ORIGINAL ARTICLE

Partially pooled propensity score models for average treatment effect estimation with multilevel data

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Funding information
Institute of Education Sciences, Grant/Award Number: R305D150001; National Institute of Mental Health, Grant/Award Number: R01MH115487

Abstract
Causal inference analyses often use existing observational data, which in many cases has some clustering of individuals. In this paper, we discuss propensity score weighting methods in a multilevel setting where within clusters individuals share unmeasured confounders that are related to treatment assignment and the potential outcomes. We focus in particular on settings where models with fixed cluster effects are either not feasible or not useful due to the presence of a large number of small clusters. We found, both through numerical experiments and theoretical derivations, that a strategy of grouping clusters with similar treatment prevalence and estimating propensity scores within such cluster groups is effective in reducing bias from unmeasured cluster-level covariates under mild conditions on the outcome model. We apply our proposed method in evaluating the effectiveness of centre-based pre-school programme participation on children’s achievement at kindergarten, using the Early Childhood Longitudinal Study Kindergarten data.

KEYWORDS
heterogeneous treatment effect, propensity scores, weighting, unmeasured confounder

This work was conducted while Dr. Lee was a post-doctoral fellow at Johns Hopkins Bloomberg School of Public Health.
1 | INTRODUCTION

Human subjects are often clustered in communities, schools, hospitals, online social groups, etc., sharing the same environmental factors, services, interventions or physical facilities. This clustering also often makes data collection more convenient as subjects are close to one another in physical or virtual space (Arpino & Mealli, 2011; Leite et al., 2015; Rabe-Hesketh & Skrondal, 2006). As a result, clustered observational data are widely used to investigate causal relationships between treatments and outcomes that occur at the individual level, but often without complete knowledge of shared characteristics and contextual backgrounds.

When a cluster-level characteristic influences both the treatment and outcome of interest, it confounds the causal effect (Greenland et al., 1999) by inducing a spurious association between the two variables. In addition, a cluster-level characteristic may also bring about systematic variation across clusters in the direction and/or magnitude of the causal effect (Ten Have et al., 2004; Van der Weele & Robins, 2007), in which case it is called an effect modifier. In this paper, we consider the effect of centre-based pre-school education on math proficiency in elementary school as a motivating example. We estimate the average causal effect based on the sample of students clustered in multiple schools. In this case, school-level covariates (e.g. the region’s educational infrastructure) may confound the relationships between the treatment and the outcome and also modify the treatment effect. For instance, the centre-based pre-school education effect might be more evident for schools in particular regions than those in other areas due to differences in attitude about education or in the resources put into schools in that region.

Estimating an average treatment effect via propensity score analysis requires balancing the distribution of confounders and effect modifiers between different treatment arms to remove bias in the causal estimate. Unfortunately, cluster-level characteristics are often unmeasured and thus not balanced between different arms (He, 2018; Thoemmes & West, 2011; Yang, 2018), which could lead to bias. This is called the unmeasured context problem (Arpino & Mealli, 2011). Here we focus on how to tackle bias due to unmeasured contextual factors in propensity score analysis with clustered observational data.

Arguably, the ideal way to eliminate bias due to unmeasured cluster-level characteristics is to separately fit one propensity score model for each cluster and to do propensity score matching, weighting or subclassification separately within each cluster (Kim & Steiner, 2015) —we call this cluster-specific propensity score estimation and use. However, in many multilevel data sets, the cluster sample sizes are too small or too variable to allow doing this for each cluster. For instance, in the Early Childhood Longitudinal Study’s Kindergarten (ECLS-K) data set (a longitudinal nationally representative cohort of children followed starting from kindergarten (Tourangeau et al., 2009)), the first, second and third quartiles of cluster (school) sample size are 1, 18 and 22, respectively. Over a quarter of the children in the sample form their own singleton clusters. About 4% of the participating schools have only 2–5 children in the sample, and another 25% of the participating schools have 6–20 children in the sample. These cluster sample sizes are not sufficient for the number of covariates that we would usually need to adjust for. It is thus no surprise that common practice is to estimate a single propensity score model pooling observations from all the clusters and use them over all individuals in the data (which we call fully pooled propensity score estimation and use), even though this often suffers from confounding by unmeasured context.

Small cluster sizes can lead the perhaps default fixed effect approach to not be feasible. In this paper, we propose an alternative strategy that overcomes this small cluster sample size problem by grouping clusters with similar treatment prevalence into several cluster groups and then estimating propensity scores separately within each group. We call this partially pooled propensity...
score estimation. Once the clusters are grouped, any propensity score estimation approach can be used within those groups. This strategy allows variation in propensity score models across cluster groups, resulting in better control for unmeasured contextual factors and less biased causal effect estimates under the mild assumptions on the outcome model.

The paper proceeds as follows. Section 2 describes the setting, introduces the notation, and presents the estimand and assumptions. Section 3 introduces the data example and presents an exploratory analysis of a cluster-level covariate to motivate the proposed method. Section 4 reviews related work on propensity score methods with clustered data and unmeasured cluster-level confounding. Section 5 presents our proposed propensity score estimation method and the theoretical properties of two relevant inverse probability weighting estimators. Section 6 reports simulation results on the proposed method. Our method is finally applied to the ECLS-K data in evaluating the effect of centre-based pre-school education on kindergarten math scores in Section 7. We conclude the paper with a discussion. All the relevant code can be found at the first author’s github repository\(^1\).

\section{Setting, Estimand and Assumptions}

We consider clustered data in a two-level structure comprised of multiple clusters, each with possibly multiple individuals. Given this two-level structure, we index observations using dual subscripts: \(h\) indexing the cluster (\(h = 1, 2, \ldots, H\), where \(H\) is the number of clusters in the population) and \(k\) indexing the individual within cluster (\(k = 1, 2, \ldots, n_h\), where \(n_h\) is the sample size from cluster \(h\)). For individual \(k\) in cluster \(h\), \(Z_{hk}\) denotes the treatment, and \(Y_{hk}\) the outcome. Here treatment and outcome are both at the individual level. Let us assume that treatment is binary, that is \(Z_{hk} = 1\) if treated, and \(= 0\) if not. \(X_{hk} \in \mathbb{R}^p\) and \(V_h \in \mathbb{R}^q\) are observed pre-treatment covariates at the individual-level and cluster-level, respectively. Both \(X_{hk}\) and \(V_h\) can act as confounders, influencing both \(Z_{hk}\) and \(Y_{hk}\) as depicted in the causal diagram in Figure 1. Let \(C_{hk}\) denote the cluster the individual belongs to; that is, \(C_{hk} = h\). In summary, we are given a set of observations \(O_n = \{(Y_{hk}, Z_{hk}, X_{hk}, V_h, C_{hk}) : h = 1, 2, \ldots, H; k = 1, 2, \ldots, n_h, n = \sum_{h=1}^{H} n_h\}\).

