Beyond Moderation:

Exploring Person-Level Mediation with Residuals and Individual Model Fit

by

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ABSTRACT

Mediation analysis is integral to psychology, investigating human behavior's causal mechanisms. The diversity of explanations for human behavior has implications for the estimation and interpretation of statistical mediation models. Individuals can have similar observed outcomes while undergoing different causal processes or different observed outcomes while receiving the same treatment. Researchers can employ diverse strategies when studying individual differences in multiple mediation pathways, including individual fit measures and analysis of residuals. This dissertation investigates the use of individual residuals and fit measures to identify individual differences in multiple mediation pathways. More specifically, this study focuses on mediation model residuals in a heterogeneous population in which some people experience indirect effects through one mediator and others experience indirect effects through a different mediator. A simulation study investigates 162 conditions defined by effect size and sample size for three proposed methods: residual differences, delta *z*, and generalized Cook's distance. Results indicate that analogs of Type 1 error rates are generally acceptable for the method of residual differences, but statistical power is limited. Likewise, neither delta *z* nor gCd could reliably distinguish between contrasts that had true effects and those that did not. The outcomes of this study reveal the potential for statistical measures of individual mediation. However, limitations related to unequal subpopulation variances, multiple dependent variables, the inherent relationship between direct effects and unestimated indirect effects, and minimal contrast effects require more research to develop a simple method that researchers can use on single data sets.

DEDICATION

To Jesse and the Scooby Gang.

If it's no Blinsky, it's no fun.

Somehow, everything turns up Blinsky when you are around.

ACKNOWLEDGMENTS

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CHAPTER 1

INTRODUCTION

Virgil foreshadowed the merit of statistical mediation when he stated, "*Felix, qui potuit rerum cognoscere causas*," or, in the vernacular, "Happy is the person who can know the causes of things" (Virgil, 29 B.C.E, Book 2, line 490). Mediation analysis is integral to psychology because it investigates the causal mechanisms that underlie human behavior. However, the equifinal and multifinal natures of many behavioral processes as well as the multiplism inherent in scientific methods complicate the investigation of mediation (Cicchetti & Rogosch, 1996; Mayr, 1964, 1988; Shadish, 1993; Wilden, 1980). Simply put, there can be a diversity of pathways that lead to a given outcome (*equifinality*), and various endpoints can be arrived at from any given starting point (*multifinality)*. Moreover, multiple scientific strategies can be employed to predict or explain processes, while often, no single strategy can be unequivocally labeled "best" (*multiplism*).

The diversity of explanations for any given human behavior has implications for estimating and interpreting statistical mediation models. Within the context of statistical mediation models, the principle of equifinality (Cicchetti & Rogosch, 1996; Mayr, 1964, 1988; Wilden, 1980) implies that individuals can have similar observed outcomes while undergoing different causal processes, which could manifest as differences in b-paths among multiple mediating variables. Likewise, the principle of multifinality (Cicchetti & Rogosch, 1996; Mayr, 1964, 1988; Wilden, 1980) suggests that individuals can have different observed outcomes while receiving the same treatment, which could manifest as differences in a-paths from a single treatment to

multiple mediators. Finally, the principle of multiplism (Shadish, 1993) affirms that researchers can use various strategies to study individual differences in multiple mediation pathways, including moderation, latent classes and profiles, multilevel models, configural frequency mediation, individual fit measures, and analysis of residuals.

This dissertation investigates how individual residuals can identify individual differences in multiple mediation pathways. In addition, the investigation extends to three individual fit measures that are akin to residuals: delta *z* (Pek & MacCallum, 2011), generalized Cook's distance (gCd; Pek & MacCallum, 2011), and individual Chi-square (IND_{CHI}; Reise & Widaman, 1999). More specifically, this study focuses on residuals in mediation effects in a heterogeneous population such that some people experience indirect effects through one mediator while other people experience indirect effects through a different mediator, assuming the same independent and dependent variables. Currently, literature on residual analysis and person-fit has focused on influential cases, outlier identification, and the comparison of person-fit and local influence measures in the context of regression and SEM (Asparouhov & Muthen, 2015; Cook, 1986; Lee & Tang, 2004; Pek & MacCallum, 2011; Reise & Widaman, 1999; Zu & Yuan, 2010). Investigating individual case influence is necessary for understanding individual-level causal effects. Nevertheless, there appears to be no published research directly investigating person-centered mediation effects using techniques like case influence, person-fit indices, or residual analysis.

CHAPTER 2

RESIDUALS AND INDIVIDUAL MODEL FIT

Overview

Residual analysis is a diagnostic tool that assesses data quality in regression models by identifying outliers and influential cases. Cook (1986) developed a method to determine local influence based on likelihoods and differential geometry, which became the foundation for local influence research. Another common residual analysis technique uses a series of model perturbations, such as a single case deletion scheme (Pek & MacCallum, 2011).

Local influence analysis relies on the logic that outliers may exert more weight than other observations and be detectable as influential observations. Researchers are often concerned with cases that influence parameter estimates. In the context of mediation, if an observation has a true effect through a mediator that differs from the mediator specified in the model, then a downward bias towards zero would be predicted for the estimated parameter of the modeled mediator. Outliers and influential observations may also be detectable when associated with large residuals (Bollen & Arminger, 1991). Residuals may be larger when a different, unmodeled mediator is present but not included in the statistical model. Both ideas, that bias in parameter estimates and size of residuals could be indicators of which mediator is associated with an individual observation, could be investigated by comparing residuals and parameter estimates in different mediator models. The comparison between residuals for models with different mediators is the basis of the simulation proposed below.

Bollen and Arminger (1991) described the estimation of individual residuals in the context of factor analysis, which can be expanded to mediation models estimated in a latent variable framework. Unstandardized residuals can be computed as the difference between the observed dependent variable and the factor scores of the latent predictor variables. In equation 2.1, ϵ_i is the individual unstandardized residual for observation *i,* Y_i is the observed outcome, Λ is a matrix of factor loadings, and η_i is a vector of random latent variables.

$$\epsilon_i = Y_i - \Lambda\eta_i \tag{2.1}$$

The latent variables represented by η_i are the estimated weighted function of the observed outcome, as in equation 2.2, where $\hat{\eta}_i$is the estimated factor score, W is a weight matrix, and Y_i is the individual observed score on the outcome. The combination of equations 2.1 and 2.2 results in equation 2.3, which highlights the role of the weight matrix in estimating individual residuals in a latent variable model.

$$\hat{\eta}_i = WY_i \tag{2.2}$$

$$\epsilon_i = Y_i(\mathrm{I} - \Lambda W) \tag{2.3}$$

There are two common methods for estimating the weight matrix, *W.* The first is based on least square estimation and involves minimizing the sum of squares of the latent variables. The second is Bartlett's method, which minimizes the sum of squares divided by their standard deviations (Lawley & Maxwell, 1971). In a simulation study, Bollen and Arminger (1991) found that standardized residuals calculated via Bartlett's method could detect outlying variables.

Delta *z* and Generalized Cook's Distance

Local influence measures can be extended from regression diagnostics to a model fit measure in SEM. Pek and MacCallum (2011) showed that individual cases could affect different aspects of model results, like model fit and parameter estimates, which require different diagnostic measures depending on the goal of the analysis. In addition, they noted the distinction between influential cases and outliers, namely that not all outliers are necessarily influential, and not all influential cases are outliers.

