Comparison of Selected Methods for Endogenous Subgroup/Mediation Analys

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	Structural Equation Modeling (SEM)	Instrumental Variables (IV), with T/C status as instrument	Continuous Version of Analysis of Symmetrically Predicted Endogenous Subgroups (ASPES)	Discrete Version of Analysis of Symmetrically Predicted Endogenous Subgroups (ASPES)
Research Question Addressed	What is the indirect effect of the treatment on the outcome, operating through the mediator? What proportion of the effect of the treatment on the outcome is realized through the mediator (i.e., what is the ratio of the indirect effect to the total effect)?	What is the effect of the mediator on outcomes?	What is the effect of the mediator on impacts?	What is the effect on mediator-defined subgroups?

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Estimation Process	Structural equation modeling extends traditional multivariate regression techniques to explore explicitly the effects of a mediator (e.g., the number of professional development sessions attended by a teacher), *M*, on an outcome (e.g., student achievement), *Y*, where there is also exposure to a treatment, *T*, that influences *Y* both directly and indirectly through *M*. SEM models *M* as a function of *T* and models *Y* as a function of *M* and *T*: $M = \alpha_0 + \alpha_1 T + \alpha_2 \boldsymbol{X} + \varepsilon_1$ $Y = \beta_0 + \beta_1 T + \beta_2 M + \beta_3 \boldsymbol{X} + \varepsilon_2$ In this system, *X* is a vector of covariates (e.g., school, teacher, and student characteristics) included to account for plausible rival explanations for the main relationship of interest. This model estimates the indirect effect of the professional development session attendance mediator and the direct effect of the treatment (that is, the effect of *T* on *Y* that is not mediated by professional development session attendance), controlling for covariates. SEM packages in statistical programs estimate these measures using simultaneous estimation techniques such as seemingly unrelated regression (SUR) or maximum likelihood techniques.	Instrumental variables analysis is most commonly implemented using two-stage least squares (2SLS). *Stage 1:* For entire sample, regression of mediator *M* on treatment, *T*, and baseline covariates: $M = \pi_0 + \pi_1 T + \pi_2 \boldsymbol{X} + \varepsilon_1$ *Stage 2:* Regression of outcome on the predicted mediator and baseline covariates: $Y = \beta_0 + \beta_1 \widehat{M} + \beta_2 \boldsymbol{X} + \varepsilon_2$	*Stage 1:* Use a cross-validation approach to predict mediator values for both the treatment and control groups: 1. Randomly partition your sample (both T and C) into 10 or more groups of equal size. 2. Obtain predictions for group 1 by: • Estimating the prediction model on treatment individuals in groups 2-10+ $M^A = \pi_0 + \pi_1 \boldsymbol{X} + \varepsilon_1$ • Predicting the mediator for both treatment and control individuals in group 1 $\widehat{M^P} = \widehat{\pi_0} + \widehat{\pi_1} \boldsymbol{X}$ 3. Obtain predictions for group 2 by: • Estimating the prediction model on treatment individuals in groups 1, 3-10+ $M^A = \pi_0 + \pi_1 \boldsymbol{X} + \varepsilon_1$ • Predicting the mediator for both treatment and control individuals in group 2 $\widehat{M^P} = \widehat{\pi_0} + \widehat{\pi_1} \boldsymbol{X}$ 4. Repeat for groups 3-10+ *Stage 2:* OLS regression of outcome on the predicted value of mediator and treatment: $Y = \beta_0 + \beta_1 \widehat{M^P} + \beta_2 T + \beta_3 T \widehat{M^P} + \varepsilon_2$	*Stage 1:* Use a cross-validation approach to predict mediator-defined subgroup membership *M* for both the treatment and control groups: 1. Randomly partition your sample (both T and C) into 10 or more groups of equal size. 2. Obtain predictions for group 1 by: • Estimating the prediction model on treatment individuals in groups 2-10+ $M^A = \pi_0 + \pi_1 \boldsymbol{X} + \varepsilon_1$ • Predicting subgroup membership for both treatment and control individuals in group 1 $\widehat{M^P} = \widehat{\pi_0} + \widehat{\pi_1} \boldsymbol{X}$ 3. Obtain predictions for group 2 by: • Estimating the prediction model on treatment individuals in groups 1, 3-10+ $M^A = \pi_0 + \pi_1 \boldsymbol{X} + \varepsilon_1$ • Predicting subgroup membership for both treatment and control individuals in group 2 $\widehat{M^P} = \widehat{\pi_0} + \widehat{\pi_1} \boldsymbol{X}$ 4. Repeat for groups 3-10+ This process provides each individual in the sample (both treatment and control) with a continuous score, $\widehat{M^P}$, that represents their probability of being in the endogenous subgroup based on their baseline characteristics. $\widehat{M^P}$ is then converted into a binary indicator that divides the sample into two predicted endogenous subgroups. In what follows, consider two subgroups denoted Subgroup H (for High Dosage) and Subgroup L (for Low Dosage). *Stage 2:* OLS regressions of outcome on treatment and baseline covariates run separately for predicted groups (we illustrate with a split-sample approach, but a single model with relevant interaction terms is also an option): $Y = \beta_0^H + \beta_1^H T + \beta_2^H \boldsymbol{X} + \varepsilon_2^H$ for the predicted high dosage subgroup, as indicted by superscript- H; and $Y = \beta_0^L + \beta_1^L T + \beta_2^L \boldsymbol{X} + \varepsilon_2^L$ for the predicted low dosage subgroup, as indicated by superscript L. *Stage 3:* Convert impacts on predicted subgroups to impacts on actual subgroups $\hat{H} = \frac{\widehat{\beta_1^H} \widehat{w_L} - (1 - \widehat{w_H}) \widehat{\beta_1^L}}{\widehat{w_H} + \widehat{w_L} - 1}$ $\hat{L} = \frac{\widehat{\beta_1^L} \widehat{w_H} - (1 - \widehat{w_L}) \widehat{\beta_1^H}}{\widehat{w_H} + \widehat{w_L} - 1}$ Where $\hat{H}$ is the estimated impact on the actual high dosage subgroup; $\hat{L}$ is the estimated impact on the actual low dosage subgroup; w_H is the proportion of individuals predicted to be high-dosage that are actually high dosage; and w_L is the proportion of individuals predicted to be low-dosage that are actually low dosage.

