

Identification and Sensitivity Analysis for Multiple Causal Mechanisms: Revisiting Evidence from Framing Experiments

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Social scientists are often interested in testing multiple causal mechanisms through which a treatment affects outcomes. A predominant approach has been to use linear structural equation models and examine the statistical significance of the corresponding path coefficients. However, this approach implicitly assumes that the multiple mechanisms are causally independent of one another. In this article, we consider a set of alternative assumptions that are sufficient to identify the average causal mediation effects when multiple, causally related mediators exist. We develop a new sensitivity analysis for examining the robustness of empirical findings to the potential violation of a key identification assumption. We apply the proposed methods to three political psychology experiments, which examine alternative causal pathways between media framing and public opinion. Our analysis reveals that the validity of original conclusions is highly reliant on the assumed independence of alternative causal mechanisms, highlighting the importance of proposed sensitivity analysis. All of the proposed methods can be implemented via an open source R package, *mediation*.

1 Introduction

The identification of causal mechanisms is an important goal of empirical social science research. Researchers are often not only interested in the question of whether a particular causal variable of interest affects outcomes, but they also wish to understand the mechanisms through which such causal effects arise. Causal mediation analysis represents a formal statistical framework that can be used to study causal mechanisms. For example, Imai et al. (2011) identify a set of commonly invoked assumptions under which such an analysis can be justified, develop general estimation algorithms, and propose sensitivity analysis and new research design strategies. Indeed, there exists a fast-growing methodological literature about how to study causal mechanisms through the use of statistical methods (see Robins and Greenland 1992; Pearl 2001; Petersen, Sinisi, and van der Laan 2006; VanderWeele 2009; Imai, Keele, and Yamamoto 2010c; Glynn 2012; and many others). With such methodological advances, researchers can now base their mediation analysis on a rigorous statistical foundation.

Nevertheless, an important deficiency of the existing methodologies is their limited ability to handle multiple causal mechanisms of interest (see, e.g., Bullock, Green, and Ha 2010; Imai et al. 2011). Given this current state of the literature, the prevailing practice among applied researchers is to assume, often without explicitly stating it, that no causal relationship exists among these

Authors' note: The proposed methods can be implemented via open-source software *mediation* that is freely available as an R package at the Comprehensive R Archive Network (CRAN, <http://cran.r-project.org/package=mediation>). The replication archive for this article is available online as Imai and Yamamoto (2012). We are grateful to Ted Brader, Jamie Druckman, and Rune Slothuus for sharing their data with us. We thank Dustin Tingley and Mike Tomz for useful discussions that motivated this article. John Bullock, Adam Glynn, and Tyler VanderWeele provided helpful suggestions. We also thank the Associate Editor and two anonymous referees for their comments that significantly improved the paper. An earlier version of this article was circulated under the title "Sensitivity Analysis for Causal Mediation Effects under Alternative Exogeneity Conditions."

alternative mechanisms. In many applications, however, such an assumption is not credible because competing theoretical explanations are often closely tied to each other.

In this article, we consider the identification and sensitivity analysis of multiple causal mechanisms by extending causal mediation analysis to the cases involving several mediators that are causally related to each other. Figure 1 presents two causal diagrams with multiple causal mechanisms. They represent the specific scenarios studied in this article, where the treatment variable T could affect the outcome Y in three ways: through the main mediator of interest M (red arrows), through the set of alternative mediators W , and directly. The critical difference between Figs. 1(a) and (b) is that in the former M and W are assumed to have no causal relationship with one another, whereas in the latter W may possibly affect M (but not the other way around). We first show that the standard mediation analysis assumes the causal independence between multiple mediators as in Fig. 1(a), regardless of whether alternative mediators W are measured. We then develop a set of statistical methods to relax this assumption and address the situation in Fig. 1(b).¹

In Section 2, we introduce three experimental studies from the political psychology literature on framing effects, which we use as illustrative examples throughout this article. All of these studies investigate how issue frames affect opinions and behavior through multiple causal pathways, such as changes in perceived issue importance or belief content. To identify these multiple mechanisms, they rely on a traditional path analytic method which, as we show, implicitly assumes the absence of causal relationship between the corresponding mediators. This assumption is problematic from a theoretical point of view because, for example, framing may also alter the perceived importance of the issue by changing the factual content of beliefs (Miller 2007).

In Section 3, we use a formal statistical framework of causal inference and show that the standard causal mediation analysis, including the ones conducted in these framing studies, assumes causal independence between multiple causal mechanisms. Under this independence assumption, there is no need to measure alternative mediators W to identify the main mediation effect of interest. After reviewing the assumptions required for the identification of independent multiple mechanisms, we reanalyze the data from the framing experiments under these assumptions. The results suggest that the empirical conclusions in the original studies are largely valid so long as these mechanisms are independent of each other, although statistical significance appears to be somewhat lessened for some of the studies.

In Section 5, we develop new statistical methods that allow for the existence of multiple causal mechanisms that are causally related to each other. Our methods are formulated within a framework of familiar linear regression models, and yet the models are much more flexible than standard regression models because every coefficient is allowed to vary across individual observations in an arbitrary fashion: the model can be considered as semiparametric, making no distributional assumption about varying coefficients. Thus, the proposed model encompasses a large class of realistic statistical models that can be applied to various social science research projects while maintaining the simplicity and interpretability of the linear model.

Under this framework of multiple mechanisms, we first consider the identification assumptions originally introduced by Robins (2003), which allow for the presence of competing mechanisms. Robins assumes that the treatment assignment is exogenous given a set of observed pretreatment covariates and that the mediator of interest is also exogenous conditional on observed pre- and posttreatment covariates. This setup allows for the existence of alternative mediators that confound the relationship between the main mediator of interest and the outcome, provided that they are all observed. Under this circumstance, Robins shows that the assumption of no interaction effect between the treatment and the mediator (i.e., the causal effect of the mediator on the outcome does not depend on the treatment status) is sufficient for identifying the causal mechanism of interest.

However, as previously noted by many researchers, including Robins himself (e.g., Petersen, Sinisi, and van der Laan 2006; Imai, Tingley, and Yamamoto 2013), the assumption of no

¹The other important situation involving multiple mechanisms is cyclic causation, i.e., both W and M cause each other. Identifying whether the causal relationship is cyclic or acyclic is difficult without some help of prior theoretical or empirical information, and care must be taken so that the assumed causal ordering is plausible. Here, we focus on the case of Fig. 1(b), in part motivated by the primary concern in the framing literature (see the discussion in Section 4.1).

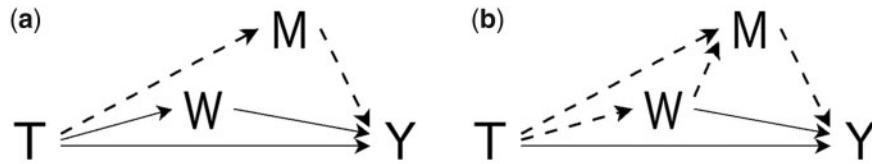


Fig. 1 Causal diagrams with multiple causal mechanisms. In Panel (a), the treatment variable T can affect the outcome variable Y either through the mediator of interest M (dashed arrows), through alternative mediators W , or directly (solid bottom arrow). In addition, the causal relationship between M and W is assumed to be nonexistent. Panel (b) represents an alternative scenario, where W can affect Y either directly or through M , thereby allowing for the potential causal relationship between M and W . The quantity of interest for the proposed methods is the ACME with respect to M , which is represented by the dashed arrows connecting T and Y through M in both panels.

treatment–mediator interaction is often too strong in empirical applications. For example, in framing experiments, the effect of the perceived issue importance on opinions may well depend on which frame was initially given. Therefore, we relax this key identification assumption by developing a new sensitivity analysis that assesses the robustness of empirical results to the potential violation of this key identification assumption. Our sensitivity analysis also directly addresses “the product and difference fallacies” pointed out by Glynn (2012) because his model is a special case of our model. We illustrate the proposed methods by applying them to the framing experiments from the political psychology literature. Our analysis reveals that the validity of original findings is highly reliant on the assumed independence of alternative causal mechanisms, implying the essential role of identification and sensitivity analyses in the study of multiple mediators.

Causal mediation analysis, like many other tools of causal inference, relies on untestable assumptions. Thus, it is essential for applied researchers to examine the robustness of empirical findings to the violation of key identification assumptions. While there exist sensitivity analysis tools in the literature, many of them deal with *pretreatment* confounders and essentially assume the absence of causally related alternative mediators (e.g., Hafeman 2008; Imai, Keele, and Tingley 2010a; Imai, Keele, and Yamamoto 2010c; VanderWeele 2010). In contrast to these previous studies, the sensitivity analysis proposed in this article addresses the possible existence of *posttreatment* confounders. Sensitivity analyses that can handle posttreatment confounders are just beginning to appear in the methodological literature on causal mediation (e.g., Albert and Nelson 2011; Tchetgen Tchetgen and Shpitser 2011).

Nevertheless, one limitation of the proposed approach is that, even though it addresses the potential violation of the no treatment–mediator interaction assumption, it still hinges on another untestable assumption of no unmeasured confounder for the mediator. To address this issue, in Section 7, we consider the extensions of the proposed method to the new experimental designs recently developed by Imai, Tingley, and Yamamoto (2013). In particular, we consider the “parallel design” where the sample is randomly divided into two groups and separate randomized experiments are conducted in parallel for those groups.² We also consider the “parallel encouragement design,” a natural generalization of the parallel design for the situation, where the direct manipulation of the mediator is difficult. While the randomization of the treatment and the mediator is not sufficient for the identification of the ACME (Imai, Tingley, and Yamamoto 2013), both experimental designs relax the unconfoundedness assumption with respect to the treatment and the mediator. This eliminates the need to measure alternative mediators and allows us to develop a sensitivity analysis for the key identification assumption. While these experimental designs are new and hence have not yet been used by many applied researchers, we hope that their future use can improve the credibility of causal mediation analysis. In addition, these new

²In one experiment, researchers randomize the treatment and observe the values of the mediator and the outcome. In the other experiment, both the treatment and the mediating variables are randomized and subsequently the values of the outcome variable are recorded.

experimental designs can serve as templates for designing observational studies (see Imai et al. 2011 for some examples).

Finally, Section 8 offers concluding remarks and some suggestions for applied researchers who wish to study multiple causal mechanisms. All of our proposed methods can be implemented through the open-source software program *mediation* (Imai et al. 2010b; Tingley et al. 2012), which is freely available at the Comprehensive R Archive Network (CRAN, <http://cran.r-project.org/package=mediation>).

2 Framing Experiments in Political Psychology

In this section, we introduce three empirical studies of framing effects, which serve as examples throughout the article. In political psychology, scholars are interested in whether and how the framing of political issues in mass media and elite communications affects citizens' political opinion and behavior. Psychological theory suggests that issue framing, or a presenter's deliberate emphasis on certain aspects of an issue, may affect how individuals perceive the issue and change their attitudes and behavior (Tversky and Kahneman 1981).

If citizens are prone to such cognitive biases when interpreting media contents, political elites may be able to influence, or even manipulate, the public opinion by carefully choosing the languages they use in their communication through mass media (Zaller 1992). While early studies focused on identifying the issue areas in which such framing effects manifest (e.g., Kinder and Sanders 1990; Iyengar 1991; Nelson and Kinder 1996), recent studies address the question of the mechanisms through which framing affects public opinion and political behavior (e.g., Nelson, Clawson, and Oxley 1997; Callaghan and Schnell 2005; Chong and Druckman 2007).

Below, we briefly describe three experimental studies that are aimed at the identification of such mechanisms. Aside from their prominence in the literature, these studies all explicitly examine more than one causal mechanism by measuring multiple mediators corresponding to those competing possible pathways. In each of these studies, the authors implicitly assume that the multiple causal pathways under study are independent of one another. As we discuss below, however, this assumption is theoretically implausible and statistically problematic, for its violation can lead to a substantial bias in the estimated importance of causal mechanisms.

2.1 Druckman and Nelson's Campaign Finance Reform Experiment

One of the most important debates in the framing effects literature concerns whether issue framing affects citizens' opinions by shifting the perceived importance of the issue (hereafter the "importance" mechanism) or changing the content of their belief about the issue (hereafter the "content" mechanism). As part of their experimental study on the interaction between issue framing and interpersonal conversations, Druckman and Nelson (2003) examine this question using a path analytic approach. First, they randomly assign each of their 261 study participants to one of the two conditions. In one condition, the subject is asked to read an article on proposed campaign finance reform that emphasizes its possible violation of free speech. In the other condition, the assigned article emphasizes the potential of campaign finance reform to limit special interests. Then, after additionally randomizing whether the participants will engage in discussion, the authors measured the two mediators: the participants' perceived importance of free speech and special interests as well as their belief about the impact of the proposed reform on these items. Finally, the authors measured the outcome variable, the overall level of support for the proposed campaign finance reform. The substantive question of interest for the original authors is whether the effect of the frames on the support levels are mediated by the importance mechanism or the content mechanism.

2.2 Slothuus' Social Welfare Reform Experiment

Following Druckman and Nelson (2003), Slothuus (2008) conducted a randomized experiment to analyze the above two mechanisms of framing effects. Using a sample of 408 Danish students,

the author examined how two versions of a newspaper article on a social welfare reform bill—one emphasizing the reform’s supposed positive effect on job creation and the other focusing on its negative impact on low-income population—affect differently the participants’ opinion about the reform. After randomly assigning participants to either the “job frame” or the “poor frame,” the author measured the two mediators, i.e., the importance and the content of issue-related considerations, by asking a series of 5-point-scale questions. Finally, the outcome variable was measured by asking the participants whether and to what extent they agree or disagree with the proposed welfare reform. Similar to the previous study, the key substantive question of interest is whether the framing effects transmit through the importance mechanism or the content mechanism.

2.3 Brader, Valentino, and Suhay’s Immigration Experiment

The third study we analyze also investigates the causal mechanisms underlying framing effects but focuses on the role of emotions as opposed to more conscious beliefs about the issue. Brader, Valentino, and Suhay (2008) report the results of their randomized experiment on the framing of immigration policy. As part of a nationally representative survey of 354 White non-Latino adults, they randomly assigned different versions of a mock *New York Times* article about immigration by varying the origin of the featured immigrant as well as the tone of the story. The article featured either a European or Latino immigrant and emphasized either the positive or negative consequences of increased immigration. After the treatment, the authors measured the participants’ belief about the likely negative impact of immigration (hereafter the “perceived harm” mechanism) as well as their emotions by asking how they feel about increased immigration (hereafter the “anxiety” mechanism). Finally, the authors recorded the participants’ opinions and behavioral reactions to an increase in immigration. The main goal was to identify the mechanism through which the framing effects of the news stories operated.