Pek and MacCallum (2011) measured case influence on overall model fit by adapting the likelihood distance deletion statistic. The deletion statistic measures the displacement in the likelihood function that occurs when a case is deleted. The direction of change of a parameter estimate is computed as the standardized difference between the parameter value with and without the observation in question ($\delta\theta$; see equation 2.4). This computation is much like the DFBETAS from a regression analysis. A positive $\delta\theta$ indicates a reduction in the parameter, while a negative $\delta\theta$ indicates an increase in the parameter. Significant model effects that are robust to the deletion of any single observation would have a range of $\delta\theta_i$ from all the delete-one samples that do not include the parameter value from the full sample. Pek and MacCallum (2011) found that this measure summarized case influence on model fit and indicated whether the case was consistent with the specified model.

$$\delta\theta_i = \frac{\left(\hat{\theta} - \hat{\theta}_i\right)}{\left[\widehat{Var\left(\hat{\theta}_i\right)}\right]^{\frac{1}{2}}} \tag{2.4}$$

To investigate how a single case influences the magnitude of parameter estimates, Pek and MacCallum (2011) used generalized Cook's Distance (gCd) to determine the absolute magnitude of change when case i is deleted (see equation 2.5)

$$gCD_i = \left(\hat{\theta} - \hat{\theta}_i\right)' \widehat{Var}\left(\hat{\theta}_i\right)^{-1} \left(\hat{\theta} - \hat{\theta}_i\right) \tag{2.5}$$

Equation 2.5 can also be written in terms of $\delta\theta_i$.

$$gCD_i = [\delta\theta_i]^2 \tag{2.6}$$

The delete-one observation perturbation scheme described by Pek and MacCallum (2011) can be applied to indirect effects using the Sobel z statistic. In equation 2.7, the change in the general parameter estimate, θ, is replaced by the change in the mediated effect calculated as the product of two regression coefficients (MacKinnon, 2008; Pek & MacCallum, 2011).

$$\Delta z = \frac{\widehat{ab} - \widehat{a_i b_i}}{\sqrt{\hat{a_i}^2 s_{\hat{b_i}}^2 + \hat{b_i}^2 s_{\hat{a_i}}^2}} \tag{2.7}$$

Individual Chi-Square

Model fit measures generally rely on the log-likelihood to evaluate goodness of fit. Reise and Widaman (1999) demonstrated a method of determining model fit for individual observations based on the log-likelihood of individual responses, individual Chi-square (IND_{CHI}). IND_{CHI} is a person fit statistic for structural equation models that quantifies an individual's contribution to the overall model Chi-square statistic by comparing individual fit in hypothesized and saturated models, similar to the overall model Chi-square statistic.

Individual model fit is assessed with IND_{CHI} values by partitioning a model's overall log-likelihood into contributions made by each observation using equation 2.8

(Lange et al., 1976), where p is a vector of variables, x_i is a vector of responses for individual i, and M is a vector of sample means. The first part of the formula is a constant that applies to all the observations in a sample, while the second part is the Mahalanobis squared distance for the individual.

$$P_{LL} = -\frac{1}{2}\left[p\ln(2\pi) + \ln|\Sigma^*| + (x_i - M)\Sigma^{*-1}(x_i - M)\right] \quad (2.8)$$

Coffman and Millsap (2006) found that individual contribution to misfit was low, even among the highest -2PLL values. However, the rank order of percent contribution was robust, such that observations with lower -2PLL values had even lower percent contributions to overall model misfit. Logically, as the number of observations increases, the maximum percentage that any single observation can be expected to contribute towards the overall model Chi-square decreases. Therefore, making relative rather than absolute comparisons of percentage contribution is reasonable.

The individual LL is calculated for a saturated model and the proposed model. The difference between these two person-level log-likelihoods is the individual contribution of that observation to the overall model's Chi-square. As the absolute value of IND_{CHI} increases for an individual observation, that observation is less likely given the proposed model and therefore contributes more to the misfit of the model implied covariance matrix and observed covariance matrix. IND_{CHI} is computed as -2 times the difference between the individual level log-likelihoods of the estimated and saturated models (see equation 2.9). Individuals with large IND_{CHI} values contribute more to the model's misfit.

$$IND_{CHI} = -2\left(P_{LL_{hyp}} - P_{LL_{sat}}\right) \quad (2.9)$$

Reise and Widaman (1999) found that IND_{CHI} was less confounded with individual trait levels than raw individual log-likelihood values, resulting in correlations between the statistic and latent trait scores near zero in simulated data.

IND_{CHI} can be used to identify observations that are not congruous with estimated parameters. For mediation, this statistic has potential as an indicator of observations inconsistent with an estimated mediator because of true effects transmitted through a different mediator. When a direct effect is established between treatment and outcome, it is reasonable to theorize that more than one mediation pathway may connect the two variables and that individuals may have different pathways at work. Given that, if individual observations are inconsistent with the modeled mediation pathway, then it is also reasonable to hypothesize that effects for those individuals may be transmitted from the X to Y through a different mediation pathway.

There is a conceptual difference between an individual for whom a proposed mediation path is not applicable and an individual for whom a different mediation path is applicable. An individual may not conform to a specified mediation model because there is no mediating mechanism at work, rather than having a different mechanism at work. These two scenarios may be distinguished by the total effect of X on Y. In the first case, where an individual has no mediation mechanisms at work, there are two possibilities. Either there is no relation between X and Y, or the change in Y is completely explained by X. The existence of a relation between X and Y can be tested statistically. Whether X can completely explain Y is a matter of theory and relies on subject knowledge expertise and the aggregation of prior evidence. The

current project assumes that if an observation is inconsistent with the estimated mediation mechanism, then there is an alternative, unmodeled mechanism that can, at least partially, explain the relationship observed between X and Y, the independent variable, and the outcome.

In terms of interpreting IND_{CHI} values, "relatively large" negative individual log-likelihoods indicate outliers that are "relatively unlikely" given the proposed model (Reise & Widaman, 1999). Because IND_{CHI} values are -2 times the difference in PLLs, relatively large positive values of IND_{CHI} would suggest a greater contribution to model misfit. Negative values would be interpreted as contributing to model fit (Sterba & Pek, 2012). "Relatively large" and "relatively unlikely" are unhelpful terms for deciding whether any observation is consistent with a specified mediation mechanism. Therefore, one aspect of this project is to investigate whether there are reliably discernable values where individual misfit is suggestive of alternative mediating mechanisms rather than random noise.

CHAPTER 3

TRADITIONAL MEDIATION

Single Mediator Model

The purpose of mediation analysis is to explain how the effect of an independent variable, X, is transmitted to a dependent variable, Y. The causal process or processes that relate X and Y can be described as the effect of X on one or several mediators and the subsequent effect of the mediating variable(s) on Y. A basic model includes a single mediator, M, a single predictor, X, and a single outcome, Y. Equations 2.1-2.3 describe the relationships between these variables through three regression equations. The total effect of X on Y is represented by the c coefficient. The effect of X on M is represented by the a coefficient. If X is a randomized experimental variable and there are no unmeasured confounders, the effect of X on both M and Y will have causal interpretations. The effect of M on Y is represented by the b coefficient, which does not have a causal interpretation even when X is randomized. Causal interpretations of the b-path require additional assumptions regarding the absence of unmeasured confounders of the M to Y relation and added design-based or statistical techniques to strengthen causal interpretations (Valente et al., 2017).

$$Y = i_1 + cX + e_1 \tag{3.1}$$

$$M = i_2 + aX + e_2 \tag{3.2}$$

$$Y = i_3 + c'X + bM + e_3 \tag{3.3}$$

The indirect effect of X on Y that is transmitted through M can be estimated in several ways. Point estimates can be computed as the difference $c - c'$ using coefficients from equations 3.1 and 3.3 or as the product ab using coefficients from equations 3.2 and 3.3. The product of coefficients is the most general method for mediation analysis and therefore is used for the proposed study (MacKinnon et al., 2002). The statistical significance of the ab estimate is generally tested using either a Sobel standard error or confidence limits computed from the asymmetric distribution of the product (MacKinnon et al., 2007; Tofighi & MacKinnon, 2011).

Parallel Mediators

The single mediator model in equations 3.1-3.3 can be expanded to include more than one mediating variable. In a parallel two-mediator model, the indirect effect of a single independent variable X is transmitted to a single outcome variable, Y, through two different mediators that are not directly connected in a causal path ($M1$, $M2$). Equations 3.4-3.6 describe the relationships between the four variables. In the first two equations, the effect of X on each of the mediators is estimated separately. The third equation estimates the outcome using X, $M1$, and $M2$, as predictors.

$$M_1 = i_{M1} + a_1 X + e_{M1} \tag{3.4}$$

$$M_2 = i_{M2} + a_2 X + e_{M2} \tag{3.5}$$

$$Y = i_Y + c'X + b_1 M_1 + b_2 M_2 + e_Y \tag{3.6}$$

Three indirect effects can be estimated in this two-mediator model: two specific indirect effects through each mediator individually and one total indirect effect, which is the sum of the two specific effects. Point estimates of these effects can be computed using the product of coefficients method, where the two specific indirect effects are the estimates $a1b1$ from the coefficients in equations 3.4 and 3.6 and $a2b2$ from the coefficients in equations 3.5 and 3.6. The point estimate for the total indirect effect is the sum of the two products, $a1b1 + a2b2$. The significance of each indirect effect and the equality of the two specific indirect effects can be tested. Finally, adding a second mediator introduces the possibility that the paths that make up the indirect effects can differ in sign and magnitudes in ways that appear to cancel each other out or otherwise complicate the interpretation of the total indirect effect.

