Unpacking the Black Box of Causality: Learning about Causal Mechanisms from Experimental and Observational Studies

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Joint work with
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Causal inference is a central goal of scientific research

Scientists care about causal mechanisms, not just about causal effects \(\rightsquigarrow\) external validity

Policy makers want to devise better policies

Randomized experiments often only determine whether the treatment causes changes in the outcome

Not how and why the treatment affects the outcome

Common criticism of experiments and statistics:

- \textbf{black box} view of causality

Qualitative research \(\rightsquigarrow\) process tracing

Question: How can we learn about causal mechanisms from experimental and observational studies?
Overview of the Talk

Present a general framework for statistical analysis and research design strategies to understand causal mechanisms

1. Show that the sequential ignorability assumption is required to identify mechanisms even in experiments
2. Offer a flexible estimation strategy under this assumption
3. Introduce a sensitivity analysis to probe this assumption
4. Illustrate how to use statistical software mediation
5. Consider research designs that relax sequential ignorability
6. Multiple mechanisms
7. Causal mediation Q&A
Causal Mediation Analysis

- Graphical representation

\[ \text{Mediator, } M \]

\[ \text{Treatment, } T \quad \rightarrow \quad \text{Outcome, } Y \]

- Goal is to decompose total effect into direct and indirect effects
- Alternative approach: decompose the treatment into different components
- Causal mediation analysis as \textit{quantitative process tracing}
- How large is the mediation effect relative to the total effect?
Mexican Universal Health Insurance Program

- Seguro Popular (2003): cover all 50M uninsured Mexicans
- Matched-pair cluster randomized design

Treatment $T$:
- building hospitals and clinics
- encouragement to sign up for SP

Post-treatment measures:
- financial protection
- healthcare utilization
- health

Mediation analysis:
- $M$: reduction in catastrophic expenditure
- $Y$: health outcome
Decomposition of Incumbency Advantage

- Incumbency effects: one of the most studied topics
- Consensus emerged in 1980s: incumbency advantage is positive and growing in magnitude

- New direction in 1990s: Where does incumbency advantage come from?
- **Scare-off/quality effect**: the ability of incumbents to deter high-quality challengers from entering the race
- Alternative causal mechanisms: name recognition, campaign spending, personal vote, television, etc.

- Mediation analysis:
  - $T$: incumbency status
  - $M$: quality of challenger
  - $Y$: election outcome
The Standard Estimation Method

- Linear models for mediator and outcome:
  \[ Y_i = \alpha_1 + \beta_1 T_i + \xi_1^T X_i + \epsilon_{1i} \]
  \[ M_i = \alpha_2 + \beta_2 T_i + \xi_2^T X_i + \epsilon_{2i} \]
  \[ Y_i = \alpha_3 + \beta_3 T_i + \gamma M_i + \xi_3^T X_i + \epsilon_{3i} \]

where \( X_i \) is a set of pre-treatment or control variables

1. Total effect (ATE) is \( \beta_1 \)
2. Direct effect is \( \beta_3 \)
3. Indirect or mediation effect is \( \beta_2 \gamma \)
4. Effect decomposition: \( \beta_1 = \beta_3 + \beta_2 \gamma \).

- Some motivating questions:
  1. What should we do when we have interaction or nonlinear terms?
  2. What about other models such as logit?
  3. In general, under what conditions can we interpret \( \beta_1 \) and \( \beta_2 \gamma \) as causal effects?
  4. What do we really mean by causal mediation effect anyway?
Potential Outcomes Framework of Causal Inference

- Observed data:
  - Binary treatment: \( T_i \in \{0, 1\} \)
  - Mediator: \( M_i \in M \)
  - Outcome: \( Y_i \in Y \)
  - Observed pre-treatment covariates: \( X_i \in X \)

- Potential outcomes model (Neyman, Rubin):
  - Potential mediators: \( M_i(t) \) where \( M_i = M_i(T_i) \)
  - Potential outcomes: \( Y_i(t, m) \) where \( Y_i = Y_i(T_i, M_i(T_i)) \)

- Total causal effect:

\[
\tau_i \equiv Y_i(1, M_i(1)) - Y_i(0, M_i(0))
\]

- **Fundamental problem of causal inference**: only one potential outcome can be observed for each \( i \)
Back to the Examples

- $M_i(1)$:
  1. Level of catastrophic health expenditure for an individual $i$
  2. Quality of challenger if politician $i$ is an incumbent

- $Y_i(1, M_i(1))$:
  1. Health outcome that would result if individual $i$ pays catastrophic health expenditure $M_i(1)$
  2. Election outcome that would result if politician $i$ is an incumbent and faces a challenger whose quality is $M_i(1)$

- $M_i(0)$ and $Y_i(0, M_i(0))$ are the converse
Causal Mediation Effects

- Causal mediation (Indirect) effects:
  \[ \delta_i(t) \equiv Y_i(t, M_i(1)) - Y_i(t, M_i(0)) \]