In addition to these observed variables, \(U_h\) denotes an unobserved cluster-level variable that is a confounder and possibly also an effect modifier. \(U_h\) may represent the cluster’s contextual (e.g.

\(^1\)https://github.com/youjin1207/PartialIPW
a school’s distance from the nearest city) or compositional (e.g. percent of the school’s children who live under the poverty line) characteristics that are not captured in the data set. In this work, we assume that there is at least one unmeasured cluster-level confounder \( U_h \), which is associated both with the treatment and outcome even after conditioning on \( \mathbf{X}_{hk} \) and \( \mathbf{V}_h \).

To formally represent our target estimand, we use the potential outcomes framework (Holland, 1986; Rubin, 1974). We assume the stable unit treatment value assumption (SUTVA) (Rubin, 1980), which includes no interference (i.e. the treatment of one individual does not affect the outcome of others) and irrelevance of treatment variation. In multilevel setting, the SUTVA needs a careful attention; in our case, we assume that outcomes observed within cluster are possibly correlated with each other due to shared cluster level confounders, but the treatment received by one subject does not have a direct effect on the outcome of others even in the same cluster; thus, SUTVA holds. Under this assumption, each individual has a potential outcome \( Y_{hk}(1) \) if treated and a potential outcome \( Y_{hk}(0) \) if untreated. The treatment effect for the individual is defined as the difference between these two potential outcomes, \( \tau_{hk} := Y_{hk}(1) − Y_{hk}(0) \). Our target estimand, \( \tau \), is the average treatment effect (ATE) for the population conditional on current cluster membership, that is the average of the individual treatment effects with equal weights over all the individuals in the population conditional on the observed cluster assignments (Hong & Raudenbush, 2006):

\[
\tau = \mathbb{E} [ Y_{hk}(1) − Y_{hk}(0) ].
\]

Here the target estimand \( \tau \) would be identified if we were to observe \( Z_{hk}, Y_{hk}, \mathbf{X}_{hk}, \mathbf{V}_{hk} \), and also \( U_h \). This is based on the following assumptions:

1. **Consistency**: \( Y_{hk} = Y_{hk}(z) \) if \( Z_{hk} = z \) for \( z = 0, 1 \);
2. **Positivity**: \( 0 < p(Z_{hk} = 1, |\mathbf{X}_{hk} = \mathbf{x}, \mathbf{V}_h = \mathbf{v}, U_h = u) < 1 \) for all \( (\mathbf{x}, \mathbf{v}, u) \) in the support of \( (\mathbf{X}_{hk}, \mathbf{V}_h, U_h) \);
3. **Unconfoundeness**: \( Y_{hk}(z) \perp\!
\perp Z_{hk} | \mathbf{X}_{hk}, \mathbf{V}_h, U_h \) for \( z = 0, 1 \).

Our challenge is that identification of \( \tau \) requires \( U_h \). Estimators that ignore this unobserved covariate are biased. Throughout this paper, we assume that we observe all of the individual-level covariates \( \mathbf{X}_{hk} \) and some cluster-level covariates \( \mathbf{V}_h \), but we do not observe \( U_h \).

For our proposed method to be effective in reducing bias from \( U_h \), in addition to (a)-(c), we also assume an additive effect of confounding and modification due to any form of \( U_h \) on \( Y(0) \) and \( Y(1) \). That is, \( Y_{hk}(0) − Y_{h’k}(0) = g(U_h) − g(U_{h’}) \) and \( (Y_{hk}(1) − Y_{hk}(0)) − (Y_{h’k}(1) − Y_{h’k}(0)) = f(U_h) − f(U_{h’}) \) for \( h \neq h’ \). No distributional assumptions regarding the treatment assignment are required. More details are discussed in Section 5.

## 3 Motivating Application

As an illustrative example, consider an analysis of the ECLS-K (1998–1999) data (Tourangeau et al., 2009) mentioned in the Introduction. The data is publicly available at https://nces.ed.gov/ecls/. This is a longitudinal cohort with children nested in schools in the United States. It has been widely used to study the effect of early education (Adelson et al., 2012; Carlson et al., 2008; Hong & Raudenbush, 2005) and behavioural interventions (Gershoff et al., 2018; Xu & Gulosino, 2006) on child development. We consider the effect of centre-based pre-school education on math proficiency as one illustrative example. Here centre-based pre-school education
means a child’s exposure to any centre-based childcare before kindergarten. There has been literature that reveals positive associations between exposure to centre-based education and child development. Often these associational findings have an impact on government investments in pre-school programmes (Elango et al., 2015). Therefore, it is essential to fully understand their impacts on young children. However, whether these associations imply causal effects is still intangible (Gottfried, 2015; Loeb et al., 2007).

In this example, \( Z_{hk} \) indicates whether child \( k \) in school \( h \) had primarily attended centre-based pre-school \( (Z_{hk} = 1) \) or primarily received parental care \( (Z_{hk} = 0) \) before kindergarten, and \( Y_{hk} \) is the child’s observed math score in the fall of kindergarten (generally around age 5 in the United States). We restrict the sample (i) to the 778 schools with at least one treated child and one control child, and (ii) to the children with complete data on a set of observed covariates. These covariates (e.g. demographics, family characteristics, census region, etc.) are considered confounders that should be adjusted for, and we describe them in detail in the supplementary material. This analysis takes the sample as the inference population; that is, our goal is to estimate the ATE for the children in the sample.

Within each cluster, individuals share the same value of each cluster-level variable. Hence a cluster-level confounder affects the treatment assignment probability (and the outcomes) for all individuals in the cluster in the same way given the same observed individual-level characteristics. Consequently, cluster-level confounders are generally associated with the cluster’s treatment prevalence and average outcome in the absence of the treatment (i.e. average outcome under control). In our example, we observe these two associations in the variable ‘census region’. Table 1 shows that census regions with higher treatment prevalence are also those with higher average outcome under control, unaffected by the treatment (see the left panel of Figure 2). If we still observe similar patterns in the associations even after conditioning on all the measured confounders, that suggests the presence of contextual factors associated with census region that are confounders or effect modifiers.

Even though the census regions are often known in survey data, let us hypothetically presume that we do not observe this variable and treat is as \( U_h \). The tendency of cluster-level confounders (e.g. census region) to be associated with treatment prevalence provides a hint for a solution. The right panel of Figure 2 illustrates the relationship between census region and treatment prevalence among schools stratified by each school’s treatment prevalence.

![Figure 2](https://wileyonlinelibrary.com)
For each stratum, the distribution of census region varies. Of the schools with treatment prevalence under 30% (i.e. <30% of the children had attended centre-based pre-school), half were located in the West. Among schools with treatment prevalence over 80%, however, less than one fifth were in the West, while most were in the South or the Midwest. This implies that schools that are similar in treatment prevalence are more likely to be located in the same census region given that they are similar with respect to the distribution of individual-level confounders.

