The Generalizability of Heterogeneous Treatment Effect Estimates Across Samples

Alexander Coppock^a, Thomas J. Leeper^b, and Kevin J. Mullinix^c

^aDepartment of Political Science, Yale University; ^bDepartment of Government, London School of Economics and Political Science; ^cDepartment of Political Science, University of Kansas

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The extent to which survey experiments conducted with nonrepresentative convenience samples are generalizable to target populations depends critically on the degree of treatment effect heterogeneity. Recent inquiries have found a strong correspondence between sample average treatment effects estimated in nationallyrepresentative experiments and in replication studies conducted with convenience samples. We consider here two possible explanations: low levels of effect heterogeneity or high levels of effect heterogeneity that are unrelated to selection into the convenience sample. We analyze subgroup conditional average treatment effects using 27 original-replication study pairs (encompassing 101,745 individual survey responses) to assess the extent to which subgroup effect estimates generalize. While there are exceptions, the overwhelming pattern that emerges is one of treatment effect homogeneity, providing a partial explanation for strong correspondence across both unconditional and conditional average treatment effect estimates.

Replication | Generalizability | Survey Experiments

1. Introduction

Randomized experiments are increasingly employed across the social sciences to study beliefs, opinions, and behavioral intentions (1, 2). Experiments are nevertheless sometimes met with skepticism about the degree to which results generalize (3). Indeed, it is often said that experiments achieve better internal validity than they do external validity because of the non-representative samples typically used in experimental research (e.g., (4–6), though see (7) for a critique of the claimed external validity of some nonexperimental regression estimates). In response, a series of scholars have developed methods for transporting experimental results obtained on a sample to target populations (8–11), typically by adjusting for factors that determine sample selection, treatment effect heterogeneity, or both.

Despite tremendous methodological progress in this area, the social scientific community has generated only limited theory and evidence to guide expectations about when a convenience sample and a target population are sufficiently similar to justify generalizing from one to the other. Sometimes demographic differences between, say, a student sample and the national population are taken as $prima\ facie$ evidence that results obtained on the student sample are unlikely to generalize. By contrast, several recent empirical studies suggest that convenience samples, despite drastic demographic differences, frequently yield average treatment effect estimates that are not substantially different from those obtained through nationally representative samples (12–15).

Such findings suggest that even in the face of differences in sample composition, claims of strong external validity are sometimes justified. What could explain the rough equivalence of sample average treatment effects (SATEs) across such different samples? We consider two possibilities: (A) effect homogeneity across participants such that sample characteristics are irrelevant, or (B) effect heterogeneity that is approximately orthogonal to selection. Arbitrating between these explanations is critical to predicting whether *future* experiments are likely to generalize.

2. Methods and Materials

We aim to distinguish between Scenarios A and B through reanalyses of 27 original-replication pairs collected by (12) and (13). This set of studies is useful because it constitutes a unique sample of original studies conducted on nationally representative samples and replications performed on convenience samples (namely Amazon Mechanical Turk) using identical experimental protocols. Both papers focused narrowly on replication as assessed by the correspondence between SATEs in each study pair, both finding a high degree of correspondence. Our goal here is to assess the degree of correspondence of conditional average treatment effect (CATE) estimates among 16 distinct subgroups defined by subjects' pre-treatment background characteristics.

We estimate all CATEs via difference-in-means. Our main statistic of interest is the slope of the original estimate with respect to the replication estimate, corrected for measurement error via a generalized Deming regression (16, 17). Deming regression (an errors-in-variables model) is appropriate because both the original and replication CATEs are estimated with error, itself estimated via the standard error of the CATEs. We calculate 95% confidence intervals around our slope estimates using the jackknife (17). We report separate slopes for each study (within study slopes) and demographic group (across study slopes).

Significance Statement

In experiments, the degree to which results generalize to other populations depends critically on the degree of treatment effect heterogeneity. We replicated 27 survey experiments (encompassing 101,745 individual survey responses) originally conducted on nationally-representative samples using online convenience samples, finding very high correspondence despite obvious differences in sample composition. We contend this pattern is due to low treatment effect heterogeneity in these types of standard social science survey experiments.

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 $^{^{1}\,\}text{A.C.},\,\text{T.J.L},\,\text{and K.J.M.}$ contributed equally to this work.