- Causal effect of the change in \( M_i \) on \( Y_i \) that would be induced by treatment
- Change the mediator from \( M_i(0) \) to \( M_i(1) \) while holding the treatment constant at \( t \)
- Represents the mechanism through \( M_i \)
- Zero treatment effect on mediator \( \implies \) Zero mediation effect

Examples:
1. Part of health effects that are due to the reduction in the level of catastrophic expenditure
2. Part of incumbency advantage that is due to the difference in challenger quality induced by incumbency status
Total Effect \(=\) Indirect Effect + Direct Effect

- **Direct effects:**
  
  \[
  \zeta_i(t) \equiv Y_i(1, M_i(t)) - Y_i(0, M_i(t))
  \]

- **Causal effect of** \(T_i\) **on** \(Y_i\), **holding mediator constant at its potential value that would realize when** \(T_i = t\)

- **Change the treatment from 0 to 1 while holding the mediator constant at** \(M_i(t)\)

- **Represents all mechanisms other than through** \(M_i\)

- **Total effect = mediation (indirect) effect + direct effect:**

  \[
  \tau_i = \delta_i(t) + \zeta_i(1 - t) = \frac{1}{2}\{(\delta_i(0) + \zeta_i(0)) + (\delta_i(1) + \zeta_i(1))\} 
  \]
Mechanisms

- **Indirect effects**: $\delta_i(t) \equiv Y_i(t, M_i(1)) - Y_i(t, M_i(0))$
- Counterfactuals about treatment-induced mediator values

Manipulations

- **Controlled direct effects**: $\xi_i(t, m, m') \equiv Y_i(t, m) - Y_i(t, m')$
- Causal effect of directly manipulating the mediator under $T_i = t$

Interactions

- **Interaction effects**: $\xi(1, m, m') - \xi(0, m, m')$
- The extent to which controlled direct effects vary by the treatment
What Does the Observed Data Tell Us?

- Recall the standard experimental design:
  1. randomize $T_i$
  2. measure $M_i$ and then $Y_i$

- Among observations with $T_i = t$, we observe $Y_i(t, M_i(t))$ but not $Y_i(t, M_i(1 - t))$ unless $M_i(t) = M_i(1 - t)$

- But we want to estimate

\[ \delta_i(t) \equiv Y_i(t, M_i(1)) - Y_i(t, M_i(0)) \]

- For $t = 1$, we observe $Y_i(1, M_i(1))$ but not $Y_i(1, M_i(0))$

- Similarly, for $t = 0$, we observe $Y_i(0, M_i(0))$ but not $Y_i(0, M_i(1))$

- We have the identification problem $\Rightarrow$ Need assumptions or better research designs
Counterfactuals in the Examples

1. **Health insurance evaluation:**
   - An individual lives in the treatment community \((T_i = 1)\)
   - For this person, \(Y_i(1, M_i(1))\) is the observed health outcome
   - \(Y_i(1, M_i(0))\) is his health outcome in the counterfactual world where he still lives in the treatment village but his catastrophic expenditure is at the same level as it would be if his village did not receive the treatment

2. **Incumbency advantage:**
   - An incumbent \((T_i = 1)\) faces a challenger with quality \(M_i(1)\)
   - We observe the electoral outcome \(Y_i = Y_i(1, M_i(1))\)
   - We also want \(Y_i(1, M_i(0))\) where \(M_i(0)\) is the quality of challenger this incumbent politician would face if she is not an incumbent

In both cases, we can’t observe \(Y_i(1, M_i(0))\) because \(M_i(0)\) is not realized when \(T_i = 1\)
Sequential Ignorability Assumption

- Proposed identification assumption: **Sequential Ignorability** (SI)

\[
\{ Y_i(t', m), M_i(t) \} \perp T_i \mid X_i = x, \quad (1)
\]

\[
Y_i(t', m) \perp M_i(t) \mid T_i = t, X_i = x \quad (2)
\]

- In words,
  1. \( T_i \) is (as-if) randomized conditional on \( X_i = x \)
  2. \( M_i(t) \) is (as-if) randomized conditional on \( X_i = x \) and \( T_i = t \)

- Important limitations:
  1. In a standard experiment, (1) holds but (2) may not
  2. \( X_i \) needs to include all confounders
  3. \( X_i \) must be pre-treatment confounders \( \implies \) post-treatment confounder is not allowed
  4. Randomizing \( M_i \) via manipulation is not the same as assuming \( M_i(t) \) is as-if randomized
Back to Seguro Popular:

- Treatment is randomized $\iff (1)$ is satisfied
- But $(2)$ may not hold:
  1. Pre-treatment confounder or $X_i$: health predisposition
     people with poor health are more likely to pay catastrophic health expenditure and have poor health in the future
  2. Post-treatment confounder: alternative mechanism
     Seguro Popular increases the use of preventive care, which in turn reduces catastrophic expenditure and improves future health outcome