3 Identification of Independent Multiple Mechanisms

In this section, we use the formal statistical framework of causal inference and show that the standard causal mediation analysis commonly used in empirical studies (including our running examples introduced in the previous section) implicitly assumes the independence of competing causal mechanisms. We then reanalyze the framing experiments described above under this independence assumption.

3.1 Causal Mediation Analysis with a Single Mediator: A Review

We begin by briefly reviewing the standard causal mediation analysis with a single mediator (see Imai et al. [2011] for a more detailed explanation). Suppose that we have a simple random sample of size n from a population of interest \mathcal{P} . Let T_i be a binary treatment variable, which equals one if unit i receives the treatment and is equal to zero otherwise. We use M_i and Y_i to denote the observed value of the mediator and the outcome of interest for unit i , respectively. Under the formal statistical framework of causal inference (Neyman 1923; Rubin 1974; Holland 1986), we write $M_i(t)$ to represent the potential mediator value under the treatment status $t = 0, 1$, where the observed value M_i equals the potential value of the mediator under the observed treatment status $M_i(T_i)$. Similarly, we use $Y_i(t, m)$ to denote the potential outcome under the treatment status t and the mediator value m , where the observed outcome Y_i equals $Y_i(T_i, M_i(T_i))$. We assume throughout the rest of the article that there is no measurement error in the observed values of the variables.³

In the literature of causal mediation analysis initiated by Robins and Greenland (1992) and Pearl (2001), the causal mediation effect (or indirect effect) for unit i given the treatment status t is defined as

³Little is known about the consequences of measurement error in causal mediation analysis. The simplest case is classical measurement error in one (and only one) of the variables, which will lead to an attenuation bias in the estimate of the regression coefficient corresponding to the mismeasured variable. It is likely, however, that more complex patterns of measurement error often exist in practice. Analysis of such cases is beyond the scope of the current paper.

$$\delta_i(t) \equiv Y_i(t, M_i(1)) - Y_i(t, M_i(0)), \quad (1)$$

which represents the causal effect of the treatment on the outcome that can be attributed to the treatment-induced change in the mediator. This quantity represents the change in outcome under the scenario where the treatment variable is held constant at t and the mediator is changed from $M_i(0)$ to $M_i(1)$. Similarly, the unit-level direct effect of the treatment is defined as

$$\zeta_i(t) \equiv Y_i(1, M_i(t)) - Y_i(0, M_i(t)), \quad (2)$$

which denotes the causal effect of the treatment on the outcome that can be attributed to causal mechanisms other than the one represented by the mediator. Here, the mediator is held constant at $M_i(t)$ and the treatment variable is changed from zero to one.

Finally, the sum of these direct and indirect effects equals the following total effect, which formally decomposes the total effect into the direct and indirect effects,

$$\tau_i \equiv Y_i(1, M_i(1)) - Y_i(0, M_i(0)) = \delta_i(t) + \zeta_i(1 - t). \quad (3)$$

If we assume that the indirect and direct effects do not depend on the treatment status, i.e.,

$$\delta_i(t) = \delta_i \quad \text{and} \quad \zeta_i(t) = \zeta_i, \quad (4)$$

for each t , then we have simpler decomposition $\tau_i = \delta_i + \zeta_i$. Given these unit-level causal quantities of interest, we can define the population average effect for each quantity,

$$\bar{\delta}(t) = \mathbb{E}(\delta_i(t)), \quad \bar{\zeta} = \mathbb{E}(\zeta_i(t)), \quad \text{and} \quad \bar{\tau} = \mathbb{E}(\tau_i). \quad (5)$$

The goal of causal mediation analysis is, therefore, to decompose the total treatment effect into the direct and indirect effects, where the former corresponds to the causal mechanism of interest and the latter represents all other causal mechanisms.

The standard mediation analysis most commonly used across various social science disciplines entails the process of fitting the following two linear regressions separately,

$$M_i = \alpha_2 + \beta_2 T_i + \xi_2^\top X_i + \varepsilon_{i2}, \quad (6)$$

$$Y_i = \alpha_3 + \beta_3 T_i + \gamma M_i + \xi_3^\top X_i + \varepsilon_{i3}, \quad (7)$$

where X_i represents a vector of observed *pretreatment* confounders. After fitting these two models, researchers compute the product of two coefficients, i.e., $\hat{\beta}_2 \hat{\gamma}$, and interpret it as an estimate of the ACME $\delta(t)$, whereas the estimated coefficient $\hat{\beta}_3$ is interpreted as an estimate of the average direct effect (ADE) $\zeta(t)$. Alternatively, researchers can fit the regression model given in equation (7) as well as the following regression model,

$$Y_i = \alpha_1 + \beta_1 T_i + \xi_1^\top X_i + \varepsilon_{i1}, \quad (8)$$

and compute the difference of the two coefficients, i.e., $\hat{\beta}_1 - \hat{\beta}_3$, to obtain the estimated average mediation effect. Here, $\hat{\beta}_1$ is taken as an estimate of the average total effect.

Imai, Keele, and Yamamoto (2010c) prove that this standard mediation analysis can be justified under the following sequential ignorability assumption,

$$\{Y_i(t, m), M_i(t')\} \perp\!\!\!\perp T_i | X_i = x \quad (9)$$

$$Y_i(t', m) \perp\!\!\!\perp M_i | T_i = t, X_i = x, \quad (10)$$

for any value of x, t, t', m , and every unit i . In fact, it has been shown that under this assumption, the ACMEs are nonparametrically identified, i.e., without any functional form or distributional assumptions. Imai, Keele, and Tingley (2010a) develop general algorithms to compute an estimate of the ACME and its uncertainty given any statistical models specified by applied researchers (linear, nonlinear, parametric, nonparametric models, etc.).

Is the sequential ignorability assumption plausible? While equation (9) is guaranteed to hold in a standard randomized experiment where the treatment is randomly administered, equation (10) is often difficult to justify in many practical situations. The reason is two-fold. First, equation (10)

implies that there must not be any unobserved pretreatment confounding between the observed mediator and outcome once the treatment and the observed pretreatment covariates are conditioned on. Second, equation (10) also requires that there be no posttreatment confounding between the mediator and the outcome whatsoever, observed or unobserved. If equation (10) does not hold for either (or both) of these two reasons, the sequential ignorability assumption is violated and the ACME cannot be nonparametrically identified.

To address the possibility that equation (10) may not hold, Imai, Keele, and Yamamoto (2010c) develop a sensitivity analysis under the framework of the structural linear equations model. Their method relies on the fact that under equations (6) and (7), one can summarize the degree of the violation of equation (10) by the correlation coefficient between the two error terms, i.e., $\rho \equiv \text{Corr}(\varepsilon_{i2}, \varepsilon_{i3})$. They show that the ACME can be identified given a value of ρ and also that equation (10) implies $\rho = 0$. Thus, one can analyze the robustness of the structural equations-based estimate of the ACME to the violation of sequential ignorability by examining how the value of the ACME varies as a function of ρ .

Moreover, Imai, Keele, and Yamamoto (2010c) propose an alternative formulation of the sensitivity analysis that may be easier to interpret. This version is based on the idea that the error terms ε_{i2} and ε_{i3} can each be decomposed into a common unobserved pretreatment confounder plus an independent random disturbance, i.e., $\varepsilon_{ij} = \lambda_j U_i + \varepsilon'_{ij}$ for $j=2, 3$. Then, the degree of the sequential ignorability violation can be summarized by the importance of this common term (but with different coefficients) in explaining variations in the mediator and the outcome, which is represented as the (partial) coefficients of determination, i.e., $\tilde{R}_M^2 \equiv \{\mathbb{V}(\varepsilon_{i2}) - \mathbb{V}(\varepsilon'_{i2})\} / \mathbb{V}(M_i)$ and $\tilde{R}_Y^2 \equiv \{\mathbb{V}(\varepsilon_{i3}) - \mathbb{V}(\varepsilon'_{i3})\} / \mathbb{V}(Y_i)$. They show that the ACME can also be expressed as a function of \tilde{R}_M^2 and \tilde{R}_Y^2 , making it possible to conduct the equivalent sensitivity analysis with respect to these alternative parameters.

3.2 Assumed Independence of Multiple Causal Mechanisms

While these identification results and sensitivity analysis provide a useful framework for analyzing a single causal mechanism, it leaves an important situation unaddressed. Recall that equation (10) can fail because of two reasons: unobserved pretreatment confounding or any posttreatment confounding. This means that even if the mediator can be assumed to be exogenous after conditioning on a vector of observed posttreatment confounders (denoted by W_i) as well as T_i and X_i (i.e., no unobserved confounding, but observed posttreatment confounding), sequential ignorability is violated and the standard causal mediation analysis cannot identify the average mediation effects. In addition, while the above sensitivity analysis addresses the problem of potential unobserved pretreatment confounding, it cannot cope with the possibility of posttreatment confounding, such as W_i . Therefore, this framework assumes the absence of any form of posttreatment confounding.

Now, consider the problem of multiple mechanisms. Since alternative mediators (W_i) are by definition causally affected by the treatment and influence the outcome, the assumption of no posttreatment confounding is equivalent to assuming that these alternative mediators do not causally influence the mediator of interest. Going back to Fig. 1, this means that the standard analysis based on the sequential ignorability assumption implicitly presupposes a situation like Fig. 1(a), where the causal relationship between the mediator of interest M_i and alternative mediators W_i is absent. In contrast, Fig. 1(b) corresponds to a case where alternative mediators W_i causally affect the mediator of interest M_i . It is easy to see that the latter scenario violates the sequential ignorability assumption because W_i is a set of posttreatment variables that confound the relationship between M_i and Y_i . Thus, the standard causal mediation analysis assumes the independence between the causal mechanism of interest and other alternative mechanisms. As shown below, another difficulty resulting from this limitation of the standard causal mediation analysis is that this independence assumption is not directly testable from the observed data.

We now formalize the above argument. To make the existence of alternative mediators explicit, we introduce potential values of those other mediators W_i and denote them as $W_i(t)$ for $t=0, 1$, implying that W_i is a posttreatment variable and hence possibly affected by the treatment. If there is

no causal relationship between M_i and W_i as in Fig. 1(a), the potential value of M_i is not a function of W_i and thus can be written as before, i.e., $M_i(t)$. The potential outcomes, on the other hand, depend on both M_i and W_i and are denoted by $Y_i(t, m, w)$ for any t, m, w . The observed outcome Y_i equals $Y_i(T_i, M_i(T_i), W_i(T_i))$. We emphasize that W_i could be either a single alternative mediator or a vector of multiple alternative mediators, and that those mediators may or may not be causally related to each other. Our framework does not require that researchers specify causal relationships among these alternative mechanisms.

Under this setup, we can define the two types of causal mediation effects, one with respect to M_i and the other with respect to W_i ,

$$\delta_i^M(t) \equiv Y_i(t, M_i(1), W_i(t)) - Y_i(t, M_i(0), W_i(t)), \quad (11)$$

$$\delta_i^W(t) \equiv Y_i(t, M_i(t), W_i(1)) - Y_i(t, M_i(t), W_i(0)), \quad (12)$$

for $t=0, 1$, where $\delta_i^M(t)$ ($\delta_i^W(t)$) represents the unit-level indirect effect of the treatment on the outcome through the mediator M_i (W_i) while holding the treatment at t and the other mediator at its value that would be realized under the same treatment status, i.e., $W_i(t)$ ($M_i(t)$). For example, in framing experiments, such as those by Druckman and Nelson (2003) and Slothuus (2008), $\delta_i^M(t)$ represents the effect of issue frames on opinions that goes through changes in the perceived importance of the issue induced by the framing effect, whereas $\delta_i^W(t)$ equals the portion of the framing effect that operates through changes in belief content. As before, for each of these two causal mediation effects, we can define the ACME as $\bar{\delta}^M(t) \equiv \mathbb{E}(\delta_i^M(t))$ or $\bar{\delta}^W(t) \equiv \mathbb{E}(\delta_i^W(t))$ by averaging it over the target population \mathcal{P} .

We emphasize that both $\delta_i^M(t)$ and $\delta_i^W(t)$ are counterfactual quantities because, for example, $M_i(t)$ and $W_i(t')$ are never jointly observed at the same time when $t \neq t'$. Similarly, $Y_i(t, M_i(t'), W_i(t))$ can never be observed unless $M_i(t') = M_i(t)$. This counterfactual nature leads some scholars to question the value of causal mediation analysis (e.g., Rubin 2004). Nevertheless, these indirect effects formalize causal processes that are often of scientific importance to applied researchers, and hence our goal is to offer a set of statistical methods that take seriously the required identification assumptions.

In addition, the unit-level direct effect can be defined as

$$\zeta_i(t, t') \equiv Y_i(1, M_i(t), W_i(t')) - Y_i(0, M_i(t), W_i(t')) \quad (13)$$

for each $t, t' = 0, 1$, where $\zeta_i(1, 0)$, for example, represents the direct effect of the treatment while holding the mediators at $(M_i(1), W_i(0))$. Again, in Druckman and Nelson's experiment, this quantity represents the effect of frames that does not operate through either the importance mechanism or the content mechanism. As before, the ADEs are given by $\bar{\zeta}(t, t') \equiv \mathbb{E}(\zeta_i(t, t'))$. Given these definitions, we can decompose the total effect as the sum of direct and mediation (indirect) effects,

$$\begin{aligned} \tau_i &\equiv Y_i(1, M_i(1), W_i(1)) - Y_i(0, M_i(0), W_i(0)) \\ &= \delta_i^M(t) + \delta_i^W(1 - t) + \zeta_i(1 - t, t), \end{aligned}$$

for $t=0, 1$.⁴ As in the case of a single mediator, we can simplify this expression under the no-interaction assumptions,

⁴To verify this relationship, note that

$$\begin{aligned} \delta_i^M(1) + \delta_i^W(0) + \zeta_i(1, 0) &= Y_i(1, M_i(1), W_i(1)) - Y_i(1, M_i(0), W_i(1)) \\ &\quad + Y_i(0, M_i(0), W_i(1)) - Y_i(0, M_i(0), W_i(0)) \\ &\quad + Y_i(1, M_i(0), W_i(1)) - Y_i(0, M_i(0), W_i(1)) = \tau_i, \end{aligned}$$

and

$$\begin{aligned} \delta_i^M(0) + \delta_i^W(1) + \zeta_i(0, 1) &= Y_i(0, M_i(1), W_i(0)) - Y_i(0, M_i(0), W_i(0)) \\ &\quad + Y_i(1, M_i(1), W_i(1)) - Y_i(1, M_i(1), W_i(0)) \\ &\quad + Y_i(1, M_i(1), W_i(0)) - Y_i(0, M_i(1), W_i(0)) = \tau_i. \end{aligned}$$

$$\delta_i^M(t) = \delta_i^M, \quad \delta_i^W(t) = \delta_i^W, \quad \text{and} \quad \zeta_i(t, t') = \zeta_i, \quad (14)$$

for any t, t' . Under these conditions, we have a simpler decomposition relationship, i.e., $\tau_i = \delta_i^M + \delta_i^W + \zeta_i$.