 $^{^2}$ To whom correspondence should be addressed. E-mail: alex.coppock@yale.edu

For readers unfamiliar with these sample platforms, Amazon Mechanical Turk provides non-probability, fully opt-in samples of participants who complete online tasks for financial compensation. Some studies find that Mechanical Turk workers generate high quality survey data (18, 19). The original studies rely mostly upon samples provided by Time Sharing Experiments for the Social Sciences (TESS) that are obtained from GfK's KnowledgePanel. KnowledgePanel panelists are recruited using probability-based random-digit dialing or address-based sampling methods and provided internet access if they do not have it. Panelists are then sampled using standard sampling methods. GfK/TESS differs from other online samples like Amazon Mechanical Turk in that it relies upon probability-based sampling methods through panel construction and is designed to provide panelists that are representative of the US adult population.

Because of the varied experimental protocols for each of the 54 separate experiments (27 study pairs) reanalyzed here, the largest challenge we face is measuring subject characteristics in an identical manner across experiments. While some studies measure a rich set of demographic, psychological, and political attributes, others only measure a few. We have identified six attributes that are measured in nearly all studies. These attributes are not always measured in the same way, so we have coarsened each to a maximum of three categories in order to maintain comparability across studies: age (18-39, 40-59, 60+), education (Less than College, College, Graduate School), gender (Men, Women), ideology (Liberal, Moderate, Conservative), partisanship (Democrat, Independent, Republican), and race (Nonwhite, White). We acknowledge that our covariate measures are rough and that many subtleties of scientific interest will unfortunately be masked. In particular, we regret the extreme coarsening of race and ethnicity into Nonwhite/White, but smaller divisions left us with far too little data in some cases.

A complete description of each experiment and replication procedures are available in the original papers and their supplementary materials (12, 13). The full list of studies, with the sample sizes used in the analyses reported here, is presented in Table 2. An examination of the discipline of the first, original study author reveals that our replication studies encompass political science, psychology, public health, communication, business, sociology, law, education, and public policy. By and large, these experiments estimate the effects of stimuli on social and political attitudes and are broadly representative of the sorts of persuasion, attitude-formation, and decision-making studies that are common in social science experiments. They do not include experiments used primarily for measurement, such as conjoint or list experiments.

3. Results

Figure 1 displays scatterplots of the estimated CATEs subgroup by subgroup. The relationship between the conditional average treatments in the original and Mechanical Turk versions of the studies is unequivocally positive for all demographic subgroups. Whereas previous analyses of these datasets showed strong correspondence of average treatment effects, this analysis shows that the same pattern holds at every level of age, gender, race, education, ideology, and partisanship that we measure.

The figure also indicates whether the CATEs are statis-

tically significantly different from each other.* Out of 393 opportunities, the difference-in-CATEs is significant 59 times, or 15% of the time. In zero of 393 opportunities do the CATEs have different signs while both being statistically distinguishable from zero. There is also a close correspondence of significance tests for CATEs across study pairs. Of the 156 CATEs that were significantly different from no effect in the original, 118 are significantly different from no effect in the MTurk replication. Of the 237 CATEs that were statistically indistinguishable from no effect in the original, 158 were statistically indistinguishable from zero in the MTurk version.

The overall "significance match" rate is therefore 70%. We must be careful, however, not to overinterpret conclusions based on this statistic, as it is confounded by the power of the studies. If the studies were infinitely powered, all estimates of non-zero CATEs in both versions of the study would be statistically significant and therefore the match rate would be 100%. By contrast, if all studies were severely underpowered, almost all estimates would be nonsignificant, again implying a match rate of 100%. We therefore prefer evaluating correspondence across-studies based on (error-corrected) regression slopes since they direct operate on the estimates themselves rather than on arbitrary significance levels.

The estimated slopes across CATEs are shown in Table 1. The slopes are all positive, ranging from 0.71 to 1.01. The smallest slope is for the conservative group, which aligns with a common belief that conservatives on MTurk are especially idiosyncratic, though this view is challenged in (21). A true slope of 1 would indicate perfect correspondence of original and replication CATEs within demographic subgroups. All but one of the 95% confidence intervals include 1, but the intervals are sometimes quite wide, so we resist "accepting the null" of perfect correspondence. We do conclude, however, that in this set of studies, the estimated CATEs within demographic subgroups are quite similar.

We now have two basic findings to explain: average treatment effects are approximately the same in probability and nonprobability samples and so are conditional average treatment effects. Which of our explanations (no heterogeneity or heterogeneity orthogonal to selection) can account for both findings?