- Pre-treatment confounders $\iff$ measure and adjust for them
- Post-treatment confounders $\iff$ adjusting is not sufficient
Nonparametric Identification

Under SI, both ACME and average direct effects are **nonparametrically identified** (can be consistently estimated without modeling assumption)

- **ACME** $\bar{\delta}(t)$
  \[
  \int \int \mathbb{E}(Y_i \mid M_i, T_i = t, X_i) \{dP(M_i \mid T_i = 1, X_i) - dP(M_i \mid T_i = 0, X_i)\} \ dP(X_i)
  \]

- **Average direct effects** $\bar{\zeta}(t)$
  \[
  \int \int \{\mathbb{E}(Y_i \mid M_i, T_i = 1, X_i) - \mathbb{E}(Y_i \mid M_i, T_i = 0, X_i)\} \ dP(M_i \mid T_i = t, X_i) \ dP(X_i)
  \]

Implies the general mediation formula under any statistical model
Traditional Estimation Methods: LSEM

- **Linear structural equation model (LSEM):**
  \[
  M_i = \alpha_2 + \beta_2 T_i + \xi_2^T X_i + \epsilon_{i2},
  \]
  \[
  Y_i = \alpha_3 + \beta_3 T_i + \gamma M_i + \xi_3^T X_i + \epsilon_{i3}.
  \]

- Fit two least squares regressions separately
- Use **product of coefficients** ($\hat{\beta}_2 \hat{\gamma}$) to estimate ACME
- Use asymptotic variance to test significance (Sobel test)

- Under SI and the **no-interaction assumption** ($\bar{\delta}(1) \neq \bar{\delta}(0)$), $\hat{\beta}_2 \hat{\gamma}$ consistently estimates ACME
- Can be extended to LSEM with interaction terms
- Problem: Only valid for the simplest LSEM
**Popular Baron-Kenny Procedure**

- **The procedure:**
  1. Regress $Y$ on $T$ and show a significant relationship
  2. Regress $M$ on $T$ and show a significant relationship
  3. Regress $Y$ on $M$ and $T$, and show a significant relationship between $Y$ and $M$

- **The problems:**
  1. First step can lead to false negatives especially if indirect and direct effects in opposite directions
  2. The procedure only anticipates simplest linear models
  3. Don’t do star-gazing. Report quantities of interest
1. Model outcome and mediator
   - Outcome model: \( p(Y_i \mid T_i, M_i, X_i) \)
   - Mediator model: \( p(M_i \mid T_i, X_i) \)
   - These models can be of any form (linear or nonlinear, semi- or nonparametric, with or without interactions)

2. Predict mediator for both treatment values \((M_i(1), M_i(0))\)

3. Predict outcome by first setting \(T_i = 1\) and \(M_i = M_i(0)\), and then \(T_i = 1\) and \(M_i = M_i(1)\)

4. Compute the average difference between two outcomes to obtain a consistent estimate of ACME

5. Monte-Carlo or bootstrap to estimate uncertainty
Two logistic regression models:

\[
\begin{align*}
\Pr(M_i = 1 \mid T_i, X_i) &= \text{logit}^{-1}(\alpha_2 + \beta_2 T_i + \xi_2^\top X_i) \\
\Pr(Y_i = 1 \mid T_i, M_i, X_i) &= \text{logit}^{-1}(\alpha_3 + \beta_3 T_i + \gamma M_i + \xi_3^\top X_i)
\end{align*}
\]

- Can’t multiply \(\beta_2\) by \(\gamma\)
- Difference of coefficients \(\beta_1 - \beta_3\) doesn’t work either

\[
\Pr(Y_i = 1 \mid T_i, X_i) = \text{logit}^{-1}(\alpha_1 + \beta_1 T_i + \xi_1^\top X_i)
\]

- Can use our algorithm (example: \(\mathbb{E}\{Y_i(1, M_i(0))\}\))
  1. Predict \(M_i(0)\) given \(T_i = 0\) using the first model
  2. Compute \(\Pr(Y_i(1, M_i(0)) = 1 \mid T_i = 1, M_i = \hat{M}_i(0), X_i)\) using the second model
Sensitivity Analysis

- Standard experiments require sequential ignorability to identify mechanisms
- The sequential ignorability assumption is often too strong
- Need to assess the robustness of findings via sensitivity analysis
- **Question**: How large a departure from the key assumption must occur for the conclusions to no longer hold?
- Parametric sensitivity analysis by assuming

\[
\{ Y_i(t', m), M_i(t) \} \perp \perp T_i \mid X_i = x
\]

but not

\[
Y_i(t', m) \perp \perp M_i(t) \mid T_i = t, X_i = x
\]