Mediation and Moderation

Inferences from mediation analysis generally apply to the population, and for good reason, as there are many applications of mediation that show mechanisms that seem consistent across a population of persons (MacKinnon, 2008). Traditional mediation is a nomothetic approach for investigating causal relations among three or more variables. One of the mathematical assumptions of statistical mediation is that the mediating process is homogeneous in the population. Therefore, estimated mediation effects are assumed to describe uniform processes within a population (MacKinnon, 2008).

However, researchers and clinicians may be interested in making inferences about individual clients or subpopulations of people. Traditional mediation analysis

does not account for mechanisms that may differ among subgroups of a population or between individuals (Collins et al., 1998; Faldowski, 2009; von Eye et al., 2009). Carroll (2021) argued for a personalized medicine approach to studying substance use disorders (SUD) and their treatment, citing heterogeneity in individuals with SUD, the expression of the disorder along several dimensions (e.g., type of substance, severity, comorbidities), and the types of interventions necessary to target a variety of relevant mechanisms of addiction. Similarly, Witkiewitz et al. (2007) and Hsiao et al. (2020) discuss heterogeneity of drinking outcomes following treatment, noting that these observations are not independent but may be conditionally independent based on subgroups of alcohol use disorder types. Similarly, studies of the natural history of some diseases, such as rheumatoid arthritis, suggest multiple possible disease processes that may be distinguished by individual characteristics (Deane & Holers, 2019; Holers et al., 2018).

Moderation is currently the standard method for examining individual differences in mediation. If a population comprises subpopulations that experience different mediation processes, then an individual difference variable can moderate the mediation effect (Baron & Kenny, 1986; Edwards & Lambert, 2007; James & Brett, 1984). Models with concurrent mediators and moderators can address several research questions, such as whether participants experience different processes through which an intervention works and whether mediation explains an interaction effect (Fairchild & MacKinnon, 2009). The first question concerns generalizability and is answered by testing whether the *a*-path, *b*-path, or both depend on a moderator. The second question concerns causality and is answered by testing whether an interaction effect predicts a mediator that, in turn, predicts the outcome.

Adding moderating variables creates a non-linear mediation model that can estimate different mechanisms based on categories of participants. Statistical and conceptual complexities can make interpreting the relevant interactions difficult. Including mediators and moderators in a study requires resources to determine relevant a priori theories, careful attention to the statistical and conceptual complexities of interpreting such a model, and pragmatic concerns related to data collection. The interaction between a mediator and moderator may result in *mediated moderation* (Edwards & Lambert, 2007), where a mediator transmits the interaction effect to the outcome, or *moderated mediation* (James & Brett, 1984), where the effects of the mediator are contingent upon values of the moderator. Morgan-Lopez and MacKinnon (2006) framed this distinction as whether a moderator influenced the *a*-path (mediated moderation) or the *b*-path (moderated mediation).

Baseline values of mediators and outcomes can also be informative moderators. Tien et al. (2004) analyzed real-world intervention data using baseline values of the mediators and outcomes as moderators. The intervention investigated the effects of a parenting program on internalizing and externalizing behaviors in children of divorced parents and found that the program's effects on internalizing problems occurred for a subgroup of children who had poor mother-child relationships before the intervention (Wolchik et al., 2000). Improvement in relationship quality induced by the intervention program mediated the effect. These findings confirmed a hypothesis about the mediators outlined in Wolchik et al. (2000), who illustrated how evidence from individualized mediation analysis could

provide information for improving the efficiency of interventions by identifying populations for whom the program is likely to be most effective.

A great deal of methodological and applied work has been done using models with concurrent mediators and moderators over the last four decades; however, there are still serious limitations to consider. For example, the concurrent effects of mediators and moderators are often assessed using a regression approach (Baron & Kenny, 1986; Edwards & Lambert, 2007; Hayes, 2018; MacKinnon, 2008; Muller et al., 2005; Preacher et al., 2007). However, the regression approach assumes no measurement error in the variables, an assumption that is expected to be violated (Cheung & Lau, 2017).

In addition, there is statistical ambiguity as to whether a variable operates as a mediator or a moderator. No general statistical framework can determine whether a variable addresses causality in a model (i.e., mediator) or generalizability (i.e., moderator). To make this distinction, a researcher must employ theory and critical thinking to determine the function of the variables in question in any dataset (Edwards & Lambert, 2007; MacKinnon, 2008; Muller et al., 2005).

Generalized models with moderation and mediation involve multiple interaction terms, and the power to detect these effects is often low in real data (Fairchild & MacKinnon, 2009; MacKinnon, 2008; Morgan-Lopez & MacKinnon, 2006). However, researchers can improve arguments for moderation effects by considering effect sizes even if the coefficient is not significant. The magnitude of an effect indicates whether a more powerful test (e.g., a larger sample size) might detect the moderation effect, which can inform future studies and replications. Statistical power can also be improved by incorporating a mediational selection

strategy for participant recruitment (Pillow et al., 1991). This method involves screening participants based on baseline values of a target mediator, assuming those with lower values will see greater benefits from the intervention (see Howe, 2019). Participant screening can improve program cost-effectiveness and reduce the potential for iatrogenic effects. Pillow et al. (1991) developed a mediational screening instrument for children of divorce and found that it reliably predicted symptoms of behavioral problems.

A limitation of particular interest is that of defining individual difference variables, which are often related to demographics (e.g., race, sex, class) and are colloquially referred to as "usual suspects" (Howe, 2019). A more individualized approach might include response patterns that cut across a range of observed variables as a categorical moderator rather than natural subgroupings. Configural frequency analysis (von Eye, 1990, 2002) is a method for analyzing response patterns. Previous work on configural frequency mediation (Smyth & MacKinnon, 2021; von Eye et al., 2009; Wiedermann & Von Eye, 2021) can be extended to include the moderation of the mediated effect.

CHAPTER 4

INDIVIDUAL MEDIATION

Individual Mediation Effects

Researchers with hypotheses involving differing mechanisms among subgroups or individuals have limited statistical methods to test individualized mediation. Ignoring individual differences can have consequential effects, especially for research in prevention, medicine, clinical psychology, epidemiology, and public health, where a non-significant effect may obscure iatrogenic treatments (Fairchild & Mackinnon, 2014; MacKinnon, 2011). Inconsistent mediation or suppression effects can manifest if subgroups respond differently to the mediator in a single mediator model or respond to different mediators in a multiple mediator model. For both scenarios, a traditional test of the mediated effect may be non-significant, which can conceal significant but opposing subgroup effects.

In contrast, individual mediation acknowledges that mediating processes may differ across persons, thereby avoiding the ecological fallacy of treating population-level effects as true for individuals. Despite the potential for individual differences in mediating processes, there are few well-studied person-oriented mediation methods in the literature. Recognizing the need for personalized intervention, Howe et al. called for ". . . a flexible framework. . . from genetic moderation of preventative intervention effects to prescriptive implications for prevention" (Howe et al., 2016, p.1). The framework must deal with persistent constraints on individualized inference, such as statistical fallacies. Krull and MacKinnon (1999) pointed out that although statistical methods can evaluate interventions, the clinical purpose of an intervention is usually to effect change on

individuals, not on an aggregated group. However, inferences about subgroups or individuals cannot be made from traditional analysis, as doing so would be an ecological fallacy.

Similarly, Reise and Widaman assert that while a good-fitting statistical model can be descriptive of a portion of the population under some circumstances, "…there simply is no such thing as a universalistic, nomothetically applicable CSA model that adequately represents important psychological phenomena equally well for all individuals in a given population." (1999, p. 4). This echoes Bem and Allen's claim that constructs in the realm of personality psychology apply to "some of the people, some of the time" (Bem & Allen, 1974, p. 506), and Allport's claim that ". . . no two persons ever have precisely the same trait. . . (Allport, 1937, p. 295).