We now generalize the sequential ignorability assumption to the case with multiple mediators, where the causal independence between the main mediator of interest M_i and alternative mediators W_i is assumed.

Assumption 1 (Sequential Ignorability with Multiple Causally Independent Mediators). *We assume that the following three conditional independence statements hold:*

$$\{Y_i(t, m, w), M_i(t'), W_i(t'')\} \perp\!\!\!\perp T_i | X_i = x, \quad (15)$$

$$Y_i(t', m, W_i(t')) \perp\!\!\!\perp M_i | T_i = t, X_i = x, \quad (16)$$

$$Y_i(t', M_i(t'), w) \perp\!\!\!\perp W_i | T_i = t, X_i = x, \quad (17)$$

where $0 < \Pr(T_i = t | X_i = x)$ and $0 < p(M_i = m, W_i = w | T_i = t, X_i = x)$ for any x, t, t', m, w .

Under this assumption, it can be shown that the ACMEs, $\bar{\delta}^M(t)$ and $\bar{\delta}^W(t)$, as well as the ADEs $\bar{\zeta}(t, t')$ are nonparametrically identified and expressed by the same formula as Theorem 1 of Imai, Keele, and Yamamoto (2010c). Proof of this identification result is provided in Appendix A1.

Can the assumption of no causal relationship between these mediators be tested using the observed data? We note that Assumption 1 neither implies nor is implied by the conditional independence between the observed values of M_i and W_i given the treatment T_i and observed pretreatment confounders X_i . This means that there exists no direct test of the assumed independence between causal mechanisms. However, we suggest that researchers at least check the degree of statistical dependence between M_i and W_i given (T_i, X_i) as well as the association between W_i and T_i given X_i because these strong dependent relationships are likely to indicate the violation of Assumption 1. Another important limitation of Assumption 1 is that equations (16) and (17) assume what is sometimes called the “cross-world” independence. For example, the independence in equation (16) conditions on $T_i = t$, whereas the potential outcome $Y_i(t', m, W_i(t'))$ conditions on a possibly different treatment condition, i.e., $T_i = t'$. This issue is further discussed in Section 5, where we relax the restriction.

Next, we show that the standard analysis of multiple mediators in many social science applications, such as those described in Section 2, implicitly relies on Assumption 1. While our fundamental point applies more generally, we use the following linear structural equations model, which is by far the most commonly used method, to illustrate the point clearly:

$$M_i = \alpha_M + \beta_M T_i + \xi_M^\top X_i + \varepsilon_{iM}, \quad (18)$$

$$W_i = \alpha_W + \beta_W T_i + \xi_W^\top X_i + \varepsilon_{iW}, \quad (19)$$

$$Y_i = \alpha_3 + \beta_3 T_i + \gamma M_i + \theta^\top W_i + \xi_3^\top X_i + \varepsilon_{i3}. \quad (20)$$

In the standard procedure, the product of the estimated regression coefficients that correspond to the mechanism of interest (e.g., $\hat{\beta}_M \hat{\gamma}$) is then computed and interpreted as an estimate of the ACME for that mechanism. The three studies of framing effects described in Section 2 all use this path analytic approach and investigate the statistical significance of the estimated ACMEs for either mechanisms (or their standardized values).

It can be shown that, under Assumption 1, the product of the unstandardized path coefficients in equations (18–20) can be justified as an estimate of the corresponding ACME. The result is proved in Appendix A2. However, it is important to note that Assumption 1 is merely an alternative representation of the same assumption, i.e., sequential ignorability given in equations (9) and (10). Thus, under Assumption 1, the same ACME can also be estimated by applying the standard causal mediation analysis to one of the mechanisms at a time. That is, under Assumption 1, the product of coefficients based on equations (6) and (7) also consistently estimates

the ACME with respect to M_i , and the same analysis can be conducted for W_i using the linear structural equations model analogous to equations (6) and (7).

Earlier, we recommended that applied researchers should check the conditional independence between M and W given T and X . If these two mediators are dependent, then it is likely that Assumption 1 is violated. Indeed, if the separate application of the standard causal mediation analysis procedure to M and W gives results that are different from those obtained by using the model specified in equations (18–20), then Assumption 1 is unlikely to hold because W is likely to affect M . A straightforward way to explore this possibility in the linear structural equations framework is to regress M on (W, T, X) and conduct an F -test with respect to W to see whether M and W are correlated even after conditioning on T and X . If one finds a statistically significant relationship, M is likely to be causally related to W , thereby violating the key identification assumption of the standard causal mediation analysis. A similar analysis should be conducted to examine the conditional association between W and T given X . We also note that these analyses should be interpreted with care: the failure to reject the null hypothesis of no conditional association does not necessarily imply the complete absence of such relationships.

In sum, from a causal inference perspective, the standard path analytic procedure for multiple mediators does not fundamentally add anything to the simpler one-mechanism-at-a-time procedure. In fact, they are based on the same exact assumption about the causal independence of multiple mediators. This implies that if one's sole goal is to identify the ACME with respect to M , there is no need to measure alternative mediators W if they are causally independent of M . Nevertheless, we suggest that researchers measure W and examine the statistical relationship between M and W . Strong statistical dependence between those mediators implies that the sequential ignorability assumption is likely to be violated and researchers need a different statistical methodology in order to conduct causal mediation analysis in the presence of causally dependent multiple mediators. Before we turn to our proposed methodology for such analysis in Section 5, we next analyze the framing experiments under the assumption of independent causal mechanisms.

4 Empirical Analysis under the Independence Assumption

We reanalyze the data from the three experiments under the assumption of independence between mechanisms using the framework of causal mediation analysis outlined by Imai et al. (2011). We also conduct a sensitivity analysis that addresses the potential violation of the sequential ignorability assumption by explicitly calculating how much the estimate of the ACME could change if the assumption is violated to a specified degree. Since the original analyses of these experiments also made the independence assumption implicitly, our sensitivity analysis here serves as a check on whether the original conclusions are robust to unobserved pretreatment confounders. Unlike the one proposed later in this article, however, the limitation of this existing sensitivity analysis is that it only allows for the possible existence of unobserved pretreatment confounders and assumes no posttreatment confounder. In other words, the sensitivity analysis maintains the assumption of independent causal mechanisms and relaxes the exogeneity of the mediator by introducing a certain degree of pretreatment confounding.

Below, we apply this procedure to the three framing experiments. We find that their original conclusions are largely (though less conclusively for some of the studies) valid and robust to unobserved confounders, *so long as we maintain the independence assumption*. Unfortunately, we also find evidence suggesting that alternative causal mechanisms are causally dependent on each other, possibly invalidating the original conclusions. Thus, the analyses in this section provide empirical examples where the independence assumption is probably violated. We also speculate that such violation is likely to be prevalent in many other similar studies of survey experiments.

4.1 Druckman and Nelson's Campaign Finance Reform Experiment

In their analysis, Druckman and Nelson find that the framing effect is mediated by the importance mechanism but not by the belief content mechanism. For the group of subjects who were not allowed to discuss the issue with other subjects, “the frame shaped the belief importance ratings,

which in turn substantially affected overall opinions,” whereas “the frames had minimal impact on the content measures and even when they did, [...] this effect did not carry through to overall opinions” (p. 737). However, even under the assumption of independent mechanisms, a potential problem is that the estimated mediation effects may be biased due to unobserved pretreatment confounding between the mediator and the outcome variable. For example, participants with libertarian ideology may think freedom of speech is more important than nonlibertarian participants, and at the same time, they may also be more opposed to campaign finance reform. If the available pretreatment covariates fail to adjust for libertarianism, the estimated causal mediation effect may be upward biased.

4.1.1 Estimation of Causal Mediation Effects

To address this concern about confounding, we apply the framework of Imai et al. (2011) and estimate ACME and examine its robustness to the violation of sequential ignorability due to an unobserved pretreatment confounder of the mediator and the outcome, as explained in Section 3.1.⁵ Here, we focus on the no-discussion group and the causal mechanism corresponding to the perceived importance of freedom of speech (hereafter the “free speech importance” mechanism), which the original study found the most significant. As shown above, ignoring the other mechanisms should not affect the result under Assumption 1. The results are presented in the first column of Fig. 2. The top figure presents the estimated ACME under sequential ignorability along with its 90% confidence interval. In addition to the overall estimate shown at the top, we also estimate the ACMEs for the treatment and the control conditions separately in the plot in order to allow for the possibility that the ACME may differ depending on the baseline treatment status.

We find that the frame difference affected the participants’ support for the campaign finance reform by approximately 0.286 points on the seven-point scale via the free speech importance mechanism, with a 90% confidence interval of (0.025, 0.625). Since the average total causal effect of the frame difference is estimated to be 0.969 points, it suggests that about 28.6% of the total effect was transmitted through changes in the perceived importance of free speech. The ACME, however, appears to slightly differ depending on the baseline value of the treatment, as the estimate for the treatment condition (0.197) is closer to zero than the estimate under the control condition (0.375). In fact, the 90% confidence interval for the former overlaps with zero (−0.019, 0.497), whereas the interval for the latter does not (0.035, 0.819). Overall, though, the result confirms the original finding that the free speech importance mechanism significantly mediates the framing effect in the theoretically expected direction, although the estimation uncertainty is relatively large.

4.1.2 Sensitivity Analysis

The remaining two plots show the results of the sensitivity analysis with respect to two alternative sensitivity parameters. First, we calculate the estimated ACME as a function of the parameter ρ , the correlation between the error terms in the mediator and the outcome models. A large value of ρ indicates the existence of strong confounding between the mediator and the outcome, and thus a serious violation of the sequential ignorability assumption. The result suggests that the ACME equals zero when ρ is below −0.43, indicating a moderate degree of robustness when compared to other similar studies (see Imai et al. 2011). The lower bound of the 90% confidence band, however, immediately crosses the zero line once we allow a slight negative correlation between the errors. Thus, a larger sample size may be needed in order to establish the robustness of the original findings to the possible existence of an unobserved pretreatment confounder that affects the mediator and the outcome in different directions (e.g., libertarianism).

⁵We also include a set of observed pretreatment covariates in the model in order to make the sequential ignorability assumption as plausible as the data permit. These covariates are year in college, age, gender, ethnicity, level of political knowledge, tendency for online processing, partisanship, and left–right ideology.

Finally, the bottom plot shows the estimated true ACME as contour lines with respect to the \tilde{R}_M^2 and \tilde{R}_Y^2 parameters, the proportions of the total variance in the mediator and the outcome variables, respectively, which would be explained by an unobserved pretreatment confounder. The contours correspond to the scenario that the unobserved confounder affects the mediator and the outcome in opposite directions (i.e., ρ is negative), as it is the only case where the estimated ACME can become negative. The result shows that the ACME can be estimated negative if the product of the two parameters are greater than 0.078. For example, the estimated ACME will be exactly zero, if the unmeasured libertarianism explains 37% of the variation in the perceived importance of freedom of speech and 21% of the variation in the campaign finance reform opinions.

4.1.3 Discussion

The above results indicate that Druckman and Nelson's original conclusion about the free speech importance mechanism is largely valid under the assumption of independent causal mechanisms. The perceived importance of freedom of speech seems to mediate the framing effect, and the estimate is reasonably robust to the violation of the sequential ignorability assumption once we adjust for a set of pretreatment covariates, including partisanship and standard left–right ideology. However, we emphasize that these results were obtained under the assumption that the free speech importance mechanism operates independently of other mechanisms, including the one represented by the participants' belief about the impact of the campaign finance reform on freedom of speech (hereafter the “free speech belief” mechanism). The assumption would be violated if, for example, the change in the content of the participant's belief about the reform due to framing differences then caused any changes in their perceived importance of free speech. Indeed, this is of major concern because, as Miller (2007) points out on the basis of her experimental study, “individuals use information obtained from the media to evaluate how important issues are” (p. 711) and “when media exposure to an issue causes negative emotional reactions about the issue, increased importance judgments will follow” (p. 712).

The data in fact underline the concern. By regressing the free speech importance mediator on the belief content mediator, the treatment, and the other covariates, we find that the coefficient of the belief mediator is negative and significantly different from zero at the 0.1 level (−0.23, with a p -value of 0.093). Moreover, regressing the belief content mediator on the treatment and the same set of pretreatment covariates yields a statistically significant coefficient (0.61, with a p -value of 0.086), as found in the original study. As discussed earlier, these analyses suggest that there may exist an additional causal pathway linking the treatment to the free speech belief mechanism and then to the importance mechanism. Such a dependent mediator lying between the treatment and the primary mediator of interest can be seen as a *posttreatment confounder*, whose existence, whether observed or unobserved, can cause a substantial bias in the estimate of ACME and invalidate the above results (see also the Appendix of Imai et al. [2011]).

4.2 Slothuus' Social Welfare Reform Experiment

Contrary to Druckman and Nelson (2003), Slothuus (2008) finds both importance and content mechanisms to be at work and concludes that “both the importance change process and the content change process mediate the framing effects” (p. 18). Like Druckman and Nelson, Slothuus relies on a path analytic method to estimate the mediation effects. We therefore estimate ACME for the importance mechanism and evaluate its robustness to unobserved pretreatment confounding between the mediator and the outcome variable under the assumption of independent causal mechanisms.⁶ For simplicity, we focus on one of the mediators used in the original analysis (the “incentive to work” importance), which was found to be the most statistically significant.

⁶Again, we add a set of observed pretreatment covariates to the model to make the sequential ignorability assumption as plausible as possible. The covariates are gender, education, level of political interest, self-placement on a left–right ideology scale, school, year of birth, political knowledge, and extremity of political values.

4.2.1 Estimation of Causal Mediation Effects

The results are shown in the middle column of Fig. 2. The top panel shows that the ACME for the incentive-to-work importance mechanism is estimated to be about 0.230 on a seven-point scale, with a 90% confidence interval of (0.082, 0.402). This represents 21.3% of the total framing effect, which is estimated to be 1.064 points (0.640, 1.490). Unlike the Druckman and Nelson study, this proportion does not appear to vary depending on the baseline treatment frame: the estimated ACME is largely similar for the treatment [0.205 (0.052, 0.400)] and control [0.255 (0.092, 0.445)] conditions. Overall, the result confirms the original finding that the importance mechanism significantly mediates the framing effect for the social welfare reform.