- Possible existence of unobserved *pre-treatment* confounder
Parametric Sensitivity Analysis

- **Sensitivity parameter**: \( \rho \equiv \text{Corr}(\epsilon_{i2}, \epsilon_{i3}) \)
- Sequential ignorability implies \( \rho = 0 \)
- Set \( \rho \) to different values and see how ACME changes

- **Result:**

\[
\bar{\delta}(0) = \bar{\delta}(1) = \frac{\beta_2 \sigma_1}{\sigma_2} \left\{ \tilde{\rho} - \rho \sqrt{\frac{1 - \tilde{\rho}^2}{1 - \rho^2}} \right\},
\]

where \( \sigma_j^2 \equiv \text{var}(\epsilon_{ij}) \) for \( j = 1, 2 \) and \( \tilde{\rho} \equiv \text{Corr}(\epsilon_{i1}, \epsilon_{i2}) \).

- When do my results go away completely?
- \( \bar{\delta}(t) = 0 \) if and only if \( \rho = \tilde{\rho} \)
- Easy to estimate from the regression of \( Y_i \) on \( T_i \):

\[
Y_i = \alpha_1 + \beta_1 T_i + \epsilon_{i1}
\]
Interpreting Sensitivity Analysis with R squares

- Interpreting $\rho$: how small is too small?

- An unobserved (pre-treatment) confounder formulation:

\[ \epsilon_{i2} = \lambda_2 U_i + \epsilon'_{i2} \quad \text{and} \quad \epsilon_{i3} = \lambda_3 U_i + \epsilon'_{i3} \]

- How much does $U_i$ have to explain for our results to go away?

- Sensitivity parameters: R squares
  1. Proportion of previously unexplained variance explained by $U_i$

\[ R^2_M \equiv 1 - \frac{\text{var}(\epsilon'_{i2})}{\text{var}(\epsilon_{i2})} \quad \text{and} \quad R^2_Y \equiv 1 - \frac{\text{var}(\epsilon'_{i3})}{\text{var}(\epsilon_{i3})} \]

  2. Proportion of original variance explained by $U_i$

\[ \tilde{R}^2_M \equiv \frac{\text{var}(\epsilon_{i2}) - \text{var}(\epsilon'_{i2})}{\text{var}(M_i)} \quad \text{and} \quad \tilde{R}^2_Y \equiv \frac{\text{var}(\epsilon_{i3}) - \text{var}(\epsilon'_{i3})}{\text{var}(Y_i)} \]
Then reparameterize $\rho$ using $(R_{2*}^M, R_{2*}^Y)$ (or $(\tilde{R}_M^2, \tilde{R}_Y^2)$):

$$\rho = \text{sgn}(\lambda_2 \lambda_3) R^*_M R^*_Y = \frac{\text{sgn}(\lambda_2 \lambda_3) \tilde{R}_M \tilde{R}_Y}{\sqrt{(1 - R^2_M)(1 - R^2_Y)}},$$

where $R^2_M$ and $R^2_Y$ are from the original mediator and outcome models.

- $\text{sgn}(\lambda_2 \lambda_3)$ indicates the direction of the effects of $U_i$ on $Y_i$ and $M_i$.

- Set $(R_{2*}^M, R_{2*}^Y)$ (or $(\tilde{R}_M^2, \tilde{R}_Y^2)$) to different values and see how mediation effects change.
Reanalysis: Estimates under Sequential Ignorability

- Original method: **Product of coefficients with the Sobel test**
  — Valid only when both models are linear w/o $T–M$ interaction (which they are not)
- Our method: Calculate ACME using our general algorithm

<table>
<thead>
<tr>
<th>Outcome variables</th>
<th>Product of Coefficients</th>
<th>Average Causal Mediation Effect ($\delta$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Decrease Immigration</td>
<td>.347</td>
<td>.105</td>
</tr>
<tr>
<td>$\tilde{\delta}(1)$</td>
<td>[0.146, 0.548]</td>
<td>[0.048, 0.170]</td>
</tr>
<tr>
<td>Support English Only Laws</td>
<td>.204</td>
<td>.074</td>
</tr>
<tr>
<td>$\tilde{\delta}(1)$</td>
<td>[0.069, 0.339]</td>
<td>[0.027, 0.132]</td>
</tr>
<tr>
<td>Request Anti-Immigration Information</td>
<td>.277</td>
<td>.029</td>
</tr>
<tr>
<td>$\tilde{\delta}(1)$</td>
<td>[0.084, 0.469]</td>
<td>[0.007, 0.063]</td>
</tr>
<tr>
<td>Send Anti-Immigration Message</td>
<td>.276</td>
<td>.086</td>
</tr>
<tr>
<td>$\tilde{\delta}(1)$</td>
<td>[0.102, 0.450]</td>
<td>[0.035, 0.144]</td>
</tr>
</tbody>
</table>
ACME > 0 as long as the error correlation is less than 0.39 (0.30 with 95% CI)
Reanalysis: Sensitivity Analysis w.r.t. $\tilde{R}^2_M$ and $\tilde{R}^2_Y$