Building on this insight, our proposed method pools clusters (e.g. schools) into several groups so that within each group, the clusters have similar observed treatment prevalence. Even though the observed treatment prevalence reflects the influence of a collection of observed and unobserved covariates that may or may not be confounders, it still contains information about the unobserved confounder $U_h$. With this grouping scheme, clusters with similar $U_h$ values are more likely to be grouped together than clusters with distant $U_h$ values. This is expected to reduce bias in the causal estimate due to the omitted $U_h$ through the propensity scores that at least partially take into account the unobserved $U_h$. In this work, we consider the number of groups fixed for simplicity, but one may choose the number of groups based on data.

4 | RELATED WORK

There are two stages of using propensity score methods to estimate causal effects in non-experimental settings: estimation and use. The focus of the current paper is propensity score estimation. Of the different ways the estimated propensity scores can be used in estimating the ATE, we only examine inverse probability weighting (IPW), to focus attention on the benefit of improved propensity score estimation as one example of the use of propensity scores in outcome analysis. Given this scope of the investigation, we review relevant strategies for propensity score estimation and briefly introduce IPW estimation in a multilevel setting, followed by a summary of existing methods designed specifically to handle unmeasured cluster-level confounding.

4.1 | Propensity score estimation with clustered data

Propensity score estimation with fixed and/or random effects has been often used in practice in multilevel settings, as compared to cluster-specific propensity score estimation; this is mostly due to small cluster size and stringent identification conditions within clusters, for example cluster-specific propensity score estimation requires $Z_h = (Z_{h,1}, \ldots, Z_{h,n_h}) \neq 0, 1$ for all clusters. Instead, multilevel propensity score models incorporate the clustered structure with fixed or random cluster effects, thus allowing some degree of model heterogeneity across clusters. Many
studies have shown that adding fixed or random effects in a propensity score model and/or outcome model improves performance (Arpino & Mealli, 2011; Hong & Raudenbush, 2006; Li et al., 2013; Thoemmes & West, 2011). In our context with unobserved $U_h$, a propensity score model with fixed or random intercepts seems suitable, as the varying intercepts help absorb the effect of $U_h$ on treatment assignment. Therefore, if the cluster sizes are large enough, using a cluster fixed effects model might be the most reasonable approach.

However, these models still have practical challenges. A fixed effects model requires a large number of parameters ($H-1$ parameters for any association, for example intercept or slope coefficient, that is allowed to vary across the clusters). This makes the model unstable or not identified if clusters are very small, as there may not be enough information to estimate cluster-specific parameters (Li et al., 2013). This is the same problem with cluster-specific propensity score estimation, just to a lesser degree. Random effects models use fewer parameters than fixed effects models, and cluster-specific effects for small clusters simply collapse to the population mean. However, a random effects model requires a distributional assumption for each cluster-varying parameter, and this assumption may not be correct. Perhaps more importantly, a random effects model assumes no correlation between the predictors in the model and the random effects, that is, no correlation between observed and unobserved covariates (He, 2018; Li et al., 2013; Skinner, 2011; Yang, 2018), an assumption that is unlikely to hold in practice.

4.2 Outcome analysis for the ATE

This discussion of the outcome analysis presupposes that propensity scores have been estimated based on the whole observed data with random effects; we refer to this propensity score estimation as (full-RE), which is standard practice for most of propensity score-based estimators with a small cluster size. We focus our outcome analysis on the IPW estimation, but this discussion of how much locally the treatment effects are measured first can be extended to other estimators, for example doubly robust estimators, that use the propensity scores. Denote the treatment probability of the individual by $e_{hk}: = p(Z_{hk} = 1 | X_{hk}, V_h, U_h)$. Denote the estimated propensity score by $\hat{e}_{hk}(\text{full-RE})$. IPW estimation is based on the inverse probability of assigned treatment weight, $w_{hk} = Z_{hk}\hat{e}_{hk}^{-1} + (1 - Z_{hk})(1 - e_{hk})^{-1}$, in this case estimated by $\hat{w}_{hk}(\text{full-RE}) = Z_{hk}\hat{e}_{hk}(\text{full-RE})^{-1} + (1 - Z_{hk})(1 - \hat{e}_{hk}(\text{full-RE}))^{-1}$.

We discuss two such estimators. The first is the simple IPW estimator that is agnostic to whether the setting is multilevel. It involves weighting each individual by $\hat{w}_{hk}(\text{full-RE})$ and taking the difference between the weighted mean outcome of treated individuals and the weighted mean outcome of untreated individuals to estimate $\tau$. Formally,

$$\hat{\tau}_{\text{(full-RE, full)}} = \frac{\sum_{h=1}^{H} \sum_{k=1}^{n_h} Z_{hk}\hat{w}_{hk}(\text{full-RE}) Y_{hk}}{\sum_{h=1}^{H} \sum_{k=1}^{n_h} Z_{hk}\hat{w}_{hk}(\text{full-RE})} - \frac{\sum_{h=1}^{H} \sum_{k=1}^{n_h} (1 - Z_{hk})\hat{w}_{hk}(\text{full-RE}) Y_{hk}}{\sum_{h=1}^{H} \sum_{k=1}^{n_h} (1 - Z_{hk})\hat{w}_{hk}(\text{full-RE})}.$$  \hspace{1cm} (2)

We index this estimator (and all other estimators of $\tau$) by double subscripts: the first component indicates the propensity score estimation strategy, and the second indicates the IPW strategy for ATE estimation given the estimated propensity score weights. $\hat{\tau}_{\text{(full-RE, full)}}$ involves fully pooled propensity score estimation with random effects (intercepts), followed by fully pooled (or marginal) IPW. The latter labelling is appropriate, as the weighted averaging of the outcomes pools all treated individuals and all untreated individuals over the full sample. A marginal IPW would result in consistent
estimation of $\tau$ had the estimated weights been consistent for the correct weights $w_{hk}$ under the identification assumptions; but the problem here is that the estimated propensity scores and weights, $\hat{\tau}_{hk(\text{full}-\text{RE})}$ and $\hat{\tau}_{hk(\text{full}-\text{RE})}$, do not fully take into account the unobserved $U_h$, leading to a biased estimate $\hat{\tau}_{(\text{full}-\text{RE}, \text{full})}$.