4.2.2 Sensitivity Analysis

How robust is this conclusion to the possible existence of unobserved pretreatment confounding? The middle panel shows that the ACME for the importance mechanism equals zero, when the error correlation between the mediator and the outcome models becomes >0.37 . This indicates a degree of robustness slightly less than that of the Druckman and Nelson study. However, the estimated ACME has smaller estimation uncertainty and hence the lower confidence bound does not cross zero until the ρ parameter becomes >0.20 . The analysis with respect to the \tilde{R}^2 parameters leads to similar conclusions as shown in the bottom panel. The estimated ACME becomes negative when the product of \tilde{R}_M^2 and \tilde{R}_Y^2 is >0.05 , which again indicates a slightly greater sensitivity than the result of the Druckman and Nelson study. For example, the estimate equals zero if an unobserved pretreatment confounder explains 25% of the variation in the perceived importance of work incentives and 20% of the variation in the welfare reform opinion.

4.2.3 Discussion

The Slothuus study is subject to the same potential problem as the Druckman and Nelson study because both are based on the assumption that alternative causal mechanisms are independent of one another. For example, the change in the content of participants' considerations about the welfare reform may not only directly affect their opinions but also influence the perceived importance of work incentives, thereby also affecting the outcome indirectly through further changes in the importance mediator. If this were to be the case, there exists a causal arrow linking the content mediator to the importance mediator, violating the independence assumption. Indeed, regressing the importance mediator on the content mediator and the treatment (as well as the set of pretreatment covariates), we find a large positive and statistically significant coefficient on the content mediator (0.350, with a p -value of 0.000). Moreover, a regression of the content mediator on the treatment and the pretreatment covariates reveals that the effect of the treatment on the content mediator is positive and close to statistical significance (0.052, with a p -value of 0.120), which raises a legitimate concern about the existence of a confounding causal pathway. Thus, similar to the Druckman and Nelson study, the possibility of bias due to posttreatment confounding is an important issue.

4.3 Brader, Valentino, and Suhay's Immigration Experiment

The original analysis of Brader, Valentino, and Suhay (2008) reveals that the combination of Latino cues and negative framing changed the participant's opinions and behavior in the anti-immigration direction. That is, the participants who read the Latino article emphasizing the negative consequences of immigration became more opposed to increased immigration than the rest of the participants. More importantly, the authors find that the frame affected the outcome variables through the anxiety mechanism rather than the perceived harm mechanism.⁷ Like the other

⁷In their own words, "The conjunction of Latino cues and negative news about immigration influenced levels of anxiety. Anxiety then caused shifts in policy attitudes, information seeking, and political action" (p. 969). In contrast, "belief

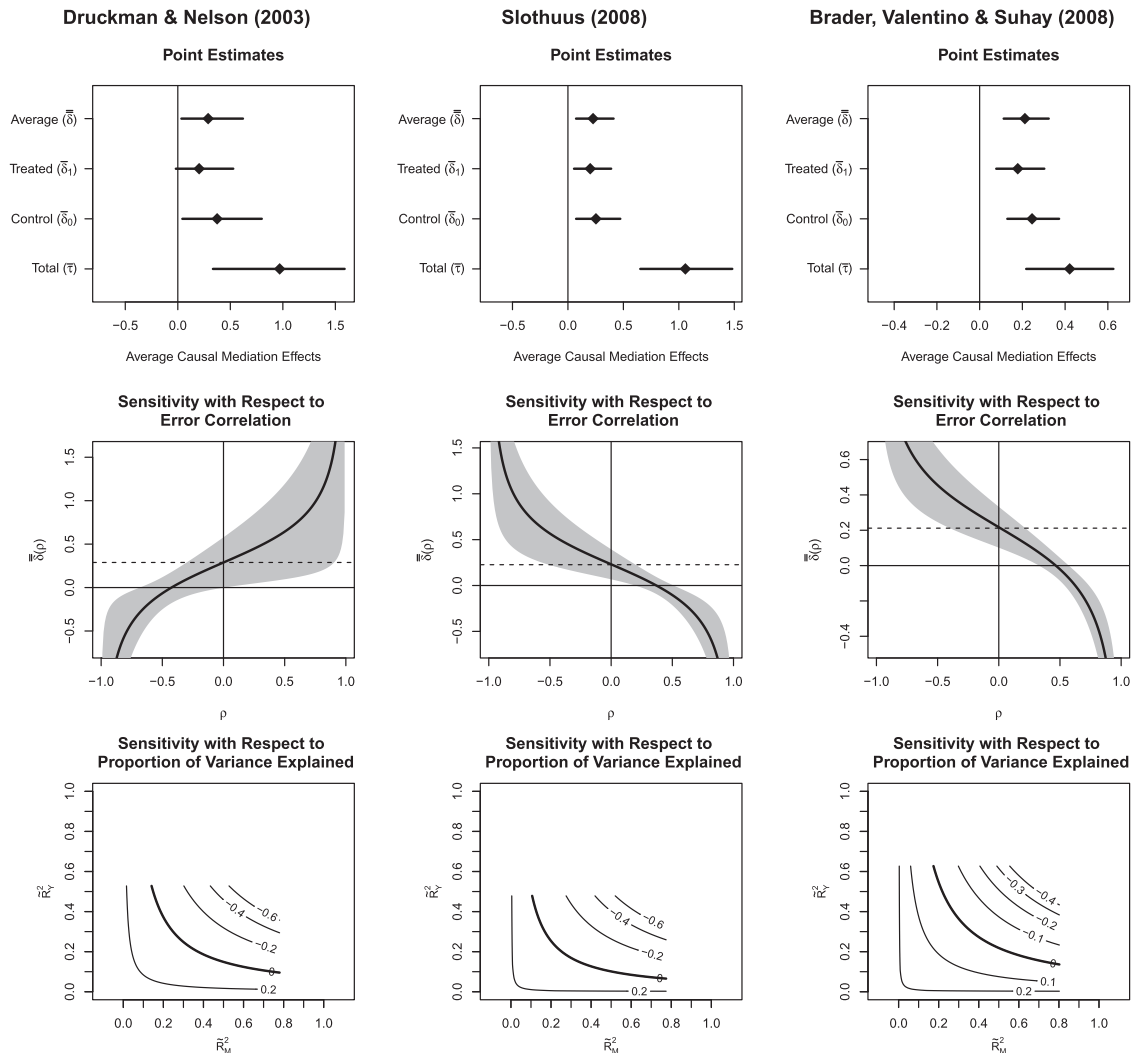


Fig. 2 Estimation and sensitivity analysis under the assumption of independent causal mechanisms. The top panels present the estimated ACMEs under the sequential ignorability assumption (Assumption 1) along with their 90% confidence intervals for each of the three studies indicated at the top. The panels in the middle row show the estimated true values of ACMEs as functions of the sensitivity parameter ρ , which represents the correlation between the error terms in the mediator and the outcome models. The thick lines and gray bands represent the point estimates of the ACME and their 90% confidence intervals, respectively. The bottom panels show the same sensitivity analyses, except that the ACME estimates are plotted against $(\hat{R}_Y^2, \hat{R}_M^2)$, the proportions of the total variance in the outcome and mediator variables, respectively, that would be explained by a hypothetical unobserved pretreatment confounder. Overall, the results suggest that under the assumption of independence between mechanisms, the causal mediation effects are positive and moderately statistically significant in all of the three studies, and the estimates are fairly robust to the possible unobserved pretreatment mediator–outcome confounding to varying degrees.

two studies, Brader, Valentino, and Suhay rely on the structural equation modeling approach of Baron and Kenny (1986). However, the validity of this causal mediation analysis crucially hinges on the sequential ignorability assumption. The estimated effects will be biased if, for example, unmeasured job skills of the participants affect both their levels of anxiety and opinions about increased

about the severity of the immigration problem does not mediate the interactive effect of ethnic cues and news emphasis” (p. 969).

immigration. This is likely if low-skilled workers feel more anxious about immigrants and are also more anti-immigrants than high-skill participants.

4.3.1 Estimation of Causal Mediation Effects

We estimate the ACME for the anxiety mechanism by focusing on one of the outcome variables in the original study (whether immigration to the United States should be increased or decreased) and examine how much of the total effect of the negative Latino frame on the variable operated through the anxiety mediator. The point estimates are shown in the top panel of the left column in Fig. 2 along with their 90% confidence intervals. The result supports the original conclusion, indicating that the negative Latino frame made the participants 0.216 points more opposed to immigration on the five-point scale via the anxiety mechanism, with a 90% confidence interval of (0.120, 0.329). This represents roughly half of the estimated total framing effect, which is 0.423 points. The estimate varies slightly across the treatment values [0.184 with (0.083, 0.311) for the treated; 0.247 with (0.136, 0.360) for the control].

4.3.2 Sensitivity Analysis

The middle and the bottom plots show the result of sensitivity analysis. According to this analysis, the ACME is estimated to be negative when the correlation between the error terms in the mediator and the outcome models is larger than 0.47, and the ACME is statistically indistinguishable from zero at the 90% level when the parameter ρ is >0.33 . Thus, the result of this study is slightly more robust to the sequential ignorability violation than those of the above two studies. Finally, in terms of the coefficient of determination parameters, the product of \tilde{R}_M^2 and \tilde{R}_Y^2 must be at least as large as 0.08, implying that an unobserved pretreatment confounder must explain 20% of the variation in the participants' anxiety and 40% of the variation in their immigration opinions, for example. These results confirm the original finding that the anxiety mechanism plays a major role in the framing effect on opinions toward increased immigration.

4.3.3 Discussion

As in the previous two studies, the above analysis rests on the key assumption that the anxiety mechanism is independent of other mechanisms underlying the framing effect. In particular, the participants' conscious belief about the negative impact of increased immigration—the other mechanism explicitly studied in the original article—is assumed to have no impact on their levels of anxiety. This assumption may not be entirely plausible because the increased level of perceived harm of immigration due to the negative Latino framing can cause the participants to feel more anxious about increase in immigration (Isbell and Ottati 2002). Indeed, there is a strong statistical dependence between the two mediators. Regressing the anxiety mediator on the perceived harm mediator as well as the treatment and the pretreatment covariates, we find that the estimated coefficient of the perceived harm mediator is positive and large (1.016, with a p -value of 0.000). Furthermore, regressing the perceived harm mediator on the treatment and the covariates yields a positive and significant estimate of the treatment effect on the perceived harm mediator (0.436, with a p -value of 0.070). Thus, like in the above two examples, we must address the issue of the posttreatment confounding between the mediator and the outcome variable in order to evaluate the empirical findings of this study.

5 Statistical Analysis of Causally Related Multiple Mechanisms

In this section, we consider statistical analysis of causally related multiple mechanisms. In particular, we focus on the situation depicted in Fig. 1(b) where other mediators W confound the relationship between the mediator of interest M and the outcome Y . We assume the exogeneity of the mediator M conditional on the pretreatment covariates X , the treatment T , and the posttreatment confounders W . Unlike in Section 3, we must measure alternative mediators W when allowing for

the causal relationship between M and W (see Section 7 for the designs that do not require causally related multiple mediators to be measured). In this setting, we first review the identification result of Robins (2003) that the ACMEs (represented by red arrows in the figure) can be nonparametrically identified under an additional assumption of no interaction between the treatment T and the mediator M .

However, as noted by many researchers, including Robins (2003) himself (e.g. Petersen, Sinisi, and van der Laan 2006; Imai, Tingley, and Yamamoto 2013), this assumption is unrealistic in many applications. In particular, the assumption must hold not just on average but rather for every observation. To overcome this limitation, we propose a new sensitivity analysis, which can be used to examine how empirical results change as we gradually relax the no-interaction assumption. Such an analysis reveals how robust one's empirical results are to the potential violation of the key identification assumption. We develop this methodology in the context of a fairly general varying-coefficient linear regression model where each coefficient can vary arbitrarily across observations. We then apply the proposed method to the framing experiments introduced earlier.

5.1 The Setup and Assumptions

We use the framework of causal mediation analysis and its associated notation introduced in Sections 3.1 and 3.2. Recall that the key difference between Figs. 1(a) and (b) is that we allow the mediator of interest M to be causally affected by a set of alternative mediators W . This means that the potential values of M are now a function of W . That is, we use $M_i(t, w)$ to denote the potential value of the mediator of interest for unit i when the treatment status is t and the alternative mediators W take the value of w . Then, the observed value of the mediator for this unit is given by $M_i = M_i(T_i, W_i(T_i))$.

Under this setting, for each unit, we define the causal mediation (with respect to M) and direct effects as

$$\delta_i(t) \equiv Y_i(t, M_i(1, W_i(1)), W_i(t)) - Y_i(t, M_i(0, W_i(0)), W_i(t)) \quad (21)$$

$$\zeta_i(t) \equiv Y_i(1, M_i(t, W_i(t)), W_i(1)) - Y_i(0, M_i(t, W_i(t)), W_i(0)) \quad (22)$$

for $t=0, 1$, where $\delta_i(t)$ corresponds to the causal effect of the treatment on the outcome that transmits through the mediator of interest M (i.e., the red arrows in Fig. 1(b)). In the framing experiment of Druckman and Nelson, for example, this represents the portion of the framing effect due to the change in issue importance induced by the framing manipulation, while the belief content is held constant at the value that would be naturally observed when one of the issue frames is given. On the other hand, $\zeta_i(t)$ represents the rest of the treatment effect (denoted by the black arrow at the bottom of the figure and the combination of the red and black arrows that go from T to Y through W but not through M). In Druckman and Nelson's experiment, this represents the fraction of the framing effect that does not go through the issue importance mechanism, regardless of whether it gets transmitted through the belief content mechanism or through other unspecified mechanisms. Notice that these two effects represent the quantities identical to those in equations (1) and (2); the only difference is that the expressions in equations (21) and (22) make the existence of the alternative mediators W explicit, whereas equations (1) and (2) do not. Thus, as expected, the sum of these two effects equals the total treatment effect,

$$\tau_i \equiv Y_i(1, M_i(1, W_i(1)), W_i(1)) - Y_i(0, M_i(0, W_i(0)), W_i(0)) = \delta_i(t) + \zeta_i(1 - t), \quad (23)$$

for $t=0, 1$. Again, we are interested in estimating the ACME, i.e., $\bar{\delta}(t) \equiv \mathbb{E}(\delta_i(t))$, and the average direct and total effects can also be defined in an analogous manner, i.e., $\bar{\zeta}(t) \equiv \mathbb{E}(\zeta_i(t))$ and $\bar{\tau} \equiv \mathbb{E}(\tau_i)$.

What assumptions do we need to make in order to identify the ACME in this scenario? We modify Assumption 1 and consider the following weaker version of the sequential ignorability assumption,

Assumption 2 (Sequential Ignorability with Multiple Causally Dependent Mediators). We assume that the following three conditional independence statements hold:

$$\{Y_i(t, m, w), M_i(t, w), W_i(t)\} \perp\!\!\!\perp T_i \mid X_i = x \quad (24)$$

$$\{Y_i(t, m, w), M_i(t, w)\} \perp\!\!\!\perp W_i \mid T_i = t, X_i = x \quad (25)$$

$$\{Y_i(t, m, w)\} \perp\!\!\!\perp M_i \mid W_i(t) = w, T_i = t, X_i = x, \quad (26)$$

for any t, m, w, x .