An unobserved confounder can account for up to 26.5% of the variation in both $Y_i$ and $M_i$ before ACME becomes zero.
Open-Source Software “Mediation”

Model-Based Inference

Mediator Model
\[ f(M \mid T, X) \]
\[ \text{model.m} \]

Outcome Model
\[ f(Y \mid T, M, X) \]
\[ \text{model.y} \]

Causal Mediation Analysis
\[ \text{m.out} \leftarrow \text{mediate(model.m, model.y, ...)} \]

Sensitivity Analysis
\[ \text{s.out} \leftarrow \text{medsens(m.out, ...)} \]

Design-Based Inference

Single Experiment Design
\[ \text{d.out} \leftarrow \text{mediate.sed(outcome, mediator, treat, ...)} \]

Parallel Design
\[ \text{d.out} \leftarrow \text{mediate.pd(outcome, mediator, treat, ...)} \]

Parallel Encouragement Design
\[ \text{d.out} \leftarrow \text{mediate.ped(outcome, mediator, treat, encourage, ...)} \]

Crossover Encouragement Design
\[ \text{d.out} \leftarrow \text{mediate.ced(outcome, mediator, treat, encourage, ...)} \]
Implementation Examples

1. Fit models for the mediator and outcome variable and store these models

   > m <- lm(Mediator ~ Treat + X)
   > y <- lm(Y ~ Treat + Mediator + X)

2. **Mediation analysis**: Feed model objects into the `mediate()` function. Call a summary of results

   > m.out <- mediate(m, y, treat = "Treat",
                      mediator = "Mediator")
   > summary(m.out)

3. **Sensitivity analysis**: Feed the output into the `medsens()` function. Summarize and plot

   > s.out <- medsens(m.out)
   > summary(s.out)
   > plot(s.out, "rho")
   > plot(s.out, "R2")
## Data Types Available via mediation

<table>
<thead>
<tr>
<th>Mediator Model Types</th>
<th>Linear</th>
<th>GLM</th>
<th>Ordered</th>
<th>Censored</th>
<th>Quantile</th>
<th>GAM</th>
<th>Survival</th>
</tr>
</thead>
<tbody>
<tr>
<td>Linear (lm/lmer)</td>
<td>✓</td>
<td>✓</td>
<td>✓*</td>
<td>✓</td>
<td>✓</td>
<td>✓*</td>
<td>✓</td>
</tr>
<tr>
<td>GLM (glm/bayesglm/glmer)</td>
<td>✓</td>
<td>✓</td>
<td>✓*</td>
<td>✓</td>
<td>✓</td>
<td>✓*</td>
<td>✓</td>
</tr>
<tr>
<td>Ordered (polr/bayespolr)</td>
<td>✓</td>
<td>✓</td>
<td>✓*</td>
<td>✓</td>
<td>✓</td>
<td>✓*</td>
<td>✓</td>
</tr>
<tr>
<td>Censored (tobit via vglm)</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Quantile (rq)</td>
<td>✓*</td>
<td>✓*</td>
<td>✓*</td>
<td>✓*</td>
<td>✓*</td>
<td>✓*</td>
<td>✓*</td>
</tr>
<tr>
<td>GAM (gam)</td>
<td>✓*</td>
<td>✓*</td>
<td>✓*</td>
<td>✓*</td>
<td>✓*</td>
<td>✓*</td>
<td>✓*</td>
</tr>
<tr>
<td>Survival (survreg)</td>
<td>✓</td>
<td>✓</td>
<td>✓*</td>
<td>✓</td>
<td>✓</td>
<td>✓*</td>
<td>✓</td>
</tr>
</tbody>
</table>

Types of Models That Can be Handled by `mediate`. Stars (*) indicate the model combinations that can only be estimated using the nonparametric bootstrap (i.e. with `boot = TRUE`).
Additional Features

- Treatment/mediator interactions, with formal statistical tests
- Treatment/mediator/pre-treatment interactions and reporting of quantities by pre-treatment values
- Factoral, continuous treatment variables
- Cluster standard errors/adjustable CI reporting/p-values
- Support for multiple imputation
- Multiple mediators
- Multilevel mediation

Please read our vignette file [here](#).
Based on the same algorithm


`ssc install mediation`

More limited coverage of models (just bc. of time though!)
medeff (equation 1) (equation 2) [if] [in] [[weight]] , [sims(integer) seed(integer) vce(vcetype) Level(#) interact(varname)] mediate(varname) treat(varname)

Where “equation 1” or “equation 2” are of the form (For equation 1, the mediator equation):

probit M T x

or

regress M T x
Beyond Sequential Ignorability

- Without sequential ignorability, standard experimental design lacks identification power
- Even the sign of ACME is not identified