Behavioral research is costly in terms of time, money, and intangible resources, so it is preferable to extract as much information as possible from the collected data (Kraemer et al., 2001; Morgan & Winship, 2007; Tein et al., 2004). Traditional mediation analysis provides researchers with a tool that goes beyond investigating correlated effects to investigate mechanisms, processes, and even causation when appropriate assumptions are met. The ability to extend mediation analysis to estimate individualized effects can further improve the efficiency and effectiveness of research programs.

Individualized mediation could identify either subgroups or individuals who may have mediation processes that differ from the larger sample or population. For example, if there are two different but opposing mechanisms at work within a population, these effects would cancel out in traditional analysis, resulting in the erroneous conclusion that there is no mediation among the variables of interest. An

individualized mediation method could uncover the distinct processes and identify for whom each mediation path is relevant. As Muthén et al. (Muthén et al., 2002) point out, assessing differential intervention effects based on subgroup trajectories can lead to more effective intervention design strategies. Applications such as personalized medicine, clinical psychology, public health programs, and adaptive intervention programs could improve outcomes for more people. Additionally, publicly funded programs would be more effective and efficient while improving the information available for policy-making decisions.

There are robust bodies of research on both mediation analysis and individual differences. However, there is a noticeable gap in knowledge concerning individual differences in mediated effects. Only a handful of papers have been found that tackle the subject (Faldowski, 2009; Geuke et al., 2019; MacKinnon et al., 2022; Smyth & MacKinnon, 2021; von Eye et al., 2009; Wiedermann & Von Eye, 2021). By providing a better understanding of individualized mediation effects, psychological and behavioral research in social science will be improved practically by having more information to design better programs and theoretically by providing more information to refine causal theories. Ignoring individualized mediation effects can lead to unwanted results, such as concluding that there is no mediated effect when there may be multiple mechanisms at work.

A common assumption of the linear mediation model is that the causal mechanism is homogeneous in the population. However, the population might include subpopulations that experience different mediation processes. If these subpopulations are defined by a measurable individual difference variable, such as a demographic category, then a traditional moderation analysis may be sufficient to

test and describe heterogeneity in the mediated effect. However, it may be the case that individuals who respond differentially to mediating processes may not be so easily clustered together.

Combining Individual Fit and Individual Mediation

The model residuals and individual fit measures described in Chapter 1 are intended to signal when individual observations may have an outlying influence on the results of a statistical model. When one subpopulation has a mediated effect and another does not, differences in individual fit measures are expected depending on whether or not that mediator is included in the model. A scenario where a true mediator is left out of a model does not suggest that any observations would rise to the level of being an *influential* variable based on its true mediated effect. Therefore, a direct application of these methods for identifying individual mediation is unlikely to bear interesting fruit.

However, this dissertation proposes a novel use for these methods that involves studying the differences in residuals and fit measures in a simulation study where there are known differences in mediated effect within the sample. The logic of this new application rests in the difference of the values of residuals and individual fit measures in a series of models in which various mediation effects are estimated using the same dataset. One aim of this study is to investigate how the magnitude, direction, and significance of the difference in residuals is influenced when the true mediated effects in the sample are heterogeneous, and there is variation in whether all the pertinent mediators are specified in the model.

CHAPTER 5

PILOT STUDY

Methods

A pilot study was conducted to obtain preliminary information about the methods for individual-level mediation useful for a proposed simulation. The pilot study explored if residuals provide information about which mediated effect corresponds to individual observations when two different mediating processes are present in a population.

The methods under investigation in the pilot study include comparisons of residual direction and magnitude when mediators are and are not included in the estimated model. These comparisons were made with raw and standardized residuals (i.e., delta z statistic and generalized Cook's distance.) An individual likelihood-based method was also examined.

A general aim of the pilot study was to demonstrate that the methods were promising under ideal conditions first before investigating more fine-grained conditions in the proposed study. To this end, extremely large effect sizes and a large sample size were specified. The results below demonstrate that each method showed promise for individual-level mediated effects and provided motivation for evaluating the methods with effect sizes more commonly observed with real data.

Data Generation

Data for one population condition were generated using SAS software, Version 9.4 of the SAS system for Windows. Variables X, $M1$, $M2$, and Y were generated using the RAND function and random seed 1920201. The X variable was

generated as a continuous variable from $N(0,1)$. $M1$, $M2$, and Y were generated according to equations 5.1-5.3. In these equations, i_{M1}, i_{M2}, and i_Y are intercepts and have been set to 0; e_{M1}, e_{M2}, and e_Y are errors; the a-paths represent the effects of X on $M1$ and $M2$; the b-paths represent the effects of $M1$ and $M2$ on Y while controlling for X; and the c'-path represents the direct effect of X on Y, controlling for $M1$ and $M2$.

$$M_1 = i_{M1} + a_1 X + e_{M1} \tag{5.1}$$

$$M_2 = i_{M2} + a_2 X + e_{M2} \tag{5.2}$$

$$Y = i_Y + c'X + b_1 M_1 + b_2 M_2 + e_Y \tag{5.3}$$

A sample of N=1500 observations was generated from a population model with two parallel mediators and was made up of three subpopulations of equal size. The population c' path equaled 0 for all three subpopulations. The paths for $a1$ and $b1$ were equal, as were $a2$ and $b2$. For each of the two subpopulations, the true indirect effect of the independent variable was transmitted through only one of the mediators. For the first subgroup, $a1 = b1 = 9$, and $a2 = b2 = 0$. In the second subgroup, $a1 = b1 = 0$, and $a2 = b2 = 9$. The third subgroup data were generated to have no relationships between X, $M1$, $M2$, or Y. Data for the subpopulations were generated as separate files and then combined into a single data file for analysis.

The true parameter value of 9 for a and b-path effects and 81 for the indirect effects ($a1b1$ and $a2b2$) were intentionally made extremely large to examine whether the methods under investigation are at least conceptually viable from first principles. One goal of the full simulation is to ascertain whether these methods are

viable under conditions more like actual psychological and public health data. A summary of true coefficient values for this condition is in table 1.

Analysis

Index Plots

Within the dataset, observations 1-500 had a true mediated effect through paths $a1$ and $b1$, observations 501-1000 had a true mediated effect through paths $a2$ and $b2$, and observations 1001-1500 had no mediated effect. Regression equations were estimated and predicted Y values saved for three models: a single mediator model through $M1$, a single mediator model through $M2$, and a two-mediator model through both mediators. The observed and predicted values of Y from each model were plotted by observation number.

Raw Residuals

The data were analyzed using Models 0-3, described below (see equations 5.4, 5.6-5.7, 5.9-5.10, and 5.12). Residuals were calculated for individual observations (see equations 5.5, 5.8, 5.11, and 5.13). For each of the 1500 observations in the sample, four separate residual values were computed and labeled e_{ji} for $j = 0, 1, 2, 3$ and $i = 1, 2, \ldots, n$. The subscript j is the index for the four models, while subscript i is the index for individual observations. In the following equations, Y_{ji} is the observed value of Y of model j for observation i; $\hat{Y}_j$ is the predicted mean of Y for model j; n is the number of observations in the sample; e_{ji} is the residual for individual i and model j; M_1 is the first mediator; M_2 is the second mediator; X is the predictor.

Model 0 is the mean of Y across all observations in each dataset, as in equation 5.4. Residual 0 is calculated for each observation as the difference between observed Y and the mean of Y, as in equation 5.5.

$$\hat{Y}_0 = \frac{\sum_1^i Y}{n} \tag{5.4}$$

$$e_{0i} = Y_{0i} - \bar{Y}_0 \tag{5.5}$$

Model 1 is a single mediator model through mediator 1 ($M1$) estimated with equations 5.6 and 5.7. Residual 1 is calculated for each observation in equation 5.8 as the difference between observed Y and the predicted Y from equation 5.7.

$$\widehat{M}_1 = i_{M1} + a_1 X \tag{5.6}$$

$$\hat{Y}_1 = i_{Y1} + c'X + b_1 M_1 \tag{5.7}$$

$$e_{1i} = Y_{1i} - \hat{Y}_1 \tag{5.8}$$

Model 2 is a single mediator model through mediator 2 ($M2$) estimated with equations 5.9 and 5.10. Residual 2 is calculated for each observation in equation 5.11 as the difference between observed Y and the predicted Y from equation 5.10.

$$\widehat{M}_2 = i_{M2} + a_2 X \tag{5.9}$$

$$\hat{Y}_2 = i_{Y2} + c'X + b_2 M_2 \tag{5.10}$$

$$e_{2i} = Y_{2i} - \hat{Y}_2 \tag{5.11}$$

Model 3 is a two-mediator model through both mediators estimated with equations 5.6, 5.9, and 5.12. Residual 5 is calculated for each observation in equation 5.13 as the difference between observed Y and the predicted Y from equation 5.12.

$$\hat{Y}_3 = i_{Y3} + c'X + b_1 M_1 + b_2 M_2 \tag{5.12}$$

$$e_{3i} = Y_{3i} - \hat{Y}_3 \tag{5.13}$$

Delta z

A Sobel test for the indirect effect adapted from methods described in Pek and MacCallum (delta z; 2011) was conducted using 1500 jackknife replicates of (n=1499) computed by systematically removing one observation at a time. A two-mediator model was estimated in PROC CALIS for the full sample (n=1500) and a full set of jackknife samples (n=1499 each). Estimates were computed for the mediated effect through $M1$ ($a1b1$), the mediated effect through $M2$ ($a2b2$), the total mediated effect ($a1b1 + a2b2$) and the difference between the mediated effects ($a1b1 - a2b2$).