To arbitrate between these explanations, we turn to withinstudy comparisons. Within a given study we ask, are the CATEs that were estimated to be high in the original study also high in the MTurk version? Figure 2 shows that the answer tends to be no. The CATEs in the original study are mostly uncorrelated with the CATEs in the MTurk versions. Table 2 confirms what the visual analysis suggests. We see within-study slopes that are smaller than the across-study slopes and slopes of both signs.

An inspection of the CATEs themselves reveals why. Most of the CATEs are tightly clustered around the overall average treatment effect in each study version. Put differently, the treatment effects within each study version appear to be mostly homogeneous. We conclude from this preliminary analysis that the main reason why we observe strong correspondence in

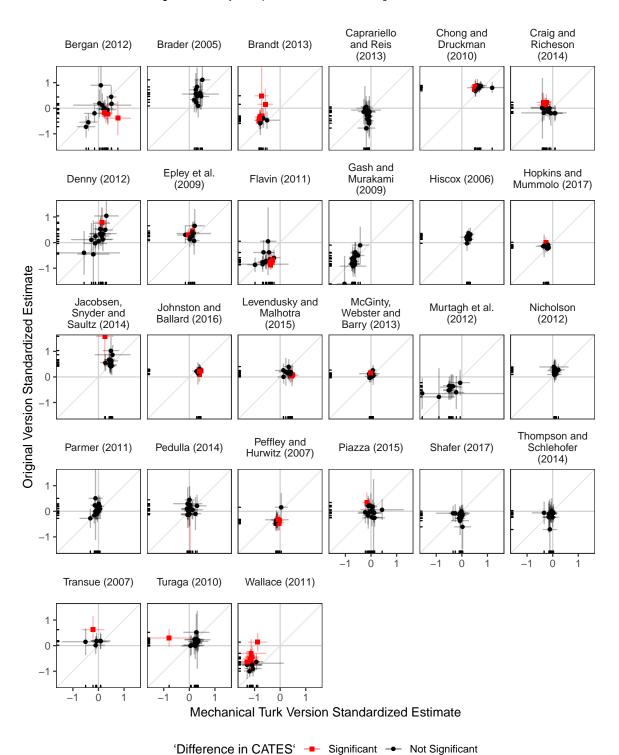
For each sample, we estimate uncertainty under a finite population model using HC2 robust standard errors (20). This approach does not account for the uncertainty associated with imagining that Mechanical Turk respondents are sampled as a cluster from a larger population. We estimate the

standard error of the difference-in-CATEs as $\sqrt{\widehat{SE_1}^2 + \widehat{SE_2}^2}$ and conduct hypothesis tests under a normal approximation. We deem a difference statistically significant if the p-value is less

'Difference in CATES' → Significant → Not Significant

Fig. 1. Across-Study Correspondence of Conditional Average Treatment Effects

Fig. 2. Within-Study Correspondence of Conditional Average Treatment Effects



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Table 1. Across-Study Correspondence of Conditional Average Treatment Effects

Covariate Class	Slope (SE)	95% CI	N Comparisons
Age: 18 - 39	0.82 (0.08)	[0.51, 1.12]	27
Age: 40 - 59	0.86 (0.08)	[0.55, 1.17]	27
Age: More than 60	0.95 (0.13)	[0.61, 1.29]	26
Less than College	0.93 (0.06)	[0.61, 1.25]	26
College	0.87 (0.10)	[0.46, 1.29]	26
Graduate School	0.72 (0.13)	[0.28, 1.15]	26
Men	0.87 (0.07)	[0.49, 1.25]	26
Women	0.91 (0.07)	[0.61, 1.20]	26
Liberal	0.71 (0.11)	[0.37, 1.05]	20
Moderate	1.01 (0.11)	[0.49, 1.53]	20
Conservative	0.75 (0.09)	[0.52, 0.99]	20
Democrat	0.88 (0.07)	[0.50, 1.26]	24
Independent	0.94 (0.16)	[0.19, 1.68]	23
Republican	0.86 (0.08)	[0.63, 1.08]	24
Nonwhite	0.95 (0.11)	[0.45, 1.46]	25
White	0.92 (0.06)	[0.66, 1.18]	27

Note: Slopes are estimated via Deming regression with unequal (estimated) variances and the 95% Cls are estimated via the jackknife.