- Need to develop alternative experimental designs for more credible inference
- Possible when the mediator can be directly or indirectly manipulated
- All proposed designs preserve the ability to estimate the ACME under the SI assumption
- Trade-off: statistical power

- These experimental designs can then be extended to natural experiments in observational studies
Parallel Design

- **Experiment 1**
  1) Randomize treatment
  2) Measure mediator
  3) Measure outcome

- **Experiment 2**
  1) Randomize treatment
  2) Randomize mediator
  3) Measure outcome

- Must assume no direct effect of manipulation on outcome
- More informative than standard single experiment
- If we assume no \( T-M \) interaction, ACME is point identified
Why Do We Need No-Interaction Assumption?

- **Numerical Example:**

<table>
<thead>
<tr>
<th>Prop.</th>
<th>$M_i(1)$</th>
<th>$M_i(0)$</th>
<th>$Y_i(t, 1)$</th>
<th>$Y_i(t, 0)$</th>
<th>$\delta_i(t)$</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.3</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>-1</td>
</tr>
<tr>
<td>0.3</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>0.1</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>0.3</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

- $\mathbb{E}(M_i(1) - M_i(0)) = \mathbb{E}(Y_i(t, 1) - Y_i(t, 0)) = 0.2$, but $\bar{\delta}(t) = -0.2$

- **The Problem: Causal effect heterogeneity**
  - $T$ increases $M$ only *on average*
  - $M$ increases $Y$ only *on average*
  - $T - M$ interaction: Many of those who have a positive effect of $T$ on $M$ have a negative effect of $M$ on $Y$ (first row)

- **A solution:** sensitivity analysis (see Imai and Yamamoto, 2013)

- **Pitfall of “mechanism experiments” or “causal chain approach”**
Why study brain?: Social scientists’ search for causal mechanisms underlying human behavior

- Psychologists, economists, and even political scientists

Question: What mechanism links low offers in an ultimatum game with “irrational” rejections?

- A brain region known to be related to fairness becomes more active when unfair offer received (single experiment design)

Design solution: manipulate mechanisms with TMS

- Knoch et al. use TMS to manipulate — turn off — one of these regions, and then observes choices (parallel design)
Encouragement Design

- Direct manipulation of mediator is difficult in most situations
- Use an instrumental variable approach:

\[ Z \rightarrow M \]

\[ T \rightarrow Y \]

- Advantage: allows for unobserved confounder between \( M \) and \( Y \)
- Key Assumptions:
  1. \( Z \) is randomized or as-if random
  2. No direct effect of \( Z \) on \( Y \) (a.k.a. exclusion restriction)
Example: Social Norm Experiment on Property Taxes

- Lucia Del Carpio. “Are Neighbors Cheating?”
- Treatment: informing average rate of compliance
- Outcome: compliance rate obtained from administrative records
- Large positive effect on compliance rate $\approx 20$ percentage points
- Mediators:
  1. social norm (not measured; direct effect)
  2. $M_1$: beliefs about compliance (measured)
  3. $M_2$: beliefs about enforcement (measured)
- Instruments:
  1. $Z_1$: informing average rate of enforcement
  2. $Z_2$: payment-reminder
- Assumptions:
  1. $Z_1$ affects $Y$ only through $M_1$ and $M_2$
  2. $Z_2$ affects $Y$ only through $M_1$
- Results:
  - Average direct effect is estimated to be large
  - The author interprets this effect as the effect of social norm
Recall ACME can be identified if we observe $Y_i(t', M_i(t))$

Get $M_i(t)$, then switch $T_i$ to $t'$ while holding $M_i = M_i(t)$

**Crossover design:**

1. Round 1: Conduct a standard experiment
2. Round 2: Change the treatment to the opposite status but fix the mediator to the value observed in the first round

Very powerful – identifies mediation effects for each subject

Must assume **no carryover effect**: Round 1 must not affect Round 2

Can be made plausible by design
Example: Labor Market Discrimination

**BERTRAND & MULLAINATHAN (2004, AER)**

- **Treatment:** Black vs. White names on CVs
- **Mediator:** Perceived qualifications of applicants
- **Outcome:** Callback from employers

**Quantity of interest:** Direct effects of (perceived) race

Would Jamal get a callback if his name were Greg but his qualifications stayed the same?