Instead of marginal IPW, in the multilevel setting, Li et al. (2013) suggest a cluster-weighted IPW estimator that combines estimates of cluster ATEs ($\tau_h := E[Y_{jk} | Z_{jk} = h]$). Using the same propensity scores estimated via fully pooled models with random effects, $\tau_h$ is estimated for each cluster:

$$\hat{\tau}_{h(\text{full}-\text{RE})} := \frac{\sum_{k=1}^{n_h} Z_{hk} \hat{\tau}_{hk(\text{full}-\text{RE})}}{\sum_{k=1}^{n_h} \hat{\tau}_{hk(\text{full}-\text{RE})}} = \frac{\sum_{k=1}^{n_h} (1 - Z_{hk}) \hat{\tau}_{hk(\text{full}-\text{RE})}}{\sum_{k=1}^{n_h} (1 - Z_{hk}) \hat{\tau}_{hk(\text{full}-\text{RE})}}. \quad (3)$$

Then these cluster-specific effects are averaged over the clusters to estimate $\tau$, weighted by the sum of the propensity score weights for each cluster, $\hat{\tau}_{(\text{full}-\text{RE}, \text{cluster})} := \frac{\sum_{h=1}^{H} \hat{\tau}_{h(\text{full}-\text{RE})}}{\sum_{h=1}^{H} \hat{\tau}_{h(\text{full}-\text{RE})}}. \quad (4)$

The second subscript of $\hat{\tau}_{(\text{full}-\text{RE}, \text{cluster})}$ reflects the use of cluster-specific IPW. This estimator requires that each cluster has at least one treated and one untreated individual, that is $Z_h = (Z_{h1}, ..., Z_{hn_h}) \neq 0, 1$ for all clusters $h = 1, 2, ..., H$. Note that this requirement is specific to this estimator, not a condition required for identification of $\tau$.

Despite additional conditions for estimation, there are several benefits of $\hat{\tau}_{(\text{full}-\text{RE}, \text{cluster})}$ for our unobserved $U_h$ setting. We found in our simulations that without effect modification, that is, when treatment effects are homogeneous across the clusters, $\hat{\tau}_{(\text{full}-\text{RE}, \text{cluster})}$ is protective against confounding due to unmeasured cluster-level characteristics. This is because the estimation of cluster-specific ATE does not suffer from imbalance in $U_h$. However, the relative weights among clusters, $\{\hat{\tau}_{h(\text{full}-\text{RE})}; h = 1, 2, ..., H\}$, are still biased. Under effect modification where $\tau_h$ varies across clusters, the errors in the estimated cluster weights $\hat{\tau}_{h(\text{full}-\text{RE})}$ due to missing $U_h$ in the propensity score model might over- or under-estimate the relative influence of $\tau_h$ in evaluating the overall ATE $\tau$, which leads to bias in ATE even if each of $\tau_h$’s were correctly estimated.

4.3 Propensity score methods for unmeasured cluster-level confounding

Yang (2018) and He (2018) proposed propensity score methods that incorporate additional information related to $U_h$, thus improving upon the estimator in (2), under the same assumption we make that there are unobserved cluster-level confounders but no unobserved individual-level confounders.

Yang (2018) proposes a calibration strategy that uses propensity score weights that satisfy the following conditions: (i) for each observed covariate, the weighted sum in each treatment arm equals the unweighted marginal sum and (ii) in each cluster, the treated individuals’ weights and the untreated individuals’ weights both sum to $n_h$.

Using these weights results in pseudo treated and untreated populations that replicate the means of observed covariates and of $U_h$ in the
inference population. Consistent $\tau$ estimation using these weights is contingent on the assumption that both the true treatment assignment and outcome models are generalized linear models.

He (2018), on the other hand, proposes conditional propensity score estimation based on sufficient statistics. Under certain conditions, He (2018) shows that conditioning on some function of the cluster’s treatment assignment vector (in addition to the observed covariates) is sufficient to guarantee ignorability in the presence of unobserved $U_h$. Assuming a logit treatment assignment model, one such sufficient statistic turns out to be the number of treated individuals in the cluster, $s_h := \sum_{k=1}^{n_h} Z_{hk}$. He (2018) estimates propensity scores via maximum likelihood conditional on this statistic. Marginal IPW based on the estimated conditional propensity scores is shown to reduce bias due to the unmeasured $U_h$.

5 | METHODS

We now describe our proposed propensity score estimation method in some detail, and point out how it relates to existing methods. We then present two IPW estimators based on the estimated propensity scores, and discuss their theoretical properties.

5.1 | Selective pooling of cluster groups

Our method relies on pooling information. This idea in fact has been used for similar purposes to ours. For example, Stuart and Rubin (2008), Arpino and Cannas (2016), and Zubizarreta and Keele (2017) each provide strategies to match individuals across clusters to increase comparability in individual-level covariates. In our context, instead of pooling information from all the clusters indiscriminately, we selectively group similar clusters to guarantee large enough samples for propensity score estimation. This method therefore overcomes the small cluster sample size problem in the sense that it allows more variation in propensity score models than the alternative of a fully pooled model in this case. Group-stratified propensity score estimation requires positivity to hold within cluster groups, which is stricter than unconditional positivity, but less strict than the positivity within clusters required by cluster-specific propensity score estimation.

As mentioned in Section 3, our selective pooling is based on the cluster’s treatment prevalence, $p_h := \sum_{k=1}^{n_h} Z_{hk} / n_h$, leveraging $U_h$’s association with treatment. Within the groups that are pooled, the variance of $U_h$ is likely smaller than its marginal variance, which means group-specific propensity score models are less misspecified than a fully pooled model. This method essentially taps into the same information about $U_h$ as Yang (2018) and He (2018): for each cluster, the number of treated individuals (used by He (2018)) carries the same information as the cluster’s treatment prevalence; and Yang (2018)’s tying the sums of the cluster’s treated and control weights to the cluster sample size is another way of using that same information. Unlike this prior work, our proposed method is not based on assumptions regarding specific treatment assignment or outcome models.

To update notation, let $H_g$ denote the set of clusters that are grouped into group $g$, $(g = 1, ..., G, G \leq H)$. Let $\hat{\epsilon}_{hk(\text{group})}$ denote the propensity score estimated for individual $k$ in cluster $h$ based on the propensity score model using all of the observed individual- and cluster-level covariates (e.g. $X_{hk}, V_h$) for the group that cluster $h$ is grouped in. Let $\hat{\omega}_{hk(\text{group})} := Z_{hk} \hat{\epsilon}_{hk(\text{group})}^{-1} + (1 - Z_{hk})(1 - \hat{\epsilon}_{hk(\text{group})})^{-1}$ denote the inverse probability weight of the individual based on $\hat{\epsilon}_{hk(\text{group})}$. 
There are many options for group selection that form multiple groups with similarity. In our case, to select $G$ groups out of $H$ clusters to minimize within-group distances in $p_h$, we use partitioning around medoids (PAM) (Park & Jun, 2009; Van der Laan et al., 2003). Other clustering methods, such as $k$-mean clustering (Hartigan & Wong, 1979) or the Bayesian nonparametric clustering (Teh et al., 2005), may also be applied. We will shortly discuss advantages of minimizing within-group distances in $p_h$'s. In this paper, we fix the number of groups to $G = 10$, but one may let data choose $G$ using the Elbow method (Thorndike, 1953) or information criteria such as Bayesian information criterion (BIC) (Hamerly & Elkan, 2004). Future work could investigate different methods to choose the number of groups.