The Sobel z statistic was computed for estimates in the complete N=1500 sample according to equations 5.14-5.17. In these equations, $a1b1$ and $a2b2$ are mediated effects through $M1$ and $M2$; $a1$ and $a2$ are the effects of X on $M1$ and $M2$; $b1$ and $b2$ are the effects of $M1$ and $M2$ on Y controlling for X; $s^2_{\hat{a}_1}$ and $s^2_{\hat{b}_1}$ are the variances of $a1$ and $b1$; $s^2_{\hat{a}_2}$ and $s^2_{\hat{b}_2}$ are the variances of $a2$ and $b2$; and $s_{\widehat{a_1}\widehat{a_2}}$ and $s_{\widehat{b_1}\widehat{b_2}}$ are the covariances for the a and b-paths.

$$z_{a1b1} = \frac{\widehat{a_1 b_1}}{\sqrt{\widehat{a_1}^2 s^2_{\widehat{b_1}} + \widehat{b_1}^2 s^2_{\widehat{a_1}}}} \tag{5.14}$$

$$z_{a2b2} = \frac{\widehat{a_2 b_2}}{\sqrt{\widehat{a_2}^2 s^2_{\widehat{b_2}} + \widehat{b_2}^2 s^2_{\widehat{a_2}}}} \tag{5.15}$$

$$z_{a1b1+a2b2} = \frac{\widehat{a_1 b_1} + \widehat{a_2 b_2}}{\sqrt{\widehat{a_1}^2 s^2_{\widehat{b_1}} + \widehat{b_1}^2 s^2_{\widehat{a_1}} + \widehat{a_2}^2 s^2_{\widehat{b_2}} + \widehat{b_2}^2 s^2_{\widehat{a_2}} + 2\widehat{a_1}\widehat{a_2} s_{\widehat{b_1}\widehat{b_2}} + 2\widehat{b_1}\widehat{b_2} s_{\widehat{a_1}\widehat{a_2}}}} \tag{5.16}$$

$$z_{a1b1-a2b2} = \frac{\widehat{a_1 b_1} - \widehat{a_2 b_2}}{\sqrt{\widehat{a_1}^2 s^2_{\widehat{b_1}} + \widehat{b_1}^2 s^2_{\widehat{a_1}} + \widehat{a_2}^2 s^2_{\widehat{b_2}} + \widehat{b_2}^2 s^2_{\widehat{a_2}} - 2\widehat{a_1}\widehat{a_2} s_{\widehat{b_1}\widehat{b_2}} - 2\widehat{b_1}\widehat{b_2} s_{\widehat{a_1}\widehat{a_2}}}} \quad (5.17)$$

The difference in the Sobel statistic between each jackknife replicate and the complete sample was computed for each estimate according to equations 5.18-5.21. In these equations, the subscript *i* refers to the jackknife sample in which observation *i* has been removed.

$$\Delta z_{a1b1} = \frac{\widehat{a_1 b_1} - \widehat{a_{1i} b_{1i}}}{\sqrt{\widehat{a_1}^2 s^2_{\widehat{b_1}} + \widehat{b_1}^2 s^2_{\widehat{a_1}}}} \quad (5.18)$$

$$\Delta z_{a2b2} = \frac{\widehat{a_2 b_2} - \widehat{a_{2i} b_{2i}}}{\sqrt{\widehat{a_2}^2 s^2_{\widehat{b_2}} + \widehat{b_2}^2 s^2_{\widehat{a_2}}}} \quad (5.19)$$

$$\Delta z_{a1b1+a2b2} = \frac{(\widehat{a_1 b_1} + \widehat{a_2 b_2}) - (\widehat{a_{1i} b_{1i}} + \widehat{a_{2i} b_{2i}})}{\sqrt{\widehat{a_1}^2 s^2_{\widehat{b_1}} + \widehat{b_1}^2 s^2_{\widehat{a_1}} + \widehat{a_2}^2 s^2_{\widehat{b_2}} + \widehat{b_2}^2 s^2_{\widehat{a_2}} + 2\widehat{a_1}\widehat{a_2} s_{\widehat{b_1}\widehat{b_2}} + 2\widehat{b_1}\widehat{b_2} s_{\widehat{a_1}\widehat{a_2}}}} \quad (5.20)$$

$$\Delta z_{a1b1-a2b2} = \frac{\left(\widehat{a_1 b_1} - \widehat{a_2 b_2}\right) - \left(\widehat{a_{1i} b_{1i}} - \widehat{a_{2i} b_{2i}}\right)}{\sqrt{\widehat{a_1}^2 s^2_{\widehat{b_1}} + \widehat{b_1}^2 s^2_{\widehat{a_1}} + \widehat{a_2}^2 s^2_{\widehat{b_2}} + \widehat{b_2}^2 s^2_{\widehat{a_2}} - 2\widehat{a_1}\widehat{a_2} s_{\widehat{b_1}\widehat{b_2}} - 2\widehat{b_1}\widehat{b_2} s_{\widehat{a_1}\widehat{a_2}}}} \quad (5.21)$$

gCd

Delta *z* is useful for determining the direction of change in parameter estimates. At the same time, generalized Cook's distance (gCd) measures the absolute change in magnitude of parameter estimates when case *i* is deleted from the sample. Equation 2.5 shows gCd computed as a function of the parameter estimates from the full sample and delete-one samples and the covariances of the

parameter estimates. In the matrix formula, $\hat{\theta}$ is a vector of parameter estimates from the full sample, $\hat{\theta}_i$ is a vector of estimates from each jackknife sample, and $\widehat{Var(\hat{\theta}_i)}$ is a covariance matrix of parameter estimates from the jackknife sample.

Delta *z* and gCd are closely related as gCd can be computed as the square of delta *z*. The gCd statistic is bounded by zero. A small value shows that excluding observation *i* leads to small changes in the parameter estimate. Larger values suggest that excluding that observation leads to a larger change in the parameter estimate.

Individual Chi-Square

IND_{CHI} is defined as -2 times the difference between the individual log-likelihood computed in the hypothetical model and the PLL computed in a saturated model, as shown in equation 2.8 (PLL; Lange et al., 1976). In equation 2.8, *p* is a vector of variables, x_i is a vector of responses for individual *i*, and *M* is a vector of sample means. The first part of the formula is a constant that applies to all the observations in a sample, while the second part is the Mahalanobis squared distance for the individual.

IND_{CHI} measures an individual observation's contribution to a structural model's Chi-square value. The PLL of the saturated model is treated as the expected value for that person (which parallels the logic of comparisons in configural frequency mediation). Large IND_{CHI} values indicate an observation that contributes more to the overall misfit of the model. In the simulated data, it is known that some observations have an effect through an unmodeled mediator. Therefore, it is expected that these observations would contribute to the misfit of the model.

Results

Index Plots for Observed and Predicted Y

The index plot suggested that there was more variability in observed Y for the first 1000 observations than the last 500, showing that observed values of Y appeared to have more variability around the mean when a mediator was included in the data generation process (see panels 1a.-3a. of figure 1). This pattern is expected.

There was more variability in predicted Y for the first 500 observations when $M1$ was modeled, the second 500 observations when $M2$ was modeled, and the first 1000 observations when both $M1$ and $M2$ were modeled. Predicted values of Y had more variability when the modeled mediator matched the mediator in the data generation process.