**Round 1:** Send Jamal’s actual CV and record the outcome
**Round 2:** Send his CV as Greg and record the outcome

**Assumption:** Their different names do not change the perceived qualifications of applicants

Under this assumption, the direct effect can be interpreted as blunt racial discrimination
Designing Observational Studies

- Key difference between experimental and observational studies: treatment assignment
- Sequential ignorability:
  1. Ignorability of treatment given covariates
  2. Ignorability of mediator given treatment and covariates
- Both (1) and (2) are suspect in observational studies
- Statistical control: matching, propensity scores, etc.
- Search for quasi-randomized treatments: “natural” experiments
- How can we design observational studies?
- Experiments can serve as templates for observational studies
EXAMPLE Back to incumbency advantage

- Use of cross-over design (Levitt and Wolfram)
  1. 1st Round: two non-incumbents in an open seat
  2. 2nd Round: same candidates with one being an incumbent

- Assume challenger quality (mediator) stays the same
- Estimation of direct effect is possible

- Redistricting as natural experiments (Ansolabehere et al.)
  1. 1st Round: incumbent in the old part of the district
  2. 2nd Round: incumbent in the new part of the district

- Challenger quality is the same but treatment is different
- Estimation of direct effect is possible
Multiple Mediators

Quantity of interest = The average indirect effect with respect to \( M \)

\( W \) represents the alternative observed mediators

Left: Assumes independence between the two mechanisms

Right: Allows \( M \) to be affected by the other mediators \( W \)

Applied work often assumes the independence of mechanisms

Under this independence assumption, one can apply the same analysis as in the single mediator case

For causally dependent mediators, we must deal with the heterogeneity in the \( T \times M \) interaction as done under the parallel design \( \rightarrow \) sensitivity analysis
Applied social scientists often use the following model:

\[ M_i = \alpha_M + \beta_M T_i + \xi_M^T X_i + \epsilon_{iM} \]
\[ W_i = \alpha_W + \beta_W T_i + \xi_W^T X_i + \epsilon_{iW} \]
\[ Y_i = \alpha_3 + \beta_3 T_i + \gamma M_i + \theta^T W_i + \xi_3^T X_i + \epsilon_{i3} \]

The mediation effects are then estimated as \( \hat{\beta}_M \hat{\gamma} \) for \( M \) and \( \hat{\beta}_W \hat{\theta} \) for \( W \).

We can show that these are consistent for \( \bar{\delta}_i^M \) and \( \bar{\delta}_i^W \) under the above assumption and linearity.

However, because of the assumed independence between mechanisms, analyzing one mechanism at a time will also be valid, e.g.,

\[ M_i = \alpha_2 + \beta_2 T_i + \xi_2^T X_i + \epsilon_{i2} \]
\[ Y_i = \alpha_3 + \beta_3 T_i + \gamma M_i + \xi_3^T X_i + \epsilon_{i3} \]
Consider the (weak) sequential ignorability assumption:

\[
\begin{align*}
\{ Y_i(t, m, w), M_i(t, w), W_i(t) \} & \perp \perp T_i \mid X_i = x \\
\{ Y_i(t, m, w), M_i(t, w) \} & \perp \perp W_i \mid T_i = t, X_i = x \\
\{ Y_i(t, m, w) \} & \perp \perp M_i \mid W_i(t) = w, T_i = t, X_i = x
\end{align*}
\]

for any \( t, m, w, x \).

Unlike sequential ignorability:

- Unconfoundedness of \( M_i \) conditional on both pre-treatment \((X_i)\) and observed post-treatment \((W_i)\) confounders

- The no \( T \times M \) interaction assumption required for the identification of \( \overline{\delta}(t) \):

\[
Y_i(1, m, W_i(1)) - Y_i(0, m, W_i(0)) = Y_i(1, m', W_i(1)) - Y_i(0, m', W_i(0))
\]
The Proposed Framework

- **Problem:** The no interaction assumption is often too strong (e.g. Does the effect of perceived issue importance invariant across frames?)

- **We use a varying-coefficient linear structural equations model to:**
  1. Allow for **homogeneous interaction** for point identification
  2. Develop a **sensitivity analysis** in terms of the degree of heterogeneity in the interaction effect

- **Consider the following model:**

  \[
  M_i(t, w) = \alpha_2 + \beta_{2i}t + \xi_2^\top w + \mu_{2i}^\top tw + \lambda_{2i}^\top x + \epsilon_{2i},
  \]

  \[
  Y_i(t, m, w) = \alpha_3 + \beta_{3i}t + \gamma_i m + \kappa_i tm + \xi_{3i}^\top w + \mu_{3i}^\top tw + \lambda_{3i}^\top x + \epsilon_{3i},
  \]

  where \( \mathbb{E}(\epsilon_{2i}) = \mathbb{E}(\epsilon_{3i}) = 0 \)

- **Allows for dependence of \( M \) on \( W \)**

- **Coefficients are allowed to vary arbitrarily across units**
Example: Druckman and Nelson (2003) ($N = 261$)

- Treatment: News paper article on a proposed election campaign finance reform, emphasizing either its positive or negative aspect
- Outcome: Support for the proposed reform
- Primary mediator: Perceived importance of free speech
- Alternative (confounding) mediator: Belief about the impact of the proposed reform

Original analysis finds the importance mechanism to be significant, implicitly assuming its independence from beliefs
Original Analysis Assumes Independent Mechanisms