5.2 Two IPW estimators based on the partially pooled propensity scores

Given that we estimate the propensity scores after pooling clusters into several groups with similar treatment prevalence, we consider a cluster-weighted and a group-weighted estimator, denoted by $\hat{\tau}_{(\text{group,cluster})}$ and $\hat{\tau}_{(\text{group,group})}$, respectively. When we include cluster random effects in the propensity score models, we denote these cluster-weighted and group-weighted estimators by $\hat{\tau}_{(\text{group-RE,cluster})}$ and $\hat{\tau}_{(\text{group-RE,group})}$, respectively. By using a cluster-weighted (or group-weighted) IPW estimator, we can reduce the impact of unmeasured $U_h$ in causal effect estimation—especially when $U_h$ modifies causal effects—by locally estimating the treatment effects from each cluster (or group of clusters with similar $p_h$) first and marginalizing them over clusters (groups) to estimate the ATE. Figure 3 provides a skeleton summary of these two estimators.

The cluster-weighted estimator $\hat{\tau}_{(\text{group-RE,cluster})}$ is a modification of $\hat{\tau}_{(\text{full-RE,cluster})}$ replacing fully pooled weights $\hat{\omega}_{hk(\text{full-RE})}$ with group pooled weights $\hat{\omega}_{hk(\text{group-RE})}$, both with random effects. The group-weighted estimator $\hat{\tau}_{(\text{group-RE,group})}$ using $\hat{\omega}_{hk(\text{group-RE})}$ is instead based on IPW at the group level.

\[
\hat{\tau}_{(\text{group-RE,group})} := \frac{\sum_{g=1}^{G} \hat{\omega}_{g(\text{group-RE})} \hat{\tau}_{g(\text{group-RE})}}{\sum_{g=1}^{G} \hat{\omega}_{g(\text{group-RE})}},
\]

| (1) Compute the observed treatment prevalence for each cluster, $p_h$ |
| (2) Select cluster groups to minimize within-group distances in $p_h$ |
| (3) Fit a propensity score model for each group with all observed covariates and random effects and estimate propensity scores |
| (4.a) Estimate cluster-specific ATEs ($\tau_h$) |
| (4.b) Estimate group-specific ATEs ($\tau_g$) |
| (5.a) Combine the clusters’ $\tau_h$ estimates to estimate the population ATE ($\tau$) |
| (5.b) Combine the groups’ $\tau_g$ estimates to estimate the population ATE ($\tau$) |

FIGURE 3 Flow chart for inverse probability weighting estimation of population average treatment effect using partially pooled propensity scores
where

\[ \hat{\tau}_{g(\text{group−RE})} := \frac{\sum_{h \in H} \sum_{k=1}^{n_h} Z_{hk} \hat{W}_{hk(\text{group−RE})} Y_{hk}}{\sum_{h \in H} \sum_{k=1}^{n_h} Z_{hk} \hat{W}_{hk(\text{group−RE})}} − \frac{\sum_{h \in H} \sum_{k=1}^{n_h} (1 − Z_{hk}) \hat{W}_{hk(\text{group−RE})} Y_{hk}}{\sum_{h \in H} \sum_{k=1}^{n_h} (1 − Z_{hk}) \hat{W}_{hk(\text{group−RE})}} \]  

(6)

estimates the ATE for group g, \( \tau_{g} := \mathbb{E}[\tau_{hk} \mid C_{hk} \in H_{g}] \); and the group weight \( \hat{W}_{g(\text{group})} \) is the sum of the weights of all the individuals in the group, \( \hat{W}_{g(\text{group−RE})} := \sum_{h \in H} \sum_{k=1}^{n_h} \hat{W}_{hk(\text{group−RE})} \). This estimator requires at least one treated and one untreated individual in each group, which is a milder condition required than for \( \hat{r}_{(\text{group−RE, cluster})} \) (one treated and one untreated each cluster). In practice, even though the \( \hat{r}_{(\text{group−RE, cluster})} \) estimator is less biased than the \( \hat{r}_{(\text{group−RE, group})} \) estimator, the \( \hat{r}_{(\text{group−RE, cluster})} \) may not be identified with small cluster size; we will discuss this issue further in Section 6.2. We now turn to examine more closely the advantages of minimizing within-group dissimilarity in treatment prevalence.

5.3 | Decomposition of IPW estimator

We further elaborate on the derivations for \( \hat{r}_{(\text{group, group})} \) under minimal distributional assumptions on outcomes to demonstrate why selectively pooling cluster groups with respect to similar treatment prevalence can reduce bias due to unmeasured context. We also make similar arguments about \( \hat{r}_{(\text{group, cluster})} \) in the supplementary material.

Assume a continuous outcome model where an arbitrary function of unmeasured cluster-level covariates has an additive effect on the outcome. Let us ignore observed covariates \( X_{hk} \) and \( V_{h} \) for simplicity. Consider the following data generating model for potential outcomes \( \{Y_{hk}(0), Y_{hk}(1)\} \) having \( U_{h} \) as a confounder and an effect modifier via some arbitrary functions \( g(U_{h}) \) and \( f(U_{h}). \) Here we do not put any restriction on these functions other than they are bounded. Note that we do not require any assumption on the distribution of treatment assignment here.

\[ Y_{hk}(z) = \beta_0 + g(U_{h}) + \kappa z + f(U_{h})z + \epsilon_{hk}, \quad \epsilon_{hk} \sim i.i.d. (\mu_{\epsilon} = 0, \sigma_{\epsilon}^2 < \infty), \]  

(7)

for \( z \in \{0, 1\}. \) Then we can represent \( \tau \) using two potential outcomes for each individual:

\[ \tau = \mathbb{E}[Y_{hk}(1) − Y_{hk}(0)] \]

\[ = \kappa + n^{-1} \sum_{h=1}^{H} n_{h} f(U_{h}). \]

Now consider a grouping method that \( H \) clusters were classified into \( G(\leq H) \) groups. Let \( p_{g} \) denote the treatment prevalence in cluster group \( g (g = 1, 2, ..., G) \) and \( p_{g}(h) := p_{g} I(h \in H_{g}) \); and define \( \delta_{h} := p_{h} − p_{g}(h) \) as the deviation of each cluster’s treatment prevalence from the average treatment prevalence of the group that the cluster belongs to. Note that pooling the clusters with similar treatment prevalence is equivalent to selectively pooling clusters to minimize the \( \delta_{h}. \)

We can demonstrate that a grouping method that minimizes \( \delta_{h} \) reduces bias from \( U_{h} \) in ATE estimation. Under a simple scenario, where no observed covariates are correlated with \( U_{h} \), consider the estimated weights \( \hat{W}_{hk(\text{group})} \) from the partially pooled propensity score model
that does not include \( U_h \) and ignores clusters. Then we have the following decomposition of \( \hat{\tau}_{(\text{group, group})} \):

\[
\hat{\tau}_{(\text{group, group})} = \left( \sum_{g=1}^{G} \hat{\nu}_g \right)^{-1} \sum_{g=1}^{G} \hat{\nu}_g \hat{\tau}_g \\
\approx \tau + \Lambda + \Delta,
\]