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Key Assumptions	Conditional on *X*, *M* is not correlated with ε_2. Another equivalent statement of this assumption is ε_1 is not correlated with ε_2. This method will yield biased estimates of the relationship between the mediator and outcomes if there are unmeasured confounding variables (e.g., teacher ability or motivation) that simultaneously influence the mediator and the outcome. This is true even in an experiment: while T is uncorrelated with ε_1, *M* may be correlated with ε_2, which would yield biased estimates of the mediational effects.	Treatment only affects the outcome through the mediator (i.e., there is no direct effect of treatment on the outcome and no indirect effect through any other mediator).	The baseline characteristics that predict the mediator do not have a direct or indirect effect on impacts apart from their indirect effect on impacts through the mediator.	Baseline predictors of mediator-defined subgroups only affect impact magnitude through the mediator (i.e., there is no direct effect of baseline predictors on impact magnitude).
Interpretation	• Estimated indirect effect: $\widehat{\alpha_1}\widehat{\beta_2}$ • Estimated direct effect: $\widehat{\beta_1}$	• β_1 is the marginal effect of the mediator on the outcome • π_1 is the effect of treatment status on the mediator • $\beta_1\pi_1$ is the indirect effect of the treatment on the outcome, operating through the mediator • Typically, only the marginal effect is reported • By assumption, the total effect of the treatment is equal to the indirect effect • These are causal effects for those with actual experience, by assumption	• β_1 is the marginal effect of the predicted mediator on the outcome for individuals in the control group • β_3 is the effect of the mediator on impacts • $\beta_2 + \beta_3\widehat{M^P}$ is the total effect of treatment on the impacts • Causal treatment effect for those with actual experience, by assumption	• β_1^H is the effect of treatment on individuals predicted to have high dosage • β_1^L is the effect of treatment on individuals predicted to have low dosage • H is the effect of treatment on individuals who experience high dosage • L is the effect of treatment on individuals who experience high dosage • These are causal treatment effects for those in predicted subgroups • These are causal treatment effects for those in actual subgroup, by assumption
Key References	Baron & Kenny (1986); Imai, Keele, and Tingley (2010); Imai, Keele, Tingley, and Yamamoto (2011)	Angrist, Imbens, and Rubin (1996)	Peck (2003); Harvill, Peck & Bell (2013); Moulton, Harvill, Peck, and Lin (Working paper)	Peck (2003, 2013); Harvill, Peck & Bell (2013); Bell & Peck (2013); Moulton, Peck, & Bell (2014)