Druckman and Nelson, p. 738

Diagram showing causal relationships:
- Frame
- Free speech import.
- Special interests import.
- Impact of reform on free speech
- Impact of reform on special interests
- Support for McCain-Feingold

Causality paths:
- Frame → Free speech import: -.22**
- Frame → Special interests import: .17*
- Frame → Impact of reform on free speech: .19**
- Frame → Impact of reform on special interests: -.04
- Free speech import → Support for McCain-Feingold: -.31**
- Special interests import → Support for McCain-Feingold: .51**
- Impact of reform on free speech → Support for McCain-Feingold: .06
- Impact of reform on special interests → Support for McCain-Feingold: .07
- Weakly significant average indirect effects ([0.025, 0.625]), accounting for 28.6% of the total effect
- Moderate degree of sensitivity to the mediator exogeneity ($\bar{\delta} = 0$ when $\rho = -0.43$ or $\tilde{R}_M^2 \tilde{R}_Y^2 = 0.078$)
- Concern: the importance mechanism may be affected by the belief content mechanism
Similar results with slightly wider CI \([-0.021, 0.648]\)

- Lower bound on \(\bar{\delta}\) is zero when \(\sigma = 0.195\), or 51\% of its upper bound
- This translates to the interaction heterogeneity explaining 15.9\% of the variance of the outcome variable
Concluding Remarks

- Even in a randomized experiment, a strong assumption is needed to identify causal mechanisms.
- However, progress can be made toward this fundamental goal of scientific research with modern statistical tools.
- A general, flexible estimation method is available once we assume sequential ignorability.
- Sequential ignorability can be probed via sensitivity analysis.
- More credible inferences are possible using clever experimental designs.
- Insights from new experimental designs can be directly applied when designing observational studies.
- Multiple mediators require additional care when they are causally dependent.
Yes, but it is crucial to understand mechanisms:
- scientists want to test theories which are about mechanisms
- policy makers want to devise better policies
- understanding of mechanisms $\leadsto$ external validity

Two ways to address the question, “why does a treatment work?”
1. mediation $\leadsto$ causal process
2. interaction $\leadsto$ causal components
What do you think about mechanism experiments?

- “mechanism experiments” (Ludwig, Kling, and Mullainathan, 2011)
- “causal chain approach” (Spencer, Zanna, and Fong, 2005)
  1. Randomize $T$ to identify its effect on $Y$ and its effect on $M$
  2. Randomize $M$ to identify its effect on $Y$

- This is certainly a progress towards understanding mechanisms

- Two issues with this approach (Imai, Tingley, and Yamamoto, JRSSA, 2013):
  1. Effects of direct manipulation of $M$ may differ from those of “natural” change in $M$ induced by $T$
  2. Effect heterogeneity: even if the average effect of $T$ on $M$ and that of $M$ on $Y$ are both positive, the average mediation effect of $T$ on $Y$ can be negative
How sensitive do the results of sensitivity analysis have to be before doubting mediation analysis?

- What sensitivity analysis provides: the amount of hidden bias that makes one’s mediational results go away

- Traditional tests: sampling uncertainty of one’s mediational effects that are assumed to be identifiable with the infinite amount of data

- Can a scientific community agree on the required degree of sensitivity? ~ maybe not

- Rosenbaum’s example:
  1. Effect of smoking on cancer: $\Gamma = 6$
  2. Effect of coffee on myocardial infarction: $\Gamma = 1.3$

- Need to accumulate sensitivity analysis results
- Need to look for confounders that reduce sensitivity
Other Questions

1. Why can’t we just show those who have the large effects of \( T \) on \( M \) also exhibit the large effects of \( M \) on \( Y \)?
   - Yes, but those effects must be identified
   - Reducing heterogeneity helps the identification of mediation effects

2. Is mediation analysis uninformative because it can hardly be definitive?
   - No. Almost no scientific study can be definitive.
   - But mediation is about purely counterfactual quantities

3. What researchers can do to maximize the plausibility of sequential ignorability?
   - Better design with clever manipulation of mediators
   - Importance of sensitivity analysis
Project References (click the article titles)

- **General:**
  - Unpacking the Black Box of Causality: Learning about Causal Mechanisms from Experimental and Observational Studies. *American Political Science Review*

- **Theory:**

- **Extensions:**
  - A General Approach to Causal Mediation Analysis. *Psychological Methods*
  - Experimental Designs for Identifying Causal Mechanisms. *Journal of the Royal Statistical Society, Series A (with discussions)*
  - Identification and Sensitivity Analysis for Multiple Causal Mechanisms: Revisiting Evidence from Framing Experiments. *Political Analysis*

- **Software:**
  - mediation: R Package for Causal Mediation Analysis. *Journal of Statistical Software*
The project website for papers and software:

http://imai.princeton.edu/projects/mechanisms.html

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