(8)

where

\[
\Lambda = n^{-1} \sum_{h=1}^{H} \{ n_h \delta_h (p_g^{-1}(h) + (1 - p_g(h))^{-1}) g(U_h) + n_h \delta_h p_g^{-1}(h) f(U_h) \}
\]

(9)
denotes the bias introduced by an unmeasured \( U_h \), and \( \Delta \) is a weighted sum of random errors \( \varepsilon_{hk} \)'s. In the presence of observed confounders \( \mathbf{X}_{nk} \) and \( \mathbf{V}_h \) that are included in the propensity score model, the expectation of the residual term \( \Delta \) converges to zero when \( E(\varepsilon_{hk} | \mathbf{x}_{nk}, \mathbf{v}_h, h \in H_g) = 0 \). In Equation (8), we have \( \approx \) instead of \( = \) because we approximate \( \hat{\nu}_{hk(\text{group})} \) only through the treatment prevalence in each group as a proxy for partially pooled propensity scores. Details about the derivation procedures as well as the remainder \( \Delta \) are provided in the Supplementary Material S1.

The above decomposition of \( \hat{\tau}_{(\text{group, group})} \) implies that the IPW estimator is a consistent estimator for \( \tau \) if we can claim that both \( \Lambda \) and \( \Delta \) converge to zero as the number of clusters increases. However, convergence of \( \Lambda \) is much more demanding as it denotes a systematic bias due to unmeasured context while convergence of error terms in \( \Delta \) directly comes from the assumption of random errors \( \varepsilon_{hk} \sim (0, \sigma^2) \). More specifically, given that \( f(U_h) \) and \( g(U_h) \) are bounded, \( \Lambda \) involves two terms, \( n^{-1} \sum_{h=1}^{H} n_h \delta_h / p_g(h) \) and \( n^{-1} \sum_{h=1}^{H} n_h \delta_h / (1 - p_g(h)) \). Under some regularity assumptions, to have \( \Lambda \to 0 \) as \( H \to \infty \), \( H^{-1} \sum_{h=1}^{H} \delta_h / p_g(h) \) and \( H^{-1} \sum_{h=1}^{H} \delta_h / (1 - p_g(h)) \) need to go to zero. Since \( p_g(h) \) and \( (1 - p_g(h)) \) are both positive, smaller \( \delta_h \) leads to smaller bias \( \Lambda \) at fixed values of \( p_g(h) \) and \( (1 - p_g(h)) \). We now discuss the bias term \( \Lambda \) in more detail.

### 5.4 Discussion on bias

Bias \( \Lambda \) in Equation (9) shows that grouping clusters with similar \( p_g \)'s reduces bias from \( f(U_h) \) and \( g(U_h) \) by forcing \( \delta_h \) to be small, relatively smaller than \( p_g(h) \) and \( 1 - p_g(h) \). We can infer from the first term in \( \Lambda \) that we need a smaller \( \delta_h \) when the treatment prevalence of the group is either small or large (i.e. small \( p_g(h) \) or small \( 1 - p_g(h) \)), and/or when the amount of confounding is large, that is large \( |g(U_h)| \); moreover, from the second term in \( \Lambda \), it is clear that we need a smaller \( \delta_h \) when the treatment prevalence of the group is small, that is small \( p_g(h) \), and/or when the amount of effect modification is large, that is large \( |f(U_h)| \).

One caveat in partial pooling cluster groups is that smaller \( \delta_h \), that is smaller deviation of a cluster's treatment prevalence from the group's treatment prevalence, might not compensate for extreme values of \( p_g^{-1}(h) \) or/(and \( (1 - p_g(h))^{-1} \)). In fact grouping by similar treatment prevalence often leads to an extremely large value of these two inverse prevalences by grouping treatment-dominated clusters (thereby small \( (1 - p_g(h))^{-1} \)) or control-dominated clusters together (thereby small \( p_g^{-1}(h) \)). Therefore, we might need a relatively narrower window of treatment prevalence, for example allowing particularly small \( \delta_h \) in one group than others when \( p_g(h) \) is almost near zero or one.
The other thing to note is that Λ (Equation (9)) is the estimated bias when the propensity score model does not adjust for \( U_h \) in any way. This will not be true if a random effects model is fit. If a random intercept or slope for each cluster is included in the model, for example \( \hat{\gamma}_{(\text{group-RE,}.)} \) instead of \( \hat{\gamma}_{(\text{group,}.)} \), is used, Λ would over-estimate the actual bias. We may instead consider \( U_h \) as the remaining unmeasured cluster-level characteristic after adjusting for random intercepts or slopes. Then the bias is smaller, but the cluster grouping strategy proposed here will still help reduce bias.

6 NUMERICAL EXPERIMENT

Through simulations, we aim to explore (i) whether selectively pooling clusters with respect to similar \( p_h \) can restrain the influence of unmeasured cluster-level characteristics more effectively than partial pooling by random selection or based on similar observed characteristics, and (ii) whether a combination of selectively pooled propensity scores and different types of IPW estimators performs better than those using full pooling in the presence of an unmeasured cluster-level confounder and/or effect modifier.

Simulation settings are as follows. Suppose that we have \( H = 200 \) clusters with cluster size of \( n_h \sim [U(5, 25)] \). We assume a relatively small cluster size considering our research question and given common data situations. We generate continuous observed covariates \( X_{hk} \) measured at the individual level and \( Y_{hk} \sim \mathcal{N}(0, 1) \) at the cluster level, and an unobserved cluster-level covariate \( U_h \sim U(-2, 2) \). Then the data generating models for treatment assignment and potential outcomes are as below:

\[
\begin{align*}
\text{logit}(e_{hk}^*) &= \alpha_{h,0} + \alpha_1 X_{hk} + \alpha_2 (X_{hk} - \bar{X}_h) + \alpha_3 V_h + \alpha_4 (U_h - \bar{U}); \\
Y_{hk}(z) &= \beta_{h,0} + \beta_1 X_{hk} + \beta_2 (X_{hk} - \bar{X}_h) + \beta_3 V_h + \beta_4 (U_h - \bar{U}) \\
&\quad + \bar{Z} \{ \kappa_{h,0} + \kappa_1 \bar{X}_h + \kappa_2 (X_{hk} - \bar{X}_h) + \kappa_3 V_h + \kappa_4 (U_h - \bar{U})^2 \} \\
&\quad + \mathcal{N}(0, \sigma_y).
\end{align*}
\]

We further adjust treatment assignment model by taking \( e_{hk} = 0.7e_{hk}^* + 0.15 \) to assure an adequate number of treated and control individuals within each cluster. In the real data analysis, clusters with all units assigned to one treatment arm were dropped to allow for easier comparison of the IPW estimators. A set of intercepts \( (\alpha_{h,0}, \beta_{h,0}, \kappa_{h,0}) \) represent cluster-level random effects; \( \bar{X}_h \) and \( (X_{hk} - \bar{X}_h) \) represent the associations of individual characteristics as a form of an aggregated characteristic within cluster \( (\bar{X}_h) \) and an individual’s relative difference within cluster \( (X_{hk} - \bar{X}_h) \) respectively. We allow for the square of \( (U_h - \bar{U}) \) as an effect moderator to examine the performance of each estimator under non-linear treatment effects with respect to \( U_h \). A parameter set \( (\kappa_{h,0}, \kappa_1, \kappa_2, \kappa_3, \kappa_4) \) controls the effect heterogeneity through cluster random effects and observed and unobserved covariates. Detailed settings about these parameters can be found in the supplementary material.