average treatment effects is low treatment effect heterogeneity. Table 2 also presents the p-values from joint hypothesis tests against the null hypothesis of no differences in treatment effect heterogeneity by sample. We conduct the test by obtaining the F-statistic from a comparison of two nested models. The first model is ordinary least squares regression of the outcome on treatment, a sample indicator, covariates, and complete sets of interactions between treatment and covariates and the sample indicator and covariates. The second model adds to these terms an interaction of the sample indicator with the treatment by covariate interactions. The F-statistic is an omnibus summary of the extent to which the pattern of treatment effect heterogeneity differs by sample. Consistent with Figure 2, we fail to reject the null hypothesis most of the time (25 of 27 opportunities), indicating that whatever heterogeneity in treatment effects there may be, the patterns do not differ greatly across samples. The power of these Ftests to detect differences in heterogeneity varies across study pairs and in some cases may be quite low. We do not regard the failure to reject the null as an affirmation of no difference, but this finding bolsters our case that the main explanation for strong correspondence in both SATEs and CATEs is low treatment effect heterogeneity.

4. Discussion

Different samples will yield similar SATEs when either: (A) treatment effects are mostly homogeneous or (B) any effect heterogeneity is orthogonal to sample selection. Drawing on a fine-grained analysis of 27 pairs of survey experiments conducted on representative and non-representative samples and various methods of assessing the pattern of effect heterogeneity in each study, we have shown that effect heterogeneity is typically limited, so we conclude that treatment effect homogeneity is the best explanation for the correspondence of SATEs across samples.

As a result, the convenience samples we analyze provide useful estimates not only of the PATE but also of subgroup CATEs. The reason for this is that there appears to be little effect heterogeneity — as seen in the tight clustering of CATEs in each panel of Figure 2. Lacking such heterogeneity, any subgroup provides a reasonable estimate of not only the CATE but the PATE as well. In cases where some heterogeneity appears to be present, CATEs in each study pair rarely differ substantially from one another. Our results indicate that even descriptively unrepresentative samples constructed with no design-based justification for generalizability still tend to produce useful estimates not just of the SATE but also of subgroup CATEs that generalize quite well.

Important caveats are in order. First, we have not considered all possible survey experiments, let alone all possible experiments in other modes or settings. Our pairs of studies were limited to those conducted in an online mode on samples of United States residents. However, this set of studies is also quite comprehensive, drawing from multiple social science disciplines, utilizing a variety of experimental stimuli and outcome question formats. The studies are also drawn not just from published research (which we might expect to be subject to publication biases) but from a sample of experiments fielded by Time-Sharing Experiments for the Social Sciences.

Second, because we can never perfectly know the variation in treatment effects, our analysis of heterogeneity is limited by both the set of covariates that are available for direct comparison between samples and any measurement error in those covariates. We made several decisions about coarsening of covariates (for example, comparing whites to members of all other racial and ethnic groups) that reflected the need for a minimum level of measurement precision. Accordingly, our results may mask possible moderators of treatment effects (though we would note that the low levels of heterogeneity according to the covariates we were able to measure leads us to be skeptical of predictions of high levels of unmodeled effect heterogeneity). Our reliance on existing studies as the basis for our empirics is important because it means that we are evaluating the degree and pattern of effect heterogeneity using the types of samples and set of covariates typically used in survey-experimental research. Additional and more precisely measured covariates might have allowed for detection of more complex patterns of effect heterogeneity, but surveyexperimental research rarely offers such detail.

Third, the subgroup samples we analyzed were relatively small. While we may be well-powered to estimate a SATE, these studies were not necessarily designed to detect any particular source of effect heterogeneity. Larger sample sizes, oversampling of rare populations, and more precise measurement of covariates would have allowed the detection of smaller-sized variations in effect sizes across groups, but researchers rarely have access to larger samples than those used here. We are confident that larger sample sizes would turn up strong evidence that the pattern of heterogeneity differs across samples. We would argue, however, that the need for such large samples indicates that whatever the differences may be, they are not large.

