, evaluating the results in their entirety, and Table VII tries to provide these. For the purpose of Wald statistics in joint tests, I estimate the joint covariance of coefficients across equations using White's (1982) formula.[23] Calculation of this covariance estimate reveals that, unknown to readers (and possibly authors as well), coefficients presented in tables are often perfectly collinear, as the manipulation of variables and samples eventually

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22. Although a conventional equivalent of the Westfall–Young multiple-testing procedure could be calculated using the covariance estimates and assumed normality of coefficients, I report the Westfall–Young randomization and bootstrap rejection rates as a ratio of the conventional Bonferroni results to facilitate a comparison with the absolute rejection rates of the randomization and bootstrap Bonferroni tests, which are also normalized by the conventional Bonferroni results.

23. Because White's theory is based on maximum likelihood estimation, this is the one place where I modify authors' specifications, using the maximum-likelihood representation of their estimating equation where it exists. Differences in individual coefficient estimates are 0 or minimal. Some estimation methods (e.g., quantile regressions) have no maximum-likelihood representation and are not included in the tests. In the few cases where the number of clusters does not exceed the number of treatment effects, I restrict the table-level joint test to the subset of coefficients that Stata does not drop when it inverts the covariance matrix.

TABLE VII

JOINT STATISTICAL SIGNIFICANCE OF REPORTED TREATMENT EFFECTS (TABLE LEVEL)

	All papers (53 papers)		Low leverage (18 papers)		Medium leverage (17 papers)		High leverage (18 papers)		First table (45 papers)		Other tables (45 papers)	
	.01	.05	.01	.05	.01	.05	.01	.05	.01	.05	.01	.05
Significant coef.	0.662	0.818	0.617	0.788	0.602	0.753	0.764	0.908	0.711	0.889	0.630	0.786
Panel A: Joint test based on Wald statistics												
Conventional	0.493	0.622	0.337	0.487	0.431	0.522	0.706	0.850	0.422	0.556	0.483	0.606
Randomization-*t*	0.51	0.67	0.92	1.00	0.40	0.74	0.38	0.45	0.79	0.80	0.62	0.78
Bootstrap-*t*	0.21	0.33	0.33	0.51	0.18	0.41	0.18	0.19	0.38	0.45	0.23	0.40
Jackknife	0.77	0.84	0.92	0.86	0.76	0.91	0.71	0.78	0.89	0.84	0.81	0.84
Panel B: Presence of at least one significant measure in multiple testing based on Bonferroni (B) and Westfall–Young (WY) methods												
Authors' *p*-value (B)	0.377	0.542	0.329	0.489	0.275	0.475	0.521	0.659	0.400	0.556	0.349	0.491
Randomization-*t* (B)	0.61	0.81	0.88	0.98	0.63	0.75	0.43	0.72	0.78	1.00	0.69	0.84
Bootstrap-*t* (B)	0.69	0.78	1.00	1.06	0.62	0.73	0.54	0.62	0.78	0.92	0.79	0.82
Jackknife (B)	0.71	0.85	1.00	0.97	0.66	0.87	0.56	0.73	0.89	1.00	0.74	0.84
Randomization-*t* (WY)	0.77	0.91	1.18	1.06	0.67	0.96	0.55	0.77	1.00	1.12	0.79	0.92
Bootstrap-*t* (WY)	0.79	0.87	1.20	1.09	0.73	0.91	0.55	0.68	0.89	1.00	0.89	0.95

Notes. Unless otherwise noted, as in Table VI. Based on 198 tables. Comparisons for first tables limited to papers with these and other tables.

produces results that simply repeat earlier information.[24] These linear combinations are dropped in the case of joint tests, as they are implicitly subsumed in the joint test of the zero effects of the remaining coefficients. I retain them in the multiple-testing calculations based on individual *p*-values, however, because they provide a nice illustration of the advantages of Westfall–Young procedures in environments with strongly correlated coefficients.

The discrepancies between the rejection rates found using different methods in the joint tests of Table VII are much larger than those found in the preceding tables. Randomization tests show only 51% as many significant results at the .01 level as clustered/robust joint tests, while the bootstrap finds merely 21% as many significant results, and the jackknife does more to validate clustered/robust methods with 77% as many significant results. The number of treatment effects reported in tables ranges from 2 to 96, with the average table in a paper having a 53-paper mean of 19 and median of 17. As found in the Monte Carlos earlier, in high-dimensional joint tests of this type, clustered/robust and jackknife methods appear to have rejection probabilities much greater than nominal size, while Wald-based bootstraps grossly underreject. The results in Table VI are consistent with this pattern. Randomization inference based on Wald statistics in exact tests of sharp nulls is arguably the only credible basis for tests of this sort.