For numerical experiments, we vary the parameter values of \( (\alpha_4, \beta_4, \kappa_4) \), which represent the influence of an unmeasured covariate \( U_h \) on the treatment assignments \( (\alpha_4) \), outcome distributions \( (\beta_4) \), and treatment effects \( (\kappa_4) \), respectively. Non-zero \( \alpha_4 \) and \( \beta_4 \) implies the presence of confounding due to \( U_h \) other than effect modification, and non-zero \( \kappa_4 \) implies the presence of effect modification by \( U_h \). For propensity score estimation, we use random effects models (e.g. full-RE or group-RE) throughout the numerical experiments and application because they are commonly used to adjust for unmeasured heterogeneity (Arpino & Mealli, 2011; Thoemmes & West, 2011).
6.1 | Different choice of pooling methods

Figure 4 shows the average bias in the estimated ATE when different methods of cluster grouping are used given a fixed number of groups ($G = 10$). After grouping we apply a $\hat{\gamma}_{\text{group−RE, group}}$ estimator based on the partially pooled propensity scores estimated with observed covariates and random intercepts. Also, we illustrate the performance of a (full-RE, full) estimator (cyan lines), without any partial pooling. We observe that partially pooling cluster groups with similar treatment prevalence (red lines) results in the smallest average bias—even smaller than that using additional information of observed covariates $\bar{X}_h$ and $V_h$ (green lines); whereas random grouping and grouping with observed covariates do not add any benefits compared to fully pooled propensity scores.

6.2 | Different choice of IPW estimators and standard error estimation

Now only using the partial pooling with similar treatment prevalence, we compare our proposed IPW estimators—(group-RE, cluster) and (group-RE, group)—to a (full-RE, cluster) estimator. In Figure 5, we present the average of absolute bias in ATE estimates under model 10. When fully pooled propensity scores are used, a (full-RE, cluster) estimator reduces the influence from $U_h$ better compared to (full-RE, full); but it is still sensitive to effect modification due to $U_h$, that
is under non-zero $\kappa_4$. Overall, a cluster-weighted IPW analytically and theoretically has smaller bias than group-weighted IPW as it estimates $\tau_h$ while being free from unmeasured confounding. However, the cluster-weighted IPW becomes invalid to use when the cluster size is so small that some clusters have all individuals in the same treatment condition.

Standard errors assuming fixed propensity scores and coverage rates based on those are provided in the supplementary material Table S1. Table 2 above presents the simulation results under one of the scenarios when $(\alpha_4, \beta_4, \kappa_4) = (−2, −2, −2)$. Note that we could not always identify the ATE using the fixed effects or the cluster-weighted IPW estimator. Even though the fixed effects model substantially reduces bias compared to the random effects model, it produces large variability mainly due to the small cluster size. Moreover, in Table 2, it appears that a (group-RE, cluster) looks better than a (group-RE, group), but for about 10% out of $r = 1000$ replications, the (group-RE, cluster) estimator distorts the population as it discards the clusters of which $\tau_h$’s were not identified.

In the supplementary material, we provide additional simulations to examine whether those combinations perform better than the existing methods proposed for unmeasured context problems (He, 2018; Yang, 2018). The results demonstrate that the partial pooling method is most robust against interactions between the observed and unobserved covariates. We also compared the proposed methods to latent class approach (Kim & Steiner, 2015) which was not developed for the current purpose of handling unobserved cluster-level confounding, but also results in a grouping of clusters. With a continuous $U_h$, partial pooling by treatment prevalence $p_h$ performs better than partial pooling by the estimated latent class. In addition, we examine the performance

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**FIGURE 5** Average absolute bias in ATE over $r = 1000$ replicates when pooled propensity score model with random effects (full-RE, ·) or partially pooled propensity score model of cluster groups with random effects (group-RE, ·) is used; as a causal estimator, a group-weighted (·, group) and cluster-weighted (·, cluster) inverse probability weighting are applied following propensity score estimation. For comparison purposes, we also include (full-FE, full) which presents the results when propensity score models with fixed effects were used [Colour figure can be viewed at wileyonlinelibrary.com]
of our methods in the presence of a collider bias and a bias amplification, each of which is induced by conditioning on the observed covariates $X_{hk}$ and/or $V_h$. Lastly, additional simulations with random slopes of unmeasured $U_h$ can also be found in the supplementary material.

### 7 APPLICATION TO ECLS-K DATA

We apply our partially pooled propensity score method to estimate the causal effect of pre-school programmes on children’s math achievement in kindergarten. In this setting, one child’s preschool participation is not likely to affect the math achievement of other children in the same school; thus, we assume that SUTVA holds within and across clusters. We consider potential confounding due to child-level characteristics (sex, race, age, family type and motor skills) and school-level characteristics (census region (Northeast/Midwest/South/West) and location (Central City/large town/rural)) for the propensity score models. Detailed information is in Dong et al. (2020) and the supplementary material.

We focus on $H = 778$ schools with at least one student under treatment (centre-based programme) and under control (parental care) in the school to enable us to apply a cluster-weighted IPW. These schools were then categorized into $G = 10$ groups based on the treatment prevalence within school using the PAM method.

We have two observed cluster-level covariates—census region and location. Accordingly, we consider two scenarios to explore the role of partially pooled propensity scores:

1. (Model 1) Propensity score models include these two cluster-level covariates as well as the observed individual-level covariates.
2. (Model 2) Propensity score models do not include any cluster-level covariates but only include the observed individual-level covariates.

The motivation behind considering Model 2 is to compare different estimators with potential unobserved cluster-level covariates that we indeed observed. Of course, in both Models 1 and 2, there might also be other unobserved cluster-level characteristics, but we expect Model...
2 would suffer more from unobserved confounding under the absence of two cluster-level covariates. We also added a random effect to each propensity score model, considering its wide use in multilevel data settings. In Table 3, we provide the covariates’ balance table of the two cluster-level covariates before and after weighting, and weighting by fully and partially pooled propensity scores under Model 2. The results show that even in the absence of important cluster-level covariates in the propensity score model, using partially pooled propensity scores helps to reduce bias from those unmeasured covariates, resulting in reduced standard mean differences for census region and location that had not been included in the propensity score model. Details of the balance on all of the observed covariates are presented in the supplementary material.