This assumption corresponds to what Robins (1986, 2003) called the FRCISTG (fully randomized causally interpretable structural tree graph) model. Assumption 2 is similar to Assumption 1 in that exogeneity is assumed for the treatment T , the alternative mediators W , and the mediator of interest M .

However, Assumption 2 relaxes Assumption 1 in two important ways. First, the mediator of interest M is assumed to be exogenous after conditioning on alternative mediators W as well as the treatment T and the pretreatment confounders X , whereas Assumption 1 does not permit conditioning on the posttreatment confounders. In the context of Druckman and Nelson's framing experiment, this implies that Assumption 2 allows for the possibility that the perceived issue importance is affected by the content of the belief about the issue, whereas Assumption 1 rules out the existence of such causal dependence.

Second, Assumption 2 avoids specifying the conditional independence relationship between the potential outcome under the treatment status t and the potential value of mediator under the opposite treatment status t' . For example, contrast equation (16) with equation (26). The former assumes the conditional independence between $Y_i(t', m, W_i(t'))$ and $M_i(t)$ even when $t \neq t'$, whereas the latter only applies the conditional independence assumption to the relationship between $Y_i(t, m, w)$ and $M_i(t)$. Some scholars (e.g., Robins and Richardson 2010) consider this distinction to be important because one can conceive of a randomized experiment on a separate exchangeable population with which Assumption 2 can be tested, while such an experiment does not exist for Assumption 1 (Section 7) even as a purely theoretical possibility (Robins 2003). This implies that Assumption 2 can be empirically verified from observed data at least in theory, whereas Assumption 1 cannot.

Unfortunately, in the presence of causally dependent multiple mediators, Assumption 2 is not sufficient for the identification of the ACME. Robins (2003) shows that under this setting the ACMEs are nonparametrically identifiable if the following assumption of no treatment–mediator interaction effect holds:

Assumption 3 (No Interaction Between Treatment and Mediator). For every unit i , we assume the following equality,

$$Y_i(1, m, W_i(1)) - Y_i(0, m, W_i(0)) = Y_i(1, m', W_i(1)) - Y_i(0, m', W_i(0)), \quad (27)$$

for any m, m' .

The problem of this assumption is that it is unlikely to be credible in most applications because it must hold for every unit. In Druckman and Nelson's experiment, for example, Assumption 3 implies that for every subject, the causal effect of the perceived issue importance on opinions must be constant regardless of whether the subject read the positive or negative article. To overcome this limitation, we now introduce our proposed methodology that allows one to relax this no-interaction assumption.

5.2 The Proposed Methodology

Given the above setup, we show how to relax the no-interaction assumption (Assumption 3) while maintaining the exogeneity of treatment and mediator (Assumption 2). We consider the following linear structural equation model with varying coefficients,

$$M_i(t, w) = \alpha_2 + \beta_{2i}t + \xi_{2i}^\top w + \mu_{2i}^\top tw + \lambda_{2i}^\top x + \varepsilon_{2i}, \quad (28)$$

$$Y_i(t, m, w) = \alpha_3 + \beta_{3i}t + \gamma_i m + \kappa_i tm + \xi_{3i}^\top w + \mu_{3i}^\top tw + \lambda_{3i}^\top x + \varepsilon_{3i}, \quad (29)$$

where $\mathbb{E}(\varepsilon_{2i}) = \mathbb{E}(\varepsilon_{3i}) = 0$ is assumed without the loss of generality.⁸ The model generalizes the linear structural equation model commonly used by applied researchers (see equations (18) and (20)) in several important ways. First, the model reflects the particular situation depicted in Fig. 1(b) and discussed above which permits the presence of causally dependent multiple mediators. Specifically, the mediator of interest M is allowed to depend on a set of alternative mediators W , which themselves are possibly affected by the treatment, as well as the treatment variable T . Similarly, the outcome variable Y depends on W as well as M and T . Second, each coefficient is allowed to vary across individual observations in an arbitrary manner, allowing for a wide range of patterns of heterogeneous treatment effects. This is a crucial advantage over the traditional structural equation modeling framework, which typically assumes the unit homogeneity of treatment effects. Finally, we include the interaction between the treatment and each of the mediators (both M and W) so that mediation effects can vary depending on the baseline treatment status.

While the model is semiparametric and hence quite flexible, it imposes a structure by assuming additivity. In particular, the model assumes no interaction between the two mediators. While it is possible to allow for the existence of the interaction between M and W , the resulting sensitivity analysis would include additional sensitivity parameters and thus become more complex. We thus focus on the simpler model in equation (29).

How is this model related to the standard linear structural equation model? We decompose each of the varying coefficients into the mean and the deviation from it,

$$M_i(t, w) = \alpha_2 + \beta_2 t + \xi_2^\top w + \mu_2^\top t w + \lambda_2^\top x + \eta_{2i}(t, w), \quad (30)$$

$$Y_i(t, m, w) = \alpha_3 + \beta_3 t + \gamma m + \kappa t m + \xi_3^\top w + \mu_3^\top t w + \lambda_3^\top x + \eta_{3i}(t, m, w), \quad (31)$$

where $\beta_2 \equiv \mathbb{E}(\beta_{2i})$, $\beta_3 \equiv \mathbb{E}(\beta_{3i})$, $\gamma \equiv \mathbb{E}(\gamma_i)$, $\kappa \equiv \mathbb{E}(\kappa_i)$, $\xi_2 \equiv \mathbb{E}(\xi_{2i})$, $\xi_3 \equiv \mathbb{E}(\xi_{3i})$, $\mu_2 \equiv \mathbb{E}(\mu_{2i})$, $\mu_3 \equiv \mathbb{E}(\mu_{3i})$, $\lambda_2 \equiv \mathbb{E}(\lambda_{2i})$, and $\lambda_3 \equiv \mathbb{E}(\lambda_{3i})$ are the mean parameters of corresponding varying coefficients. The new error terms are given by

$$\eta_{2i}(t, w) = \tilde{\beta}_{2i} t + \tilde{\xi}_{2i}^\top w + \tilde{\mu}_{2i}^\top t w + \tilde{\lambda}_{2i}^\top x + \varepsilon_{2i} \quad (32)$$

$$\eta_{3i}(t, m, w) = \tilde{\beta}_{3i} t + \tilde{\gamma}_i m + \tilde{\kappa}_i t m + \tilde{\xi}_{3i}^\top w + \tilde{\mu}_{3i}^\top t w + \tilde{\lambda}_{3i}^\top x + \varepsilon_{3i}, \quad (33)$$

where by construction we have $\mathbb{E}(\tilde{\beta}_{2i}) = \mathbb{E}(\tilde{\beta}_{3i}) = \mathbb{E}(\tilde{\xi}_{2i}) = \mathbb{E}(\tilde{\xi}_{3i}) = \mathbb{E}(\tilde{\mu}_{2i}) = \mathbb{E}(\tilde{\mu}_{3i}) = \mathbb{E}(\tilde{\lambda}_{2i}) = \mathbb{E}(\tilde{\lambda}_{3i}) = \mathbb{E}(\tilde{\gamma}_i) = \mathbb{E}(\tilde{\kappa}_i) = 0$ and hence $\mathbb{E}(\eta_{2i}(t, w)) = \mathbb{E}(\eta_{3i}(t, m, w)) = 0$. Since Assumption 2 implies the following exogeneity conditions, $\mathbb{E}(\varepsilon_{2i}|X_i, T_i, W_i) = \mathbb{E}(\varepsilon_{3i}|T_i, W_i, M_i) = 0$, it follows that the exogeneity condition also holds for the new error terms, i.e., $\mathbb{E}(\eta_{2i}(T_i, W_i)|X_i, T_i, W_i) = \mathbb{E}(\eta_{3i}(T_i, M_i, W_i)|X_i, T_i, W_i, M_i) = 0$. Thus, the coefficients in equations (30) and (31) can be estimated without bias under Assumption 2.

We show that the ACMEs are identified if we fix two unobserved quantities, which we use as sensitivity parameters.⁹ The first parameter is the correlation between the mediator of interest $M_i(t)$ and the individual-level treatment–mediator interaction effect κ_i , i.e., $\rho_t = \text{Corr}(M_i(t), \kappa_i)$. This parameter represents the direction of the interaction effect. The second parameter is the standard deviation (SD) of the individual-level coefficient for the treatment–mediator interaction, i.e., $\sigma = \sqrt{\mathbb{V}(\kappa_i)}$, representing the degree of heterogeneity in the treatment–mediator interaction effect. In Appendix A3, we prove that the ACMEs and direct effects can be written as a function of the identifiable model parameters (under Assumption 2 but without requiring Assumption 3) and the two sensitivity parameters for $t=0, 1$,

$$\bar{\delta}(t) = \bar{\tau} - \bar{\xi}(1 - t) \quad (34)$$

⁸This model encompasses the model considered by Glynn (2012) as a special case.

⁹Albert and Nelson (2011) assume a stronger version of exogeneity that is by itself sufficient to identify the ACMEs (e.g., the sequential ignorability of Imai, Keele, and Yamamoto [2010c]) in order to consider the estimation of other path-specific effects. Tchetgen Tchetgen and Shpitser (2011) propose a semiparametric sensitivity analysis for the possible existence of unmeasured pre- and posttreatment confounders under Assumption 1 rather than the sensitivity analysis for the possible interaction under Assumption 2.

$$\bar{\zeta}(t) = \beta_3 + \kappa \mathbb{E}(M_i | T_i = t) + \rho_t \sigma \sqrt{\mathbb{V}(M_i | T_i = t)} + (\xi_3 + \mu_3)^\top \mathbb{E}(W_i | T_i = 1) - \xi_3^\top \mathbb{E}(W_i | T_i = 0). \quad (35)$$

Thus, under the situation depicted in Fig. 1(b) with Assumption 2, we can conduct a sensitivity analysis even in the presence of posttreatment confounders to examine how the estimated ACME changes as a function of the two parameters, ρ_t and σ . Several remarks are in order. First, the no-interaction assumption given in equation (27) implies that $\kappa_i = 0$ and hence $\kappa = \sigma = \rho_t = 0$. Thus, under the model considered here, the ACME and the ADE are identified as $\bar{\delta}(t) = \bar{\tau} - \beta_3 - (\xi_3 + \mu_3)^\top \mathbb{E}(W_i | T_i = 1) + \xi_3^\top \mathbb{E}(W_i | T_i = 0)$ and $\bar{\zeta}(t) = \beta_3 + (\xi_3 + \mu_3)^\top \mathbb{E}(W_i | T_i = 1) - \xi_3^\top \mathbb{E}(W_i | T_i = 0)$. These expressions correspond exactly to the procedure used by applied researchers who rely on a linear regression to model the conditional expectation of W_i given T_i (e.g., Taylor, MacKinnon, and Tein 2008). While these researchers appear to be unaware of the essential role of the no-interaction assumption, the sensitivity analysis developed here can formally assess the robustness of empirical results to the potential violation of this key identifying assumption.

Second, we can relax the no-interaction assumption of equation (27) to some extent under our linear structural equation framework by considering instead of following the homogeneous interaction effect assumption,

$$Y_i(1, m, W_i(1)) - Y_i(0, m, W_i(0)) = B_i + Cm, \quad (36)$$

for any m . Unlike the no-interaction assumption, this assumption allows for the treatment–mediator interaction in a way that is common to all observations. This homogeneous interaction effect assumption implies $\sigma = 0$ and thus we can identify the ACME and ADE whose expressions are given by $\bar{\delta}(t) = \bar{\tau} - \bar{\zeta}(1 - t)$ and $\bar{\zeta}(t) = \beta_3 + \kappa \mathbb{E}(M_i | T_i = t) + (\xi_3 + \mu_3)^\top \mathbb{E}(W_i | T_i = 1) - \xi_3^\top \mathbb{E}(W_i | T_i = 0)$, respectively.

Third, when neither of the above assumptions holds, the standard estimation procedure results in bias. For the ACME, this bias equals $-\rho_{1-t} \sigma \sqrt{\mathbb{V}(M_i | T_i = 1 - t)}$ that depends on the variance of mediator within the treatment or control group as well as the sensitivity parameters. For example, the bias is negative and large if the treatment–mediator interaction effect tends to be higher when the mediator takes a larger value, i.e., $\rho_t > 0$, and the variance of mediator and the degree of heterogeneity in these interaction effects are large.

Fourth, we note that when $\rho_t = 0$, we can identify the ACME regardless of the value of σ . However, when ρ_t is not equal to zero, we must specify both ρ_t and σ in order to estimate the ACME under Assumption 2. If the interpretation of ρ_t is difficult, we can derive the bounds on the ACME as a function of σ while allowing ρ_t to take any value between -1 and 1 .¹⁰

Fifth, for the ease of the interpretation of σ , we follow Imai, Keele, and Tingley (2010a) and Imai, Keele, and Yamamoto (2010c) and use coefficients of determination as an alternative parameterization. Specifically, we use the proportion of the unexplained or original variance of the outcome that is explained by incorporating the heterogeneity in the treatment–mediator interaction. Thus, the sensitivity parameter represents how important it would be to incorporate the interaction heterogeneity in the regression model in order to explain the variation in the outcome variable. Formally, these parameters are defined as

$$R^{2*} = \frac{\mathbb{V}(\tilde{\kappa}_i T_i M_i)}{\mathbb{V}(\eta_{3i}(T_i, M_i, W_i))} \quad \text{and} \quad \tilde{R}^2 = \frac{\mathbb{V}(\tilde{\kappa}_i T_i M_i)}{\mathbb{V}(Y_i)} \quad (37)$$

for the proportion of unexplained variance and that of the original variance explained by the heterogeneity of the treatment–mediator interaction effects, respectively. We can directly relate these quantities to the ACME via the following one-to-one relationship between σ and each of these coefficients of determination:¹¹

¹⁰The expression for such bounds is given by the following: $\bar{\tau} - \beta_3 - \kappa \mathbb{E}(M_i | T_i = 1 - t) - \sigma \sqrt{\mathbb{V}(M_i | T_i = 1 - t)} - (\xi_3 + \mu_3)^\top \mathbb{E}(W_i | T_i = 1) + \xi_3^\top \mathbb{E}(W_i | T_i = 0) \leq \bar{\delta}(t) \leq \bar{\tau} - \beta_3 - \kappa \mathbb{E}(M_i | T_i = 1 - t) + \sigma \sqrt{\mathbb{V}(M_i | T_i = 1 - t)} - (\xi_3 + \mu_3)^\top \mathbb{E}(W_i | T_i = 1) + \xi_3^\top \mathbb{E}(W_i | T_i = 0)$.