Turning to the Bonferroni multiple tests in Table VII, which rely only on the accuracy of covariance estimates for individual coefficients, the agreement between methods is better here, with a reduction in significance rates from conventional results of 61% to 81% using randomization inference and 69% to 85% using the bootstrap or jackknife. These are larger proportional reductions in significance rates than in any of the preceding tables. As shown in Section IV, size distortions grow in multiple testing as proportional deviations from the nominal size are greater at the more extreme tail cutoffs used in Bonferroni testing. This again argues in favor of the use of randomization inference, as this is the only basis to ensure accurate size, at least for tests of sharp nulls, at .001 or .0001 levels.

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24. Excluding the tables where the number of tested treatment effects is greater than or equal to the number of clusters, in the average paper 14% of tables contain perfectly collinear results. In such tables, on average one-fifth of the reported results are collinear with the remaining four-fifths.

Table VII highlights the advantages of Westfall–Young methods, using randomization or bootstrap inference to calculate the joint distribution of *p*-values within tables rather than adopting the conservative Bonferroni cutoff. Switching from the Bonferroni cutoff to the Westfall–Young calculation raises the relative number of significant randomization-*t* results by fully one-quarter (from 61% to 77%) at the .01 level and by one-eighth (from 81% to 91%) at the .05 level. This reflects the extensive repetition of information in tables, in the form of minor changes in the specification of right-hand-side variables or highly correlated or collinear left-hand-side variables. Westfall–Young methods incorporate this, raising the critical *p*-value for a finding of a significant result. This is a nice feature, as it allows the exploration of the data and presentation of correlated information without unduly penalizing authors. Critical *p*-values only become more demanding to the degree that new specifications add uncorrelated (i.e., new) information on the significance of treatment measures.

Finally, it is worth noting that Table VII reinforces Table VI's message that evidence against the null of experimental irrelevance can often be found within quadrants rather than along axes. Although in this case the average *t*- or *z*-statistic for rejection along an axis in the average paper rises from 3.4 in the Bonferroni test to 5.5 in the joint test, rejection rates in the conventional joint test are still greater than in the conventional Bonferroni test. Randomization inference produces a greater proportional reduction relative to conventional results in joint tests, but the average absolute rejection rates of the randomization-*t* in joint tests within papers at the .01 and .05 levels (25.1% and 41.9%, respectively) are comparable to those found using randomization-*t* Bonferroni tests (23.1% and 43.9%), although with Westfall–Young methods rejection rates are higher (28.9% and 49.5%). In a similar vein, conventional and randomization rejection rates are actually slightly higher once treatment effects that were not reported by authors are included (Online Appendix).

The preceding presentation is frequentist, in keeping with the emphasis on "starred" significant results in journals. In this context, all that matters are the 0/1 significance rates reported above, as a *p*-value of .011 is no more significant at the .01 level than a *p*-value of .11. Seminar participants and referees, however, often ask whether *p*-value changes are substantial, presumably reflecting quasi-Bayesian calculations involving the likelihood of
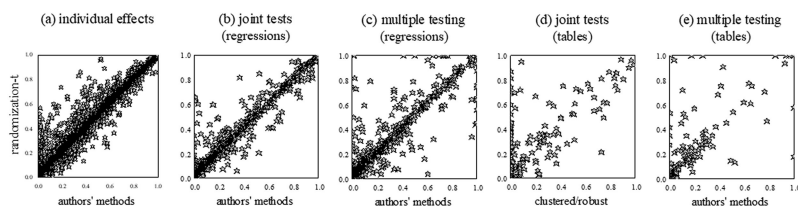
FIGURE IV

Randomization-*t* versus Conventional *p*-Values
in Tests of Reported Treatment Effects.

Multiple testing *p*-value $= \min(1, N^* p\text{min})$, where *p*min is the minimum *p*-value in the N individual tests. Joint tests for tables calculated using White's (1982) clustered/robust joint covariance estimate for multiple maximum likelihood equations.

outcomes under different hypotheses.[25] To this end, Figure IV graphs the randomization-*t* *p*-values against the conventional *p*-values for the tests discussed above. As can be seen, there are often very substantial differences, concentrated in particular in tests with conventionally statistically significant results. These patterns are consistent with those found earlier in Monte Carlos for unbalanced regression design, where conventional methods have sizable size distortions. Table VIII focuses on the average within-paper distribution of randomization *p*-values for conventional results that are statistically significant at the .01 or .05 levels. As can be seen, in tests at the coefficient level in the average paper about two-thirds of changes in significance merely bump the *p*-value into the next category (0.160/0.248 and 0.101/0.147). In contrast, in tests at the table level, the biggest movement by far is into *p*-values in excess of .20, which account for anything from 30% to 60% of all changes in .01 or .05 statistical significance in the average paper. The gaps between randomization and conventional results are greatest in high-dimensional tests where, as seen in Section IV, conventional clustered/robust tests have gross size distortions.