Raw Residuals

The residuals from Models 0-3 were saved for each observation. Model 0 was the difference between observed Y and the mean of Y with no predictors. Model 1 was a single mediator model through $M1$. Model 2 was a single mediator model through $M2$. Model 3 was a two-mediator model. For individual observations, the residuals from each model can be compared. For any given observation, the size of the residual should describe how well that model predicts for that individual.

Visual comparisons can be made using the index plots in figure 2. First, it was predicted that if someone has no mediated effect ($S3$) then the residuals would be the same across all four models. However, looking at the last third of the observations shows that the residuals for this subpopulation are only similar when

either $M1$ or $M2$ is included in the model (panels 2 and 3). For someone with effects through $M1$ ($S1$), it was predicted that residuals in Model 2 would be higher than residuals for someone with effects through $M2$ ($S2$), which is seen by comparing subpopulations 1 and 2 in panel 3. Likewise, it was expected that the residuals for $S1$ would be similar in Models 1 and 3, which are the two models in which $M1$ was modeled, which is seen by comparing $S1$ in panels 2 and 4.

Because the magnitude of effects through $M1$ and $M2$ were held constant, it was expected that effects for $S2$ would mirror those seen for $S1$. For example, in a comparison of subpopulations 1 and 2 in panel 2, residuals for individuals with the unmodeled mediator are larger on average ($S2$) than those with the modeled mediator ($S1$). The mean values of model residuals are summarized in table 2.

Delta z

It was expected that for observations with a larger effect through $M1$, the difference between $a1b1$ in the full sample and the jackknife sample that has the observation deleted would be larger than the difference between $a2b2$ in the full sample and jackknife sample. Similarly, the discrepancy for $a2b2$ would be greater for observations with a larger effect through $M2$. The plots in figure 3 show that the discrepancies in $a1b1$ (panel A) and $a2b2$ (panel B) differ for observations that have larger effects through $M1$ or $M2$, respectively. Table 2 also summarizes the mean values of delta z for subpopulations 1 and 2, which shows a similar pattern of positive values of delta z for mediated effects through the "correct" mediator (i.e., $M1$ for $S1$ and $M2$ for $S2$), and negative values for mediated effects through the

"incorrect" mediator (i.e., $M2$ for $S1$ and $M1$ for $S2$). The absolute values of delta z appear to be similar in magnitude for both mediated effects in both subpopulations.

gCd

It was expected that removing observations that had mediated effects corresponding to the modeled mediator would lead to larger changes in parameter estimates and, therefore, larger gCd values. For example, removing observations from $S1$ would lead to larger gCd values than removing observations from $S2$ when $M1$ was the only modeled mediator. Table 2 also summarizes the mean gCd values for each subpopulation and is suggestive of the expected pattern, although the range of mean gCd values is narrow.

Figure 4 shows the subpopulation with no mediated effects ($S3$) has gCd values more like the subpopulation with an unmodeled mediated effect (i.e., $S2$ in panel A and $S1$ in panel B). However, $S3$ appears to have less variability. Observations with large effects on the unmodeled mediator (i.e., $S2$ in panel A and $S1$ in panel B) have, on average, smaller gCd values than observations with large mediated effects on the modeled mediator (See table 2). Visual inspection of figure 4 suggests the mean gCd in the subpopulation with the unmodeled mediator may be driven by a few influential values.

A comparison of gCd and delta z in table 2 shows that the discrepancies between $S1$ and $S2$ have similar absolute magnitudes in both measures; however, they have opposite signs in delta z. This difference in sign is obscured in gCd because the statistic is bounded by zero. Although the magnitudes of difference are

very small in the context of statistical testing, the pattern of values and variability related to whether mediated effects are correctly modeled appears to be consistent.

Individual Chi-Square

A two-mediator model was estimated in Mplus, and PLL values were saved. The PLL values summed to the model log-likelihood (LL = -14125.134) of the two-mediator model. Because the two-mediator model is the proper data generation model, the PLLs from this model were used as the saturated individual likelihoods ($P_{LL_{sat}}$) for the IND_{CHI} formula in equation 2.9. A single mediator model through $M1$ was also estimated, and the PLL values were saved. These values sum to the model log-likelihood (LL = -11792.686) of the single mediator model and functioned as the hypothesized individual likelihoods ($P_{LL_{hyp}}$) for the IND_{CHI} formula in equation 2.9 above. Figure 5 shows that in this limited examination, a visual distinction can be made between the subpopulation with no mediation effects ($S3$) and the other two subpopulations. However, there is not a visible difference between $S1$, which has a true effect through the modeled mediator ($M1$), and $S2$, which has a true effect through an unmodeled mediator ($M2$). The mean IND_{CHI} values by subpopulation are summarized in table 2.

The difference in $S3$ suggests that IND_{CHI} has the potential to distinguish whether there is a mediated effect or not for a given observation. However, more work needs to be done to determine a method for computing IND_{CHI} values between models to distinguish between observations that have similarly sized effects on different mediators.

Summary of Pilot Results

The pilot study revealed several results that inform the proposed simulation. First, residuals for observations with no mediated effect were smallest in a model with no predictors and largest when one mediator was included in the model. Residuals were smaller for observations with effects through $M1$ compared to observations for effects through $M2$ when only $M1$ was modeled. A parallel result was seen for observations with effects through $M2$ when only $M2$ was included in the model. Residuals were visually indistinguishable between subpopulations when both mediators were included in the model.

Second, it was observed that delta z values were positive in observations whose true mediated effect matched the estimated specific mediated effect. However, delta z was negative for observations whose true mediated effect did not match the estimated specific mediated effect. In addition, there appeared to be more variability in delta z when it was positive. Third, a pattern was seen with gCd, such that the mean value of the statistic was larger in the subpopulation that had true mediated effects corresponding to the modeled mediator; however, the range of values was narrow, and the difference between groups was not statistically tested. Finally, in an index plot of IND_{CHI} values, $S3$ was visually distinct, but no difference could be seen between observations with effects through $M1$ and those with effects through $M2$.

This pilot study shows the feasibility of a simulation design comparing four models with different specified mediators. Encouraging results were achieved when investigating raw residuals and delta z, implying these two methods have the most promise for indicating which observations have effects through a given mediator. Because delta z and gCd are algebraically related, it is expected these two measures

would lead to similar conclusions in a simulation; however, because gCd is constrained to positive values, it may provide less information than delta *z*. Finally, IND_{CHI} has the potential to distinguish whether or not there is a mediated effect for a given observation. Still, the pilot study suggests this statistic may be less useful in distinguishing which mediator is at work in a given observation.

CHAPTER 6

METHODS

Overview

A Monte Carlo simulation study investigated whether individual mediation effects can be identified using model residuals. Previous work with configural frequency mediation has used statistically significant differences between expected and observed frequencies to investigate response patterns consistent with mediation in categorical data (Smyth & MacKinnon, 2021; von Eye et al., 2009; Wiedermann & Von Eye, 2021). The current study extends this logic to continuous data by substituting the difference in cell counts with comparisons of residuals and local fit measures across several mediation models. The study's primary aim is to investigate whether any of the methods in question can identify which of two mediation processes is at work for a given observation. The following terminology is used to distinguish between the nested series of pairwise differences that form the study's design. First, there are differences between analysis models, referred to as *Model Contrasts*. Second, there are differences between known subpopulations, referred to as *Subpopulation Contrasts*. For each individual fit measure (i.e., residuals, delta *z,* and gCd), there are six possible Model Contrasts (see equations 6.26-6.43) and six possible Subpopulation Contrasts for a total of 36 possible combinations. However, not all combinations are of theoretical interest, and some of the combinations are redundant. Therefore, this study will focus on Model Contrasts between a model with no predictors (Model 0) and models with effects through Mediator 1 (Model 1) or effects through Mediator 2 (Model 2).

Here is a general overview of the steps of the simulation study, with further details on each step below. First, data were generated from four known subpopulations with different true mediated effects. Second, individual fit methods (i.e., residuals, delta z, and gCd) were estimated for four analysis models with differing mediated effects that match the four known subpopulations. Third, six pairwise Model Contrasts of residuals, delta z, and gCd were computed across the four analysis models, as shown in table 4. The dependent variables for the simulation were the contrasts for differences in residuals (i.e., $\delta 1$- $\delta 6$), differences in delta z (i.e., $\delta deltaz1$- $\delta deltaz6$), and differences in gCd (i.e., $\delta gCd1$- $\delta gCd6$). Fourth, ANOVAs were conducted to assess known subpopulation differences in Model Contrasts for each individual fit method. Outcomes included raw values and rank-orders of the Model Contrasts.