We summarize the results under different schemes in Figure 6. First of all, it is evident that the causal estimate (4.36) without weighting (unweighted effect) might overestimate the causal effect of centre-based programmes over parental care given lack of adjustment for confounding, compared to any of the propensity score-weighted estimates. The following three black lines in Figure 6 show the results under Model 1 using different methods. We observed that when individuals were weighted by partially pooled propensity scores and causal effects were estimated through a group-weighted IPW, that is, using the (group-RE, group) estimator (Equation 9), the size of effect of centre-based programme (1.62) is substantially lower than that under the combination of pooled propensity scores and a marginal IPW (2.30). The result using a (group-RE, cluster) estimator (1.84) shows a slightly higher point estimate than that (1.62) using a (group-RE, group) estimation approach. The average and the standard deviation of observed math scores are 27.27 and 9.45, respectively. Therefore, the estimated effect size of $\hat{\tau} = 1.84$ represents an increase of about 0.2 standard deviations.

The results under Model 2 are presented in the last three blue lines. The results show that with partially pooled propensity score models, the estimates stay nearly constant even after missing census region and location, exhibiting slightly more robustness against missing two cluster-level covariates than (full-RE, full) estimator. In contrast, the fully pooled propensity scores still result in the estimates (2.36) closer to the unweighted effects than those under Model 1 (2.30).

8 | CONCLUSION

In this work, we discuss the use of a partially pooled propensity score estimation method to reduce bias in the causal estimate when unmeasured cluster-level characteristics influence treatment assignment and potential outcomes. We emphasize its usefulness when cluster sizes are small and the number of baseline characteristics is relatively large. We use simulation studies to examine the method’s performance and apply the partially pooled propensity score approach to estimate the effect of pre-school education programmes on children’s performance.

Throughout simulations and applications, we only considered clusters having at least one treated and at least one control individual in the sample. However, this is not a necessary condition to identify ATE ($\tau$) and the proposed method can allow clusters with treatment prevalence $p_h = 0$. Restricting the treatment prevalence is for our convenience to compare different types of causal estimators such as cluster-specific IPW (which requires $p_h > 0$) and group-specific IPW (which requires $p_g(h) > 0$). Still, it is important to note that restricting to clusters with $p_h > 0$ might distort the target population. For this reason, we can also benefit from the partial grouping of clusters by using the same group of clusters in the IPW estimator when a cluster-level IPW is not identified.
 TABLE 3  Covariate balance table for the two cluster-level covariates when observations are not weighted by propensity scores (unweighted PS), weighted by partially pooled PS and weighted by fully pooled PS. Both partially pooled propensity score model and fully pooled propensity score model omitted cluster-level variable of census region and location (Model 2) and included random effects

|                  | Unweighted PS | Partially pooled PS | Fully pooled PS |
|------------------|---------------|---------------------|-----------------|
|                  | Parental care | Centre-based | SMD | Parental care | Centre-based | SMD | Parental care | Centre-based | SMD |
| **Census region**|               |           |     |               |           |     |               |           |     |
| Northeast        | 473 (17.5)    | 1165 (19.9) | 0.211 | 1696.1 (19.9) | 1620.2 (18.9) | 0.026 | 1503.5 (19.1) | 1613.3 (19.4) | 0.080 |
| Midwest          | 549 (20.3)    | 1465 (25.0) | 0.190 | 2025.6 (23.7) | 2024.0 (23.6) | 0.041 | 1761.0 (22.4) | 2019.2 (24.3) | 0.124 |
| South            | 894 (33.1)    | 2034 (34.7) | 0.190 | 2852.1 (33.4) | 2894.1 (33.8) | 0.041 | 2632.4 (33.4) | 2855.5 (34.4) | 0.124 |
| West             | 784 (29.0)    | 1193 (20.4) | 0.190 | 1963.0 (23.0) | 2023.9 (23.6) | 0.041 | 1978.6 (25.1) | 1819.8 (21.9) | 0.124 |
| **Location**     |               |           |     |               |           |     |               |           |     |
| Central city     | 1110 (41.1)   | 2363 (40.3) | 0.190 | 3401.0 (39.8) | 3509.6 (41.0) | 0.041 | 3118.6 (39.6) | 3402.4 (41.0) | 0.124 |
| Large town       | 1000 (37.0)   | 2594 (44.3) | 0.190 | 3552.7 (41.6) | 3594.1 (42.0) | 0.041 | 3137.3 (39.8) | 3586.8 (43.2) | 0.124 |
| Small town       | 590 (21.9)    | 900 (15.4)  | 0.190 | 1583.1 (18.5) | 1458.5 (17.0) | 0.041 | 1619.6 (20.6) | 1318.5 (15.9) | 0.124 |
| Sum of weights   | 2700          | 5857       |     | 8536.83       | 8562.19     |     | 7875.49       | 8307.70     |     |
We also found room for improvement in the method used to group clusters as we briefly discussed in Section 5.4. First of all, we observed that bias in an IPW estimator depends on the group’s treatment prevalence \( p_h(h) \) as well as on the difference between the cluster’s treatment prevalence and the group’s treatment prevalence \( \delta_h \); therefore, it would be a great help to develop data-adaptive grouping methods that could possibly vary the maximum of \( \delta_h \) or vary the number of groups \( G \) depending on \( p_h(h) \). Second, we will thoroughly investigate the changes in bias and variability as the number of groups changes, instead of fixing the number of groups as done here, for example \( G = 10 \). Moreover, as we have seen in the simulation studies a noticeable improvement in bias by using a cluster-weighted or group-weighted IPW, instead of using a marginal IPW, different grouping strategies in the use of propensity scores as well as in the estimation can be further explored. Lastly, partially pooled propensity score models may aggravate the existing problem of a bias amplification by implicitly adjusting the variables that are strongly associated with the treatment (Pearl, 2011). In such a case, instead of propensity score models, one may consider fitting a prognostic score model with the observed covariates predictive of the outcome. Future research could investigate the use of partial pooling in prognostic score models.

Overall, this partial pooling method provides straightforward and effective tools to reduce bias in causal effect estimation due to unmeasured contextual factors, and has many potential avenues for further development and application.

ACKNOWLEDGEMENTS

This research was supported by the Institute of Education Sciences, U.S. Department of Education, through Grant R305D150001 awarded to Johns Hopkins University (PI’s: Stuart and Dong) and by Grant R01MH115487 from the National Institute of Mental Health (PI: Stuart). The opinions expressed are those of the authors and do not represent views of the Institute or the U.S. Department of Education. The authors are thankful to the members of the University of Pennsylvania Causal Inference Reading Group for helpful comments.
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**SUPPORTING INFORMATION**

Additional supporting information may be found online in the Supporting Information section.

**How to cite this article:** Lee Y, Nguyen TQ, Stuart EA. Partially pooled propensity score models for average treatment effect estimation with multilevel data. *J R Stat Soc Series A*. 2021;184:1578–1598. https://doi.org/10.1111/rssa.12741