In the presentation above I focused on those methods that produce the results most favorable to authors. This is of great-

25. Similarly, in a frequentist world a failure to reject doesn't confirm the null, but in a Bayesian world large *p*-values increase its posterior probability, which might explain why authors emphasize the statistical insignificance of treatment coefficients in regressions related to randomization balance and sample attrition.

TABLE VIII

DISTRIBUTION OF RANDOMIZATION *p*-VALUES
FOR CONVENTIONALLY SIGNIFICANT RESULTS

	Individual treatment effects		Joint tests (regression)		Multiple testing (regression)		Joint tests (table)		Multiple testing (table)	
	.01	.05	.01	.05	.01	.05	.01	.05	.01	.05
< .01	0.752	↓	0.747	↓	0.699	↓	0.477	↓	0.635	↓
.01–.05	0.160	0.853	0.139	0.820	0.199	0.813	0.194	0.656	0.226	0.775
.05–.10	0.068	0.101	0.033	0.075	0.044	0.099	0.071	0.083	0.006	0.077
.10–.20	0.014	0.029	0.063	0.078	0.015	0.020	0.040	0.051	0.033	0.037
> .20	0.005	0.017	0.017	0.027	0.042	0.068	0.217	0.210	0.100	0.111

Notes. Reported figures are the average across papers of the within-paper distribution of randomization-*t* *p*-values when conventional tests register a significant result at the level specified; (↓) included in the category below; multiple-testing *p*-values are the Bonferroni adjusted minimum, as in Figure IV.

est relevance in the evaluation of the impact of randomization inference on *p*-values. For example, in the 12 papers in my sample where authors systematically clustered below treatment level, I follow their lead and treat the grouping of treatment in lab sessions and geographic neighborhoods as nominal, rerandomizing across observations rather than treatment groupings. If instead I were to cluster both the conventional and randomization tests at treatment level, then in the average paper in this group the fraction of .01 conventionally individually significant treatment effects that have individual randomization *p*-values in excess of .10 rises from 0.0% to 18.6%. Similarly, when the randomization-*c* is applied or when randomization-*t* "conditional" *p*-values that do not rely on a joint zero null for all treatment effects are calculated, 7.3% and 4.7%, respectively, of .01 conventionally individually significant results in the average paper have individual randomization *p*-values in excess of .10 (see the Online Appendix). Authors deserve the benefit of the doubt, but the minimization of differences in the tables above should not be read as a guide to expected differences in randomization and conventional results, particularly in highly leveraged estimation.

VI. CONCLUSION

If there is one message in this article, it is that there is value added in paying attention to regression and experimental design. Balanced designs lead to uniform leverage with con-

ventional results that are less sensitive to outliers and less subject to size distortions with clustered/robust covariance estimates, and produce *p*-values that are nearly identical across clustered/robust, randomization, bootstrap, or jackknife procedures. Regressions with multiple treatments and treatment interactions with participant characteristics generate concentrated leverage, producing coefficients and clustered/robust standard errors that depend heavily on a limited set of observations and have a volatility typically greater than standard error and degrees-of-freedom estimates. Rather than maintain the fiction that identification and inference comes from the full sample, more accurate results might be achieved by breaking the experiment and regression into groups based on treatment regime or participant characteristics, each with a balanced treatment design.

Consideration of experimental and regression design can also play a role in multiple testing. Although the tests used in this article can evaluate the general question of treatment relevance, more discerning results can be achieved if regressions are designed in a fashion that allows step-down procedures to control the Type I error or false discovery rate (e.g., Holm 1979; Westfall and Young 1993; Benjamini and Hochberg 1995). Practically speaking, to allow for this, tests have to be set up in a fashion that allows subset pivotality (Westfall and Young 1993), where the distribution of each randomization test statistic is independent of the nulls for other treatment results. Dividing regressions into mutually exclusive samples is a trivially easy way to ensure this. In sum, if the exploration of the effects of multiple treatment regimes and the differing effects of treatment in population subgroups is indeed the original intent of an experiment, then it is best to build this into the experimental design by stratifying the application of treatment to ensure balanced regression design for each treatment/control pair and population subgroup. This will allow accurate inference for individual coefficients and enable the application of multiple-testing procedures to control error rates across families of hypotheses.

That said, there will obviously be occasions where outlier treatment values arise through factors beyond experimenters' control or a conscious attempt to achieve greater power by expanding the range of treatment variation. In such highly leveraged circumstances, as well as in the case of high-dimensional joint

and multiple testing, randomization tests of sharp nulls provide a means to construct tests with credible finite-sample rejection probabilities.

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SUPPLEMENTARY MATERIAL

An Online Appendix for this article can be found at *The Quarterly Journal of Economics* online. Data and code replicating tables and figures in this article can be found in Young (2018b), in the Harvard Dataverse, doi:10.7910/DVN/JX6HCJ.

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