¹¹We used the following equality, $\mathbb{V}(\tilde{\kappa}_i T_i M_i) = \mathbb{E}(\mathbb{V}(\tilde{\kappa}_i T_i M_i | T_i, M_i, W_i)) + \mathbb{V}(\mathbb{E}(\tilde{\kappa}_i T_i M_i | T_i, M_i, W_i)) = \mathbb{E}(T_i M_i^2 \mathbb{V}(\tilde{\kappa}_i | T_i, M_i, W_i)) = \sigma^2 \mathbb{E}(T_i M_i^2)$, where the second equality is due to the law of total variance, and the next two equalities hold because of Assumption 2.

$$\sigma = \sqrt{\frac{\mathbb{V}(\eta_{3i}(T_i, M_i, W_i))R^{2*}}{\mathbb{E}(T_i M_i^2)}} = \sqrt{\frac{\mathbb{V}(Y_i)\tilde{R}^2}{\mathbb{E}(T_i M_i^2)}}. \quad (38)$$

This implies that σ is bounded from above by $\sqrt{\mathbb{V}(\eta_{3i}(T_i, M_i, W_i))/\mathbb{E}(T_i M_i^2)}$ because $0 \leq R^{2*} \leq 1$. Furthermore, the sensitivity to the interaction heterogeneity can be assessed by studying how the ACME varies depending on the values of R^{2*} and \tilde{R}^2 . This can also be done by calculating the ratio of σ to its upper bound.

Finally, under Assumption 2 and the model in equations (28) and (29), we can also identify another possible quantity of interest, the population average of the causal mediation effect specific to the path $T \rightarrow W \rightarrow Y$. This quantity is defined as

$$\chi_i(t) = Y_i(t, M_i(t, W_i(t)), W_i(1)) - Y_i(t, M_i(t, W_i(t)), W_i(0)), \quad (39)$$

for $t=0, 1$, and corresponds to the effect of the treatment on the outcome that goes through the posttreatment confounders W but not through the primary mediator M . In Appendix A4, we prove that $\bar{\chi}(t) \equiv \mathbb{E}(\chi_i(t))$ is given by

$$\bar{\chi}(t) = (\xi_3 + t\mu_3)\{\mathbb{E}(W_i|T_i = 1) - \mathbb{E}(W_i|T_i = 0)\}, \quad (40)$$

for $t=0, 1$. It is noteworthy that under our model, this identification result only requires Assumption 2 and does not need any assumption about the treatment–mediator interaction or sensitivity parameters. This contrasts with the result by Albert and Nelson (2011) that the path-specific effect $\bar{\chi}(t)$ cannot be identified under the set of assumptions they consider, as well as the more general nonparametric identification result about path-specific effects by Avin, Shpitser, and Pearl (2005). Indeed, it can be shown that $\bar{\chi}(t)$ becomes unidentified if we include the interaction between M and W to the model. In that case, a sensitivity analysis analogous to the one about $\bar{\delta}(t)$ described above can be conducted for $\bar{\chi}(t)$ using sensitivity parameters similar to ρ_t and σ .

6 Empirical Analysis without the Independence Assumption

We now revisit the media framing experiments introduced in Section 2. The authors of these studies implicitly assumed that the mechanisms of their primary interest were causally independent from the alternative mechanisms incorporated in the original analyses. However, as discussed in Section 4, this assumption is implausible on both theoretical and empirical grounds. We therefore use the method developed in the previous section to estimate ACMEs without assuming the independence between these mechanisms. We also apply our proposed sensitivity analysis to assess how robust these estimates are to the violation of the key assumption that the effect of the primary mediator does not depend on the value of the treatment (Assumption 3).

6.1 Druckman and Nelson's Campaign Finance Reform Experiment

In Section 4.1, we noted that in Druckman and Nelson's framing experiment, the participants' belief about the effect of campaign finance reform may have influenced their perceived importance of free speech. We thus reanalyze their data to allow for the causal dependence of the importance mechanism on the belief content mechanism. That is, we first estimate the linear structural equations models in equations (30) and (31) with the same set of pretreatment covariates as in Section 4.1, and then compute the ACME as a function of the sensitivity parameters using equations (34) and (35). The results are shown in the three plots in the left column of Fig. 3.

As in Section 4, the top panel presents the estimated ACMEs (overall, treatment, and control) with the 90% confidence intervals, but this time under different assumptions (Assumptions 2 and equation (36)). The average total effect and its 90% confidence intervals that are essentially identical to those in Fig. 2 are also shown at the bottom. Unlike what we found under Assumption 1, the estimated overall ACME is statistically indistinguishable from zero, although it is similar in magnitude [0.313 with a confidence interval of (−0.021, 0.648)] and represents about 32.2% of the

total framing effect. The ACME does not vary as much according to the baseline treatment status, with the estimate being statistically insignificant in either case [0.265 (−0.077, 0.606) for the treatment condition; 0.332 (−0.092, 0.755) for the control]. Thus, the results give only modest support to the original conclusion once the assumed causal independence between the mechanisms is relaxed.

This analysis relaxes the independence assumption but still assumes that the interaction between framing effects and the effects of issue importance is homogeneous across participants (equation (36)). Since this assumption is rather strong, we examine the robustness of our conclusions to its violation via the proposed sensitivity analysis. The remaining two panels in the left column of Fig. 3 show the results. In the first panel, the sharp bounds on the true values of the overall ACME are plotted as functions of the sensitivity parameter σ (thick solid lines), which represents the degree of heterogeneity in the interaction between the issue frames and perceived importance. As discussed in Section 5, observed data imply an upper bound on the possible values of this parameter, which equals the rightmost value of σ in the figure (0.382) and represents the maximal possible violation of equation (36) given in the data. The 90% confidence intervals are computed based on the approach of Imbens and Manski (2004) with the bootstrap standard errors (SEs) and are represented by the gray region around the bounds. The point-identified value of ACME under equation (36) is represented by the dashed horizontal line.

The result shows that the lower bound of ACME does not become negative until σ becomes > 0.195 , or 51% of its largest possible value. This implies that, disregarding the statistical uncertainty in these estimates, the data from Druckman and Nelson's experiment provide some support for the importance mechanism even if we allow for a certain degree of violation of the no-interaction assumption. When we completely relax the no-interaction assumption and only make the sequential ignorability assumption (Assumption 2), however, the bounds become wide (−0.303, 0.922), with a 90% confidence interval of (−0.618, 1.159).

The bottom figure presents the result of the same sensitivity analysis using an alternative parameterization. As discussed in Section 5, the \tilde{R}^2 parameter here represents the proportion of the total variance of the outcome variable that would be explained if we could take into account the heterogeneity of the treatment–mediator interaction in the regression model. The upper bound of \tilde{R}^2 , 0.610, corresponds to the scenario where the residual variation in the outcome variable is completely attributed to the interaction heterogeneity and thus represents the severest possible violation of the no-interaction assumption given the observed data. In this alternative formulation, the bounds on the overall ACME include zero only when \tilde{R}^2 is greater than 0.159. This implies that we can maintain the conclusion of the original analysis unless the interaction heterogeneity explains $> 15.9\%$ of the total variance of the participants' opinion. In summary, if one ignores estimation uncertainty, the conclusions of Druckman and Nelson (2003) are reasonably robust to the violations of the two assumptions made in their original analysis: the causal independence between alternative mechanisms and the no treatment–mediator interaction effect. However, the generally wide confidence intervals suggest that drawing definite conclusions would require a study with a larger sample size.

6.2 Slothuus' Social Welfare Reform Experiment

The framing experiment of Slothuus is also subject to the possibility that the participants' perceived importance of work incentives may be influenced by the content of their considerations about welfare reform. If this were the case, the importance mechanism is causally dependent on the belief content mechanism. We therefore apply our proposed method to reestimate the ACME for the importance mechanism relaxing the independence assumption. Again, we use the same set of pretreatment covariates as in Section 4.2. The results are presented in the middle column of Fig. 3.

The top panel shows that under the assumptions of sequential ignorability (Assumption 2) and homogeneous interaction (equation (36)), the estimated overall ACME for the importance mechanism is about 0.159, representing 14.1% of the total effect (1.128). The 90% confidence interval for this estimate does not contain zero (0.004, 0.315). The estimates for the treatment and control conditions are slightly different [0.102 with (−0.054, 0.257) and 0.208 with (0.004, 0.411),

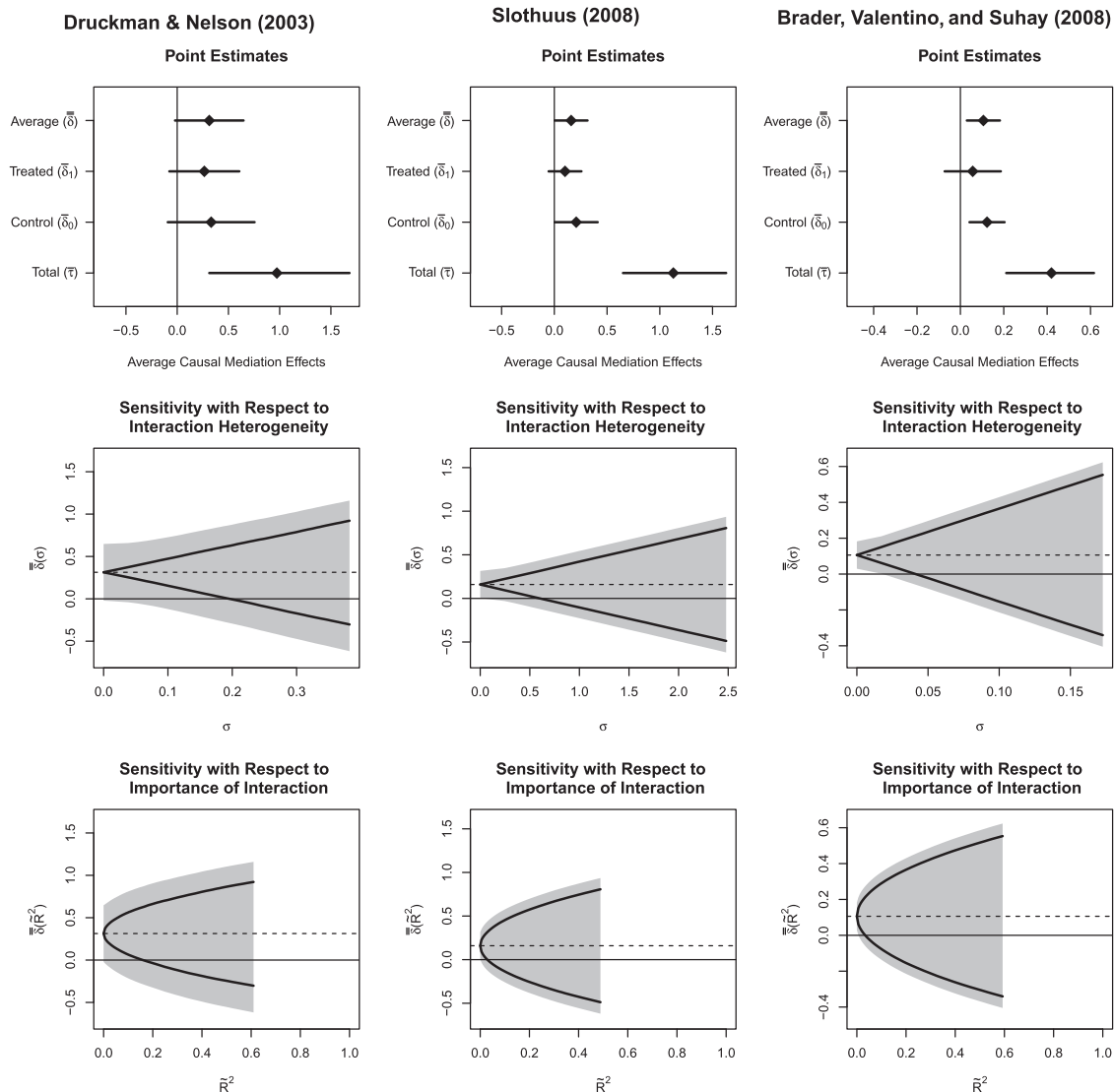


Fig. 3 Estimation and sensitivity analysis without the independence assumption. The top panels present the estimated ACMEs under the sequential ignorability and homogeneous interaction assumptions (Assumption 2 and equation (36)) with their 90% confidence intervals for the three framing studies. The panels in the middle row show the sharp bounds on the true values of ACMEs as functions of the sensitivity parameter σ , which equals the SD of the varying coefficient on the treatment-mediator interaction term and thus represents the degree of unit heterogeneity in the interaction. The bottom panels plot the same estimated ACMEs with respect to \tilde{R}^2 , the proportion of the total variance of the outcome variable that would be explained by the treatment-mediator interaction term. The results suggest that the positive mediation effects found in the original studies (Fig. 2) become smaller in magnitude and statistically either insignificant or barely significant once we allow for the causal pathway from the alternative mediator (W) to the mediator of interest (M) as in Fig. 1(b).

respectively]. These estimates are both smaller in magnitude and weaker in statistical significance than those assuming the causal independence between the importance and content mechanisms (see Section 4.2).

The other two panels show the results of the sensitivity analysis. As we gradually relax the homogeneous interaction assumption, the 90% confidence interval for the overall ACME covers zero almost immediately. The lower bound also becomes less than zero when σ is >0.607 , or 24.5% of its largest possible value implied by the data (0.987). This indicates that the result of

Slothuus's experiment is more sensitive to the heterogeneity in the treatment–mediator interaction than Druckman and Nelson's result, despite the statistical significance of the original point estimate. The result translates to the value of 0.029 in terms of the alternative \tilde{R}^2 parameter, suggesting that the interaction heterogeneity must only explain 2.9% of the total variation in the participants' opinion (out of the maximum value of 48.9%), so that the bounds on the overall ACME for the importance mechanism contain zero. In summary, once we allow for the dependence among mechanisms, the original conclusions from Slothuus's framing experiment are quite sensitive to the violation of no interaction even under the exogeneity assumptions.

6.3 Brader, Valentino, and Suhay's Immigration Experiment

Finally, we apply our proposed framework to the experiment of Brader, Valentino, and Suhay. Like the above two experiments, the assumption of independence between the anxiety and perceived harm mechanisms (Assumption 1) is problematic in Brader et al.'s study from both theoretical and empirical perspectives. The right column of Fig. 3 shows the results of our analysis using the same pretreatment covariates as in the original study, but accommodating the dependence of anxiety on perceived harm.

Unlike the other two examples, the overall ACME for the anxiety mechanism remains statistically significant at the 0.1 level under Assumption 2 and equation (36) (0.030, 0.182). However, when compared to the estimate under the independence assumption, the estimated overall ACME is substantially smaller (0.106) and represents only 25.3% of the total framing effect (0.420). Moreover, the estimated ACME for the treatment condition is even smaller (0.057) and statistically insignificant (−0.073, 0.187), whereas the estimate for the control condition is significant [0.123 (0.042, 0.204)]. The evidence for the anxiety mechanism thus appears much weaker when we allow the participants' perceived harm to influence their anxiety levels.