Data Generation

A Monte Carlo simulation was conducted using SAS software, Version 9.4 of the SAS system for Windows. Data were generated from a population model with two parallel mediators using the RAND function. Data for the subpopulations were generated as separate files with random seeds of 19800303, 19960303, 20010303, and 20220303. The four data files were combined into a single file for analysis with the proposed methods. The X variable was generated as a continuous variable from $N(0,1)$. $M1$, $M2$, and Y were generated according to equations 6.1-6.3 with a normally distributed residual. In these equations, i_{M1}, i_{M2}, and i_Y are intercepts and have been set to 0; e_{M1}, e_{M2}, and e_Y are errors; the a-paths represent the effects of X on $M1$ and $M2$; the b-paths represent the effects $M1$ and $M2$ on Y while controlling for X. The c'-

path represents the direct effect of X on Y, controlling for $M1$ and $M2$, and was set to zero for all conditions and subpopulations in the simulation study.

$$M_1 = i_{M1} + a_1 X + e_{M1} \quad (6.1)$$

$$M_2 = i_{M2} + a_2 X + e_{M2} \quad (6.2)$$

$$Y = i_Y + c'X + b_1 M_1 + b_2 M_2 + e_Y \quad (6.3)$$

Figure 6 shows a path diagram for the data generating model. Each generated population consisted of four subpopulations of equal size ($S1$-$S4$). Each subpopulation was 25%of the total population and was generated with a different mediation model by setting parameters in equations 6.1, 6.2, and 6.3 to be zero or nonzero. In each generated population, 25% percent of the sample had a true mediated effect through only $M1$ ($S1$), 25% had a true mediated effect through only $M2$ ($S2$), 25% had true mediated effects through both mediators ($S3$), and the final 25% had no associations among the variables ($S4$) (see table 3). In summary, the two-mediator model was used to generate all four subpopulations with population coefficients constrained to zero to reflect subpopulations with different mediating processes. The data generation program is in Appendix C.

Independent Variables

Two samples size were tested (200, 1000) for residual differences. Due to computational complexity, only the smaller sample size was tested for delta *z* and gCd. These sample sizes represent sizes typically used in psychological research while accommodating four equal-sized subpopulations in each sample. The size of each subgroup was one-fourth the overall sample size (50, 250). The magnitude of path coefficients also varied. Let the subscript *g*, where $g = 1\text{-}4$, represent the subpopulations that make up each dataset. Each subpopulation was defined by five

paths, $a1_g, a2_g, b1_g, b2_g,$ andc'_g. Therefore, each population condition was defined by a combination of 20 path coefficients (i.e., five paths for each of the four subpopulations). As described in the data generation section, approximately 73% of the parameters were constrained to zero. Three effect sizes were simulated for the coefficients of the a and b paths (0, .39, .99). These values approximate zero, and medium effects as defined by Cohen (1988), and an extra-large effect of .99 following the results of the pilot study that suggested that large effects may be needed for the proposed methods. These effect sizes were chosen to investigate the feasibility of the concepts proposed in the simulation across a reasonable range of effects. Corresponding a and b paths were equal in each subpopulation condition (i.e., $a_{1g} = b_{1g}$, and $a_{2g} = b_{2g}$). The c' paths were all equal to 0. There were 162 simulation conditions generated and replicated 500 times each for a total of 81,000 datasets and 48.6 million observations.

Analysis Models

Four analysis models were estimated with the simulated datasets: a model with no predictors (Model 0), a single-mediator through Mediator 1 (Model 1), a single-mediator model through Mediator 2 (Model 2), and a two-mediator model through both Mediators (Model 3). The estimated mediators for each model are summarized in table 5. Let the subscript j index the four models while subscript i is the index for individual observations. In the following equations, Y_i is the observed value of Y for observation i; $\hat{Y}_{ji}$, $\widehat{M1}_{ji}$, and $\widehat{M2}_{ji}$ are the predicted values of Y, $M1$, and $M2$ for model j and individual i, n is the number of observations in the sample;

$M1$ is the first mediator; $M2$ is the second mediator; X is the predictor, and intercepts are denoted with a normal sized *i*.

Model 0 serves as a baseline and is the mean of Y across all observations in each dataset, as in equation 6.4.

$$\hat{Y}_{0i} = \frac{\sum_1^i Y_i}{n} \tag{6.4}$$

Model 1 is a single mediator model through Mediator 1 ($M1$) estimated with equations 6.5 and 6.6.

$$\widehat{M1}_{1i} = i_{M1} + a_1 X_i \tag{6.5}$$

$$\hat{Y}_{1i} = i_{Y1} + c' X_i + b_1 M1_i \tag{6.6}$$

Model 2 is a single mediator model through Mediator 2 ($M2$) estimated with equations 6.7 and 6.8.

$$\widehat{M2}_{2i} = i_{M2} + a_2 X_i \tag{6.7}$$

$$\hat{Y}_{2i} = i_{Y2} + c' X_i + b_2 M2_i \tag{6.8}$$

Model 3 is a two-mediator model through both mediators estimated with equations 6.5, 6.7, and 6.9.

$$\hat{Y}_{3i} = i_{Y3} + c' X_i + b_1 M1_i + b_2 M2_i \tag{6.9}$$

Individual Fit Methods

Residuals

Each dataset was analyzed using the four models described above. For each observation, four residuals were calculated and labeled e_{ji} for $j = 0, 1, 2, 3$ and $i = 1, 2, \ldots, n$. Residual 0 (e_{0i}) is calculated as the difference between observed Y for individual i and the mean of Y with no predictors, as in equation 6.10.

$$\widehat{e_{0i}} = Y_i - \hat{Y}_0 \tag{6.10}$$

Residual 1 (e_{1i}) is calculated in equation 6.11 as the difference between observed Y for individual i and the predicted Y from equation 6.6.

$$\widehat{e_{1i}} = Y_i - \hat{Y}_{1i} \tag{6.11}$$

Residual 2 (e_{2i}) is calculated in equation 6.12 as the difference between observed Y for individual i and the predicted Y from equation 6.8.

$$\widehat{e_{2i}} = Y_i - \hat{Y}_{2i} \tag{6.12}$$

Residual 3 (e_{3i}) is calculated in equation 6.13 as the difference between observed Y for individual i and the predicted Y from equation 6.9.

$$\widehat{e_{3i}} = Y_i - \hat{Y}_{3i} \tag{6.13}$$

Delta *z*

Jackknife samples were computed for each dataset by iteratively deleting one observation *(i)* and saving the *(n – i)* sample. The four analysis models in equations 6.4 – 6.9 were estimated using the jackknife samples and estimates for the mediated effect through $M1$ (a_1b_1), the mediated effect through $M2$ (a_2b_2), and the total mediated effect ($a_1b_1 + a_2b_2$) were saved for the full sample and each jackknife sample. The Sobel test statistic was computed for each of the mediated effect estimates in the full sample according to equations 6.14-6.17.

$$z_{\hat{Y}_0} = \frac{\widehat{e_{0i}}}{\sigma_{\hat{Y}_0}} \tag{6.14}$$

$$z_{a1b1} = \frac{\widehat{a_1 b_1}}{\sqrt{\widehat{a_1}^2 s^2_{\widehat{b_1}} + \widehat{b_1}^2 s^2_{\widehat{a_1}}}} \tag{6.15}$$

$$z_{a2b2} = \frac{\widehat{a_2 b_2}}{\sqrt{\widehat{a_2}^2 s^2_{\widehat{b_2}} + \widehat{b_2}^2 s^2_{\widehat{a_2}}}} \tag{6.16}$$

$$z_{a1b1+a2b2} = \frac{\widehat{a_1 b_1} + \widehat{a_2 b_2}}{\sqrt{\widehat{a_1}^2 s^2_{\widehat{b_1}} + \widehat{b_1}^2 s^2_{\widehat{a_1}} + \widehat{a_2}^2 s^2_{\widehat{b_2}} + \widehat{b_2}^2 s^2_{\widehat{a_2}} + 2\widehat{a_1}\widehat{a_2} s_{\widehat{b_1}\widehat{b_2}} + 2\widehat{b_1}\widehat{b_2} s_{\widehat{a_1}\widehat{a_2}}}} \tag{6.17}$$