Finally, this discussion of generalizability has been focused exclusively on who the subjects (or units) of the experiments are and how their responses to treatment may or may not vary. The "UTOS" framework (49, 50) identifies four dimensions of external validity: units, treatments, outcomes, and setting. In our study, we hold treatments, outcomes, and setting constant by design. We ask, "what happens if we run the same experi-

Table 2. Within-Study Correspondence of Conditional Average Treatment Effects

Study	Original N	MTurk N	Slope (SE)	95% CI	N Comparisons	Joint F -test p -value
Bergan (2012)(22)	1206	1913	0.75 (0.20)	[0.37, 1.12]	16	0.09
Brader (2005)(23)	280	1709	3.56 (1.68)	[-30.74, 37.86]	12	0.69
Brandt (2013)(24)	1225	3131	4.49 (1.96)	[-6.25, 15.23]	13	0.20
Caprariello (2013)(25)	825	2729	-4.38 (2.00)	[-7.83, -0.93]	16	0.63
Chong and Druckman (2010)(26)	958	1400	0.17 (0.18)	[-0.58, 0.92]	13	0.61
Craig and Richeson (2014)(27)	608	847	-0.95 (0.36)	[-1.56, -0.34]	16	0.24
Denny (2012)(28)	1733	1913	2.83 (1.04)	[1.19, 4.47]	16	0.59
Epley et al. (2009)(29)	1019	1913	0.68 (0.64)	[-2.52, 3.88]	10	0.14
Flavin (2011)(30)	2015	2729	0.23 (0.20)	[-0.15, 0.62]	16	0.06
Gash and Murakami (2009)(31)	1022	3131	2.78 (1.01)	[1.59, 3.96]	16	0.73
Hiscox (2006)(32)	1610	2972	2.50 (1.07)	[0.94, 4.07]	16	0.96
Hopkins and Mummolo (2017)(33)	3266	2972	-1.84 (0.85)	[-4.06, 0.37]	16	0.27
Jacobsen, Snyder and Saultz (2014))(34)	1111	3171	-4.73 (1.99)	[-8.32, -1.14]	16	0.09
Johnston and Ballard (2016)(35)	2045	2985	0.13 (0.53)	[-0.26, 0.53]	16	0.06
Levendusky and Malhotra (2015)(36)	1053	1987	-0.16 (0.35)	[-1.50, 1.19]	16	0.01
McGinty, Webster, and Barry (2013)(37)	2935	2985	2.53 (1.10)	[1.09, 3.97]	16	0.72
Murtagh et al (2012)(38)	2112	3131	0.34 (0.34)	[-0.20, 0.88]	10	0.98
Nicholson (2012)(39)	781	1099	-23.05 (16.21)	[-396.69, 350.58]	12	0.94
Parmer (2011)(40)	521	3277	1.71 (0.75)	[-0.12, 3.54]	16	0.61
Pedulla (2014)(41)	1407	1913	-57.93 (17.90)	[-363.42, 247.55]	15	0.73
Peffley and Hurwitz (2007)(42)	905	1285	2.23 (1.17)	[-1.08, 5.54]	13	0.19
Piazza (2015)(43)	1135	3171	-2.15 (0.74)	[-3.83, -0.47]	16	0.81
Shafer (2017)(44)	2592	2729	-24.13 (9.75)	[-162.31, 114.05]	16	0.49
Thompson and Schlehofer (2014)(45)	591	3277	0.24 (0.60)	[-1.08, 1.56]	16	0.68
Transue (2007)(46)	345	367	-1.67 (1.02)	[-7.52, 4.17]	7	0.29
Turaga (2010)(47)	774	3277	1.44 (0.54)	[-0.86, 3.74]	16	0.73
Wallace (2011)(48)	2929	2729	4.74 (1.86)	[-7.96, 17.43]	16	0.00

Note: Slopes are estimated via Deming regression with unequal (estimated) variances and the 95% CIs are estimated via the jackknife. The p-values in the last column are derived from a joint F-test against the null of no differences in heterogeneity by sample.

ment on different people," but not "are the causal processes studied in the experiments the ones we care about outside the experiments?" This second question is clearly of great importance but is not one we address here.

Perhaps the most controversial conclusion that could be drawn from the present research is that we should be more suspect of extant claims of effect moderation. A common posthoc data analysis procedure is to examine whether subgroups differ in their apparent response to treatment. We find only limited evidence that such moderation occurs and when it does, the differences in effect sizes across groups are small. The response to this evidence should not be that any convenience sample can be used to study any treatment without concern about generalizability (51) but rather that debates about generalizability and replication must focus on the underlying causes of replication and nonreplication, among these most importantly, the variation in treatment effects across experimental units.

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