This conclusion is reinforced when we examine the sensitivity of these estimates to the violation of the homogeneous interaction effect assumption (equation (36)). The middle panel shows that the lower bound of the ACME becomes less than zero when the SD of the coefficient of the interaction term is >0.042 , or 24.5% of its largest possible value given the data (0.173). This is roughly the same degree of sensitivity as the Slothuus study. The 90% confidence interval overlaps with zero when σ is ≥ 0.013 . The bottom panel indicates that these values of σ translate to the \tilde{R}^2 values of 0.036 and 0.006, respectively, implying that the heterogeneity in the interaction between the frame and anxiety must only explain 3.6% of the total variance in the outcome variable out of its 59.3% residual variance. Thus, the evidence for the anxiety mechanism in Brader et al.'s study becomes quite fragile when we allow for its dependence on the participants' perceived harm of immigration.

7 Statistical Analysis When the Mediator Can Be Manipulated

So far we have analyzed standard randomized experiments where the treatment is randomized but the mediator is not. In this setup, the exogeneity of the mediator must be assumed instead of guaranteed by the experimental design. This remains an important limitation even though our framework described in Section 5 allows for the existence of posttreatment observed confounders. For example, in Druckman and Nelson's framing experiment, we cannot rule out the possibility that there exists an unobserved posttreatment confounder affecting both the perceived issue importance and opinions about the campaign finance reform, even after taking into account the belief content mechanism.

In this section, we extend our method to the two new experimental designs recently proposed by Imai, Tingley, and Yamamoto (2013), where the mediator is manipulated to avoid the exogeneity assumption. In particular, we first consider the parallel design where two randomized experiments are run in parallel; one experiment employs the standard design where only the treatment is randomized, whereas the other experiment randomizes both the treatment and the mediator of interest. We also consider the parallel encouragement design where the manipulation of the mediator is imperfect in the second experiment. These designs allow for the existence of causally dependent multiple mediators W and yet can make the main mediator M unconfounded by directly

or indirectly randomizing it. Thus, under these designs, we no longer need to measure W . While the randomization of the treatment and the mediator is not sufficient for the identification of the ACME (Imai, Tingley, and Yamamoto 2013), we develop a sensitivity analysis that can be used to examine the robustness of empirical findings to the potential violation of the key identification assumption under each of these two designs. While these designs are new and hence have not yet been used by many applied researchers, we hope that their future use can improve the credibility of causal mediation analysis.

7.1 The Parallel Design

Under the parallel design, we randomly split the sample into two groups and conduct a separate randomized experiment for each group in parallel. One experiment is conducted under the standard design where, after the treatment is randomized and administered, the values of the mediator and the outcome are measured. In the other experiment, both the treatment and the mediating variables are randomized and subsequently the values of the outcome variable are recorded. We assume that the values of the potential outcomes remain identical regardless of the experiment to which each unit is assigned. In other words, the potential outcomes are assumed to depend on the values of the treatment and mediator, but not how they are realized. Imai, Tingley, and Yamamoto (2013) emphasize the importance of this assumption and suggest that the manipulation of the mediator needs to be subtle in order to make sure that it affects the outcome only through the mediator value and has no direct effect.

Under this design, the first experiment allows for the existence of multiple mechanisms that may be causally related. However, in the second experiment, the randomization of the mediator removes all of its confounders including alternative mediators. Formally, let D_i be an indicator variable representing whether unit i is randomly assigned to the first ($D_i=0$) or second ($D_i=1$) experiment. The study design implies the following conditional independence,

$$\{Y_i(t, m), M_i(t')\} \perp\!\!\!\perp T_i | D_i = 0, \quad (41)$$

$$\{Y_i(t, m)\} \perp\!\!\!\perp \{T_i, M_i\} | D_i = 1, \quad (42)$$

for any t, t', m . Note that we do not assume independence between the mediator and the potential outcomes in the first experiment. This means that while the manipulated values of the mediator must be random in the second experiment, the parallel design makes no exogeneity assumption on the mediator values that would naturally realize when only the treatment is manipulated as in the first experiment. This contrasts with the scenarios depicted in Fig. 1 where observed mediators are assumed to be conditionally exogenous. The parallel design also differs from Fig. 1 in that there is no need to determine whether there exist alternative mechanisms that are causally related to the mechanism of interest.

Under the parallel design, the first experiment identifies the average effects of the treatment on the mediator and the outcome. That is, both $\mathbb{E}\{M_i(t)\}$ and $\mathbb{E}\{Y_i(t, M_i(t))\}$ are identifiable for $t=0, 1$. The second experiment, on the other hand, identifies $\mathbb{E}\{Y_i(t, m)\}$ for any t and m . Unfortunately, this is not sufficient to identify the ACME. Following Robins (2003), Imai, Tingley, and Yamamoto (2013) show that under the parallel design, the ACME is nonparametrically identified if the assumption of no-interaction effect holds. This assumption, which is essentially the same as Assumption 3, can be formally written as

$$Y_i(1, m) - Y_i(1, m') = Y_i(0, m) - Y_i(0, m'), \quad (43)$$

for any $m \neq m'$. As is the case for Assumption 3, the equality must hold at the unit level rather than in expectation, which makes this assumption unverifiable and unrealistic in most cases. However, Imai, Tingley, and Yamamoto (2013) show that without the no-interaction assumption, the ACME cannot even be bounded under the parallel design unless the outcome variable has finite bounds. They also show that even when the outcome and the mediator are binary, the sharp bounds on the ACME may often contain zero, failing to identify its sign (see also Kaufman, Kaufman, and MacLehose 2009; Sjölander 2009). Thus, below, we develop a sensitivity analysis for the

no-interaction effect assumption to assess the consequence of the violation of this key identification assumption.

7.2 The Proposed Methodology for the Parallel Design

Given the setup described above, we develop a sensitivity analysis for inference based upon the following system of linear equations with varying coefficients,

$$M_i(t) = \alpha_2 + \beta_{2i}t + \varepsilon_{2i} \quad (44)$$

$$Y_i(t, m) = \alpha_3 + \beta_{3i}t + \gamma_i m + \kappa_i t m + \varepsilon_{3i}, \quad (45)$$

for any t and m , where $\mathbb{E}(\varepsilon_{2i}) = \mathbb{E}(\varepsilon_{3i}) = 0$ without loss of generality. Like the one considered in Section 5, this model allows for arbitrary degrees of heterogeneity across units and does not make any distributional assumption about these effects. Thus, despite the linearity assumption, equations (44) and (45) represent a general class of models that are sufficiently flexible to be useful in a variety of applied research settings.

Under the parallel design, equations (44) and (45) can be fitted separately to the data from the first and the second experiments, respectively. The randomization of the treatment in the first experiment ($D_i = 0$) and that of the treatment and the mediator in the second experiment ($D_i = 1$) guarantee that the following exogeneity assumptions are satisfied:

$$\mathbb{E}(\varepsilon_{2i}|T_i, D_i = 0) = \mathbb{E}(\varepsilon_{3i}|T_i, M_i, D_i = 1) = 0. \quad (46)$$

In addition, due to the linearity and the binary nature of the treatment, the model implies the following linear relationship between the treatment and the outcome,

$$Y_i(t, M_i(t)) = \alpha_1 + \beta_{1i}t + \varepsilon_{1i}, \quad (47)$$

where $\alpha_1 + \varepsilon_{1i} = \alpha_3 + (\alpha_2 + \varepsilon_{2i})\gamma_i + \varepsilon_{3i}$, $\beta_{1i} = \beta_{3i} + \beta_{2i}\gamma_i + (\alpha_2 + \beta_{2i} + \varepsilon_{2i})\kappa_{2i}$, and $\mathbb{E}(\varepsilon_{1i}) = 0$.

Now, we can rewrite the model given in equations (44) and (45) as

$$M_i(t) = \alpha_2 + \beta_2 t + \eta_{2i}(t) \quad (48)$$

$$Y_i(t, m) = \alpha_3 + \beta_3 t + \gamma m + \kappa t m + \eta_{3i}(t, m), \quad (49)$$

where $\beta_2 = \mathbb{E}(\beta_{2i})$, $\beta_3 = \mathbb{E}(\beta_{3i})$, $\gamma = \mathbb{E}(\gamma_i)$, $\kappa = \mathbb{E}(\kappa_i)$, and $\eta_{2i}(t) = \tilde{\beta}_{2i}t + \varepsilon_{2i}$, and $\eta_{3i}(t, m) = \tilde{\beta}_{3i}t + \tilde{\gamma}_i m + \tilde{\kappa}_i t m + \varepsilon_{3i}$ with $\mathbb{E}(\tilde{\beta}_{2i}) = \mathbb{E}(\tilde{\beta}_{3i}) = \mathbb{E}(\tilde{\gamma}_i) = \mathbb{E}(\tilde{\kappa}_i) = 0$. Thus, under the parallel design, the exogeneity assumption given in equation (46) implies that among the parameters of equations (48) and (49), $(\alpha_2, \alpha_3, \beta_2, \beta_3, \gamma, \kappa)$ are identified.

To develop a sensitivity analysis, we follow the analytical strategy employed in Section 5 and write the ADE using the model parameters as follows:

$$\bar{\delta}(t) = \beta_1 - \bar{\zeta}(1 - t) \quad (50)$$

$$\bar{\zeta}(t) = \beta_3 + (\alpha_2 + \beta_2 t)\kappa + \rho_t \sigma \sqrt{\mathbb{V}(M_i|T_i = t, D_i = 0)}, \quad (51)$$

for $t = 0, 1$, where the two sensitivity parameters are $\rho_t = \text{Corr}(M_i(t), \kappa_i)$ and $\sigma = \sqrt{\mathbb{V}(\kappa_i)}$, and other parameters can be consistently estimated from the observed data because other parameters are identifiable under this design. These sensitivity parameters, ρ_t and σ , represent the degree to which the individual-level treatment–mediator interaction effect is correlated with the mediator of interest and the amount of heterogeneous interaction effect, respectively. Researchers can vary these two sensitivity parameters within their plausible range to assess the sensitivity of their empirical results to the violation of the no-interaction effect assumption under the parallel design.

This sensitivity analysis under the parallel design is essentially identical to that of Section 5 except that there is no need to consider alternative mediators W since M is randomized. Thus, most of the remarks made in Section 5 also apply here. First, the no-interaction effect assumption given in equation (43) implies $\kappa_i = 0$ for all i and thus $\kappa = \rho_t = \sigma = 0$. In this case, both the ACME and the

ADE are identified as $\bar{\delta}(t) = \beta_1 - \beta_3 = \beta_2\gamma$ and $\bar{\zeta}(t) = \beta_3$, respectively, which equal the usual mediation procedure under sequential ignorability without the treatment–mediator interaction.

Second, one can consider the following homogeneous interaction effect assumption, $Y_i(1,m) - Y_i(0,m) = B_i + Cm$, where C does not vary across units. Under the linear models considered here, this assumption implies $\tilde{\kappa}_i = 0$ for all i and thus $\sigma = 0$. Therefore, both the ACME and the ADE are identified as $\bar{\delta}(t) = \beta_1 - \beta_3 - (\alpha_2 + \beta_2(1-t))\kappa = \beta_2(\gamma + t\kappa)$ and $\bar{\zeta}(t) = \beta_3 + (\alpha_2 + \beta_2 t)\kappa$, respectively. These formulas agree with the standard mediation procedure under the linear structural modeling with the treatment–mediator interaction term (e.g., Kraemer et al. 2008). Our analysis therefore highlights the implicit assumption made when researchers apply the standard mediation analysis procedure.

Third, if neither of these assumptions holds, then the standard estimates of the ACME and the ADE will be biased even under the parallel design where Assumptions (41) and (42) are both satisfied. For example, the bias for the ADE $\bar{\zeta}(t)$ is equal to $\rho_t \sigma \sqrt{\mathbb{V}(M_i|T_i = t, D_i = 0)}$. This implies that $\bar{\zeta}(t)$ will be overestimated if the mediator positively interacts with the treatment for those units that tend to have high mediator values when the treatment status is t (i.e., $\rho_t > 0$). The magnitude of such bias will be large when the degree of heterogeneity for the treatment–mediator interaction effect is high (i.e., σ is large).

Finally, as in Section 5, this sensitivity analysis can be conducted with respect to an alternative sensitivity parameter instead of σ for easier interpretation. Specifically, we can use the proportion of the unexplained or original variance of the outcome that is additionally explained by including the heterogeneity in the treatment–mediator interaction. This quantity is represented by the following coefficients of determination: $R^{2*} = \mathbb{V}(\kappa_i T_i M_i | D_i = 1) / \mathbb{V}(\eta_{3i}(T_i, M_i) | D_i = 1)$ for the unexplained variance and $\tilde{R}^2 = \mathbb{V}(\kappa_i T_i M_i | D_i = 1) / \mathbb{V}(Y_i | D_i = 1)$ for the original variance. For example, R^{2*} represents how much of the observed variance in Y_i can be explained by the inclusion of the term $\kappa_i T_i M_i$ in the regression model. Then, it can be shown that σ can be alternatively expressed as a function of each of these coefficients of determination,

$$\sigma = \sqrt{\frac{\mathbb{V}(\eta_{3i}(T_i, M_i) | D_i = 1) R^{2*}}{\mathbb{E}(T_i M_i^2 | D_i = 1)}} = \sqrt{\frac{\mathbb{V}(Y_i | D_i = 1) \tilde{R}^2}{\mathbb{E}(T_i M_i^2 | D_i = 1)}}. \quad (52)$$

Thus, we can conduct the sensitivity analysis with the sensitivity parameters R^{2*} and \tilde{R}^2 . From this expression, it is immediate that the upper bound of σ results when the heterogeneity in the treatment–mediator interaction explains all the unexplained variance, i.e., $\sigma \leq \sqrt{\mathbb{V}(\eta_{3i}(T_i, M_i) | D_i = 1) / \mathbb{E}(T_i M_i^2 | D_i = 1)}$.

7.3 The Parallel Encouragement Design

The sensitivity analysis developed above can be extended to the “parallel encouragement design” of Imai, Tingley, and Yamamoto (2013), where the manipulation of the mediator in the second experiment of the parallel design is imperfect. This situation is more realistic for psychological studies, such as framing experiments, because manipulating psychological mechanisms is likely to be imperfect at best even with clever use of intervention techniques.

Under this design, it is assumed that the randomized manipulation of the mediator monotonically affects the mediator and this manipulation affects the outcome only through the realized value of the mediator. Then, the manipulation Z_i can be used as an instrumental variable and we can fit the two-stage least squares regression model. That is, equation (44) can be replaced with the following equation,

$$M_i(t, z) = \alpha_2 + \beta_{2i}t + \lambda_{iz} + \theta_{it}z + \varepsilon_{2i}, \quad (53)$$

whereas equation (45) remains identical. Implicit in this model is the assumption that all units in the study population can be thought of as following this structural equation as the true data-generating process. This contrasts with the nonparametric approach to encouragement designs recently developed in the methodological literature (Angrist, Imbens, and Rubin 1996) and applied to the causal mediation analysis (Imai, Tingley, and Yamamoto 2013).

Under this two-stage least squares model, it is straightforward to show that the ADE and ACME, $\bar{\zeta}(t,z) = \mathbb{E}\{Y_i(1,M_i(t,z)) - Y_i(0,M_i(t,z))\}$ and $\bar{\delta}(t,z) = \mathbb{E}\{Y_i(t,M_i(1,z)) - Y_i(t,M_i(0,z))\}$, equal the following expressions:

$$\bar{\zeta}(t,z) = \beta_3 + (\alpha_2 + \beta_2 t + \lambda z + \theta t z)\kappa + \rho_{t,z}\sigma\sqrt{\mathbb{V}(M_i|T_i = t, Z_i = z)}, \quad (54)$$

$$\bar{\delta}(t,z) = \bar{\tau} - \beta_3 - (\alpha_2 + \beta_2(1-t) + \lambda z + \theta t z)\kappa - \rho_{1-t,z}\sigma\sqrt{\mathbb{V}(M_i|T_i = 1-t, Z_i = z)}, \quad (55)$$

where $\lambda = \mathbb{E}(\lambda_i)$, $\theta = \mathbb{E}(\theta_i)$ (which are both identified due to the exogeneity of T_i and Z_i) and $\rho_{t,z} = \text{Corr}(M_i(t,z), \kappa_i)$ for $t \in \{0,1\}$ and $z \in \mathcal{Z}$ (the support of Z_i). The proof is provided in Appendix A5. The average total effect, $\bar{\tau}$ in equation (55), can be consistently estimated by regressing Y_i on T_i for the subsample that received the manipulation of the treatment alone, i.e., $Z_i = 0$. Then, the sensitivity and bounds analyses can be conducted by estimating the identifiable parameters via the two-stage least squares method and varying $\rho_{t,z}$ and σ within the ranges of plausible values.

8 Concluding Remarks and Suggestions for Applied Researchers

In this article, we have shown how to conduct causal mediation analysis in the presence of multiple mediators. The proposed methodology can be applied even when alternative mechanisms are causally related to each other so long as both the treatment and the mediating variables can be assumed to be exogenous conditional on a set of observed confounders. However, this methodology is not applicable when there exist unobserved posttreatment confounders. Thus, we also show that our methodology can be applied to new experimental designs where these exogeneity conditions can be ensured by experimenters. Finally, we conclude this article by offering a list of practical suggestions for applied researchers who wish to study multiple causal mechanisms.

- Identify a list of alternative causal mechanisms that are causally prior to the mechanism of your interest and measure mediators that represent them.
- Consider theoretically whether the identified alternative mechanisms are causally related to the causal mechanisms of interest and test empirically whether they are statistically dependent on each other even after adjusting for the treatment and pretreatment covariates.
- If alternative mechanisms are causally independent of each other, apply the standard causal mediation analysis and conduct sensitivity analysis for the possible existence of unobserved pretreatment confounders.
- If alternative mechanisms are causally related to each other, apply the mediation analysis that directly accounts for multiple mediators and conduct sensitivity analysis with respect to the existence of treatment–mediator interaction effects.
- Whenever possible, utilize research designs, experimental or observational, where the exogeneity of mediator is credible and apply sensitivity analysis for the no treatment–mediator interaction assumption.

A Mathematical Appendix

A.1 Proof of Nonparametric Identification of ACMEs under Assumption 1

The proof is a straightforward extension of that of Theorem 1 in Imai, Keele, and Yamamoto (2010c). We first consider the identification of $\bar{\delta}_i^M(t)$, the ACME with respect to M_i . Note that equation (16) implies the following conditional independence:

$$Y_i(t,m,W_i(t)) \perp\!\!\!\perp T_i \mid M_i(t') = m', X_i = x, \quad (A1)$$

for all $t, t' = 0, 1, m, m'$, and x . Now, for any t, t' , we have,

$$\begin{aligned} & \mathbb{E}(Y_i(t,M_i(t'),W_i(t))|X_i = x) \\ &= \int \mathbb{E}(Y_i(t,m,W_i(t))|M_i(t') = m, X_i = x) dF_{M_i(t')|X_i=x}(m) \end{aligned} \quad (A2)$$

$$= \int \mathbb{E}(Y_i(t, m, W_i(t)) | M_i(t') = m, T_i = t', X_i = x) dF_{M_i(t') | X_i=x}(m) \quad (\text{A3})$$

$$= \int \mathbb{E}(Y_i(t, m, W_i(t)) | T_i = t', X_i = x) dF_{M_i(t') | X_i=x}(m) \quad (\text{A4})$$

$$= \int \mathbb{E}(Y_i(t, m, W_i(t)) | T_i = t, X_i = x) dF_{M_i(t') | T_i=t', X_i=x}(m) \quad (\text{A5})$$

$$= \int \mathbb{E}(Y_i(t, m, W_i(t)) | M_i(t) = m, T_i = t, X_i = x) dF_{M_i(t') | T_i=t', X_i=x}(m) \quad (\text{A6})$$

$$= \int \mathbb{E}(Y_i | M_i = m, T_i = t, X_i = x) dF_{M_i(t') | T_i=t', X_i=x}(m) \quad (\text{A7})$$

$$= \int \mathbb{E}(Y_i | M_i = m, T_i = t, X_i = x) dF_{M_i | T_i=t', X_i=x}(m), \quad (\text{A8})$$

where the second equality follows from equation (A1), equation (16) is used to establish the third and the fifth equalities, equation (15) is used to establish the fourth and the last equalities, and the sixth equality follows from the fact that $M_i = M_i(T_i)$ and $Y_i = Y_i(T_i, M_i(T_i), W_i(T_i))$. This implies that $\bar{\delta}^M(t)$ can be identified as

$$\bar{\delta}^M(t) = \int \int \mathbb{E}(Y_i | M_i = m, T_i = t, X_i = x) \{dF_{M_i | T_i=1, X_i=x}(m) - dF_{M_i | T_i=0, X_i=x}(m)\} dF_{X_i}(x),$$

which is identical to the expression in Theorem 1 of Imai, Keele, and Yamamoto (2010c). Furthermore, the same proof applies to $\bar{\delta}^W(t)$ by considering the identification of $\mathbb{E}(Y_i(t, M_i(t), W_i(t)) | X_i = x)$ using equation (17) instead of equation (16). Finally, $\bar{\zeta}(t, t')$ is also identified because $\bar{\zeta}(t, t') = \bar{\tau} - \bar{\delta}^M(t') - \bar{\delta}^W(t)$ and $\bar{\tau}$ is identified under equation (15).

A.2 Proof of the Parametric Identification of ACMEs with the Path Analysis under Assumption 1

The proof follows a similar argument as Theorem 2 of Imai, Keele, and Yamamoto (2010c). First, note that the coefficients in equations (18–20) are all identified under Assumption 1. Next, note that given this linear structural equations model, equation (A8) can be written as

$$\begin{aligned} & E(Y_i(t, M_i(t'), W_i(t)) | X_i = x) \\ &= \int \mathbb{E}(Y_i | M_i = m, T_i = t, X_i = x) dF_{M_i | T_i=t', X_i=x}(m) \end{aligned} \quad (\text{A9})$$

$$= \int \int (\alpha_3 + \beta_3 t + \gamma m + \theta^\top w + \xi_3^\top x) dF_{W_i | M_i=m, T_i=t, X_i=x}(w) dF_{M_i | T_i=t', X_i=x}(m) \quad (\text{A10})$$

$$= \int \int (\alpha_3 + \beta_3 t + \gamma m + \theta^\top w + \xi_3^\top x) dF_{W_i | T_i=t, X_i=x}(w) dF_{M_i | T_i=t', X_i=x}(m) \quad (\text{A11})$$

$$= \alpha_3 + \beta_3 t + \gamma(\alpha_M + \beta_M t' + \xi_M^\top x) + \theta^\top(\alpha_W + \beta_W t + \xi_W^\top x) + \xi_3^\top x, \quad (\text{A12})$$

where the third equality holds because equation (16) implies $M_i \perp\!\!\!\perp W_i | T_i = t, X_i = x$ under the linear structural equations model in equations (18–20). Finally, we also have

$$\mathbb{E}(Y_i(t, M_i(t), W_i(t)) | X_i = x) = \alpha_3 + \beta_3 t + \gamma(\alpha_M + \beta_M t + \xi_M^\top x) + \theta^\top(\alpha_W + \beta_W t + \xi_W^\top x) + \xi_3^\top x. \quad (\text{A13})$$

Therefore, $\bar{\delta}^M(t) = \beta_M \gamma$. The same argument applies to $\bar{\delta}^W(t)$, and it is identified as $\beta_W \theta$. \square

A.3 Proof of the Identification of the ADE Given ρ_t and σ

We begin by expressing the ADE defined in equation (22) using the parameters of the model given in equations (28) and (29),

$$\begin{aligned}\bar{\zeta}(t) &= \mathbb{E}\{\beta_{3i} + \kappa_i(\alpha_2 + \beta_{2i}t + \xi_{2i}^\top W_i(t) + \mu_{2i}^\top t W_i(t) + \lambda_{2i}^\top x + \varepsilon_{2i}) + \xi_{3i}^\top (W_i(1) - W_i(0)) + \mu_{3i}^\top W_i(1)\} \\ &= \beta_3 + \kappa \mathbb{E}(M_i | T_i = t) + \rho_t \sigma \sqrt{\mathbb{V}(M_i | T_i = t)} + \mathbb{E}\{(\xi_{3i} + \mu_{3i})^\top W_i(1) - \xi_{3i}^\top W_i(0)\},\end{aligned}\quad (\text{A14})$$

where the two conditional moments of M_i , $\mathbb{E}(M_i | T_i = t)$ and $\mathbb{V}(M_i | T_i = t)$ can be consistently estimated using their sample counterparts. Now, note that the last term of equation (A14) can be written as

$$\mathbb{E}\{(\xi_{3i} + \mu_{3i})^\top W_i(1) - \xi_{3i}^\top W_i(0)\} = \mathbb{E}\{(\xi_{3i} + \mu_{3i})^\top W_i(1) | T_i = 1\} - \mathbb{E}(\xi_{3i}^\top W_i(0)) \quad (\text{A15})$$

$$= \mathbb{E}\{(\xi_{3i} + \mu_{3i})^\top | T_i = 1\} \mathbb{E}(W_i(1) | T_i = 1) - \mathbb{E}(\xi_{3i}^\top W_i(0)) \quad (\text{A16})$$

$$= (\xi_3 + \mu_3)^\top \mathbb{E}(W_i | T_i = 1) - \mathbb{E}(\xi_{3i}^\top W_i(0)), \quad (\text{A17})$$

where the equalities follow from equation (24) and the fact that each element of the coefficient vector $\xi_{3i} + \mu_{3i}$ is conditionally independent of $W_i(1)$ given $T_i = 1$ under Assumption 2. The latter holds because for any j and m , $\xi_{3ij} + \mu_{3ij} = Y_i(1, m, w) - Y_i(1, m, w')$, where ξ_{3ij} and μ_{3ij} denote the j th elements of ξ_{3i} and μ_{3i} , respectively, $w = (w_1, \dots, w_j, \dots, w_J)^\top$, and $w' = (w_1, \dots, w_j - 1, \dots, w_J)^\top$. Likewise, we have

$$\mathbb{E}\{(\xi_{3i} + \mu_{3i})^\top W_i(1) - \xi_{3i}^\top W_i(0)\} = \mathbb{E}\{(\xi_{3i} + \mu_{3i})^\top W_i(1)\} - \xi_3^\top \mathbb{E}(W_i | T_i = 0). \quad (\text{A18})$$

Since $\xi_{3ij} = Y_i(0, m, w) - Y_i(0, m, w')$ for any $m \in \mathcal{M}$ and $j \in \{1, \dots, J\}$. Together with equation (A17), we obtain

$$\mathbb{E}\{(\xi_{3i} + \mu_{3i})^\top W_i(1) - \xi_{3i}^\top W_i(0)\} = (\xi_3 + \mu_3)^\top \mathbb{E}(W_i | T_i = 1) - \xi_3^\top \mathbb{E}(W_i | T_i = 0), \quad (\text{A19})$$

which implies that the final term of equation (A14), $\mathbb{E}\{(\xi_{3i} + \mu_{3i})^\top W_i(1) - \xi_{3i}^\top W_i(0)\}$, is identified. Therefore, the ADE is identified given the two sensitivity parameters, ρ_t and σ , and so is the ACME, which can be obtained by subtracting the ADE from the average total effect. \square

A.4 Proof of the Identification of the Causal Mediation Effect Specific to the Path $T \rightarrow W \rightarrow Y$

First, note that the population average of the path-specific effect in equation (39) can be written using the model parameters given in equations (28) and (29) as

$$\bar{\chi}(t) = \mathbb{E}\{(\xi_{3i} + t\mu_{3i})^\top (W_i(1) - W_i(0))\}. \quad (\text{A20})$$

Now, following the same argument as in Appendix A3, we can show that under Assumption 2, this expectation can be written as a function of identified model parameters,

$$\mathbb{E}\{(\xi_{3i} + t\mu_{3i})^\top (W_i(1) - W_i(0))\} = (\xi_3 + t\mu_3)^\top \{\mathbb{E}(W_i | T_i = 1) - \mathbb{E}(W_i | T_i = 0)\}. \quad (\text{A21})$$

A.5 Proof of the Identification of the ADE Given ρ_{tz} and σ under the Parallel Encouragement Design

The expression for the ADE in equation (54) can be derived as follows:

$$\begin{aligned}\bar{\zeta}(t, z) &= \mathbb{E}\{\beta_{3i} + \kappa_i M_i(t, z)\} \\ &= \beta_3 + \mathbb{E}(\kappa_i) \mathbb{E}(M_i(t, z)) + \text{Cov}(\kappa_i, M_i(t, z)) \\ &= \beta_3 + (\alpha_2 + \beta_2 t + \lambda z + \theta t z) \kappa + \rho_{tz} \sigma \sqrt{\mathbb{V}(M_i | T_i = t, Z_i = z)},\end{aligned}$$

where the first equality follows from equation (45) and the last equality from equation (53) and the fact that both T_i and Z_i are randomized. Then, the expression for the ACME in equation (55) can be derived by substituting the above expression into the following equality: $\bar{\delta}(t, z) = \bar{\tau} - \bar{\zeta}(1 - t, z)$. \square

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