The change in z attributable to individual observations was computed by comparing estimates for the full sample with each jackknife sample as in equations 6.18 - 6.21.

$$\Delta z_{\hat{Y}_0} = \frac{\widehat{e_{0i}} - \widehat{e_{0ii}}}{\sigma_{\hat{Y}_{0i}}} \tag{6.18}$$

$$\Delta z_{a1b1} = \frac{\widehat{a_1 b_1} - \widehat{a_{1i} b_{1i}}}{\sqrt{\widehat{a_{1i}}^2 s^2_{\widehat{b_{1i}}} + \widehat{b_{1i}}^2 s^2_{\widehat{a_{1i}}}}} \tag{6.19}$$

$$\Delta z_{a2b2} = \frac{\widehat{a_2 b_2} - \widehat{a_{2i} b_{2i}}}{\sqrt{\widehat{a_{2i}}^2 s^2_{\widehat{b_{2i}}} + \widehat{b_{2i}}^2 s^2_{\widehat{a_{2i}}}}} \tag{6.20}$$

$$\Delta z_{a1b1+a2b2} = \frac{(\widehat{a_1 b_1} + \widehat{a_2 b_2}) - (\widehat{a_{1i} b_{1i}} + \widehat{a_{2i} b_{2i}})}{\sqrt{\widehat{a_{1i}}^2 s^2_{\widehat{b_{1i}}} + \widehat{b_{1i}}^2 s^2_{\widehat{a_{1i}}} + \widehat{a_{2i}}^2 s^2_{\widehat{b_{2i}}} + \widehat{b_{2i}}^2 s^2_{\widehat{a_{2i}}} + 2\widehat{a_{1i}}\widehat{a_{2i}} s_{\widehat{b_{1i}}\widehat{b_{2i}}} + 2\widehat{b_{1i}}\widehat{b_{2i}} s_{\widehat{a_{1i}}\widehat{a_{2i}}}}} \tag{6.21}$$

gCd

Pek and MacCallum (2011, p. 207) showed gCd could be expressed in terms of $\Delta\theta$; therefore, gCd values for each model were computed by squaring the delta z values above, as in equations 6.22-6.25.

$$gCd_{\hat{Y}_0} = \left(\Delta z_{\hat{Y}_{0i}}\right)^2 \tag{6.22}$$

$$gCd_{a1b1} = (\Delta z_{a1b1})^2 \quad (6.23)$$

$$gCd_{a2b2} = (\Delta z_{a2b2})^2 \quad (6.24)$$

$$gCd_{a1b1+a2b2} = (\Delta z_{a1b1+a2b2})^2 \quad (6.25)$$

Dependent Variables

The raw and rank-order values of the six Model Contrasts between the individual fit measures were the dependent variables in the analysis of the simulation results. There are six possible pairwise Model Contrasts among Models 0-3, shown in table 4. The first contrast compares the independent model and a mediator model through Mediator 1. The second contrast compares a model through Mediator 1 and a model through Mediator 2. The third contrast compares a model through Mediator 1 and a two-mediator model through Mediator 1 and Mediator 2. The fourth contrast compares the independent model and a model through Mediator 2. The fifth contrast compares a model through Mediator 2 with a two-mediator model. The final contrast compares the independent model and a two-mediator model. These six Model Contrasts were made for the three individual fit measures: regression residuals, delta z, and gCd.

Residual Model contrasts

The six pairwise Model Contrasts of residuals from Models 0-3 are shown in equations 6.26-6.31. In the first three Model Contrasts (δ1- δ3), residuals from a model with effects through Mediator 1 (equation 6.6) are compared to residuals from a model with no predictors (equation 6.4), a model with effects through Mediator 2 (equation 6.8), and the two-mediator model (equation 6.9).

$$\delta 1 = e_{0i} - e_{1i} \quad (6.26)$$

$$\delta 2 = e_{1i} - e_{2i} \tag{6.27}$$

$$\delta 3 = e_{1i} - e_{3i} \tag{6.28}$$

The next two Model Contrasts (δ4 and δ5) compare the residuals from a model with effects through Mediator 2 (equation 6.8) with residuals from a model with no predictors (equation 6.4) and the two-mediator model (equation 6.9).

$$\delta 4 = e_{0i} - e_{2i} \tag{6.29}$$

$$\delta 5 = e_{2i} - e_{3i} \tag{6.30}$$

The final Model Contrast (δ6) is between the residuals from a model with no predictors (equation 6.4) and the two-mediator model (equation 6.9).

$$\delta 6 = e_{0i} - e_{3i} \tag{6.31}$$

Delta *z* Model Contrasts

The Model Contrasts of delta *z* in equations 6.32-6.34 mirror the residual differences computed in equations 6.26-6.31. In the first three Model Contrasts, delta *z* for the mediated effect through Mediator 1 (a_1b_1) is compared to delta *z* for a model with no predictors (equation 6.32), delta *z* for the mediated effect through Mediator 2 (a_2b_2) (equation 6.33), and delta *z* for the total mediated effect ($a_1b_1 + a_2b_2$) (equation 6.34).

$$\delta deltaz1 = \Delta z_{\hat{Y}_0} - \Delta z_{a1b1} \tag{6.32}$$

$$\delta deltaz2 = \Delta z_{a1b1} - \Delta z_{a2b2} \tag{6.33}$$

$$\delta deltaz3 = \Delta z_{a1b1} - \Delta z_{a1b1+a2b2} \tag{6.34}$$

In equations 6.35 and 6.36, delta *z* for the mediated effect through Mediator 2 (a_2b_2) is compared with delta *z* from the mean of Y, and delta *z* for the total mediated effect.

$$\delta deltaz4 = \Delta z_{\hat{Y}_0} - \Delta z_{a2b2} \tag{6.35}$$

$$\delta deltaz5 = \Delta z_{a2b2} - \Delta z_{a1b1+a2b2} \quad (6.36)$$

The final Model Contrast is between delta z for the mean of Y with no predictors and delta z for the total mediated effect (equation 6.37).

$$\delta deltaz6 = \Delta z_{\hat{Y}_0} - \Delta z_{a1b1+a2b2} \quad (6.37)$$

gCd Model contrasts

Model Contrasts for the change in generalized Cook's distance were computed according to equations 6.38-6.43. These Model Contrasts mirror the differences in delta z computed in equations 6.32-6.37.

$$\delta gCd1 = gCd_{\hat{Y}_0} - gCd_{a1b1} \quad (6.38)$$

$$\delta gCd2 = gCd_{a1b1} - gCd_{a2b2} \quad (6.39)$$

$$\delta gCd3 = gCd_{a1b1} - gCd_{a1b1+a2b2} \quad (6.40)$$

$$\delta gCd4 = gCd_{\hat{Y}_0} - gCd_{a2b2} \quad (6.41)$$

$$\delta gCd5 = gCd_{a2b2} - gCd_{a1b1+a2b2} \quad (6.42)$$

$$\delta gCd6 = gCd_{\hat{Y}_0} - gCd_{a1b1+a2b2} \quad (6.43)$$

Statistical Analysis

A one-way ANOVA was conducted to test the null hypothesis that the mean values of individual fit Model Contrasts were equal among the known subpopulations, shown in equation 6.44. A priori contrasts were also conducted to test simple comparisons between known subpopulations, as in equations 6.45-6.50.

$$H0_1: \mu_{S1} = \mu_{S2} = \mu_{S3} = \mu_{S4} \quad (6.44)$$

$$H0_2: \mu_{S1} = \mu_{S2} \quad (6.45)$$

$$H0_3: \mu_{S1} = \mu_{S3} \quad (6.46)$$

$$H0_4: \mu_{S1} = \mu_{S4} \quad (6.47)$$

$$H0_5: \mu_{S2} = \mu_{S3} \quad (6.48)$$

$$H0_6: \mu_{S2} = \mu_{S4} \quad (6.49)$$

$$H0_7: \mu_{S3} = \mu_{S4} \quad (6.50)$$

Coffman and Millsap (2006) observed that while the contribution of individual cases to model misfit is small (especially as sample size increases), the rank ordering of the percent contribution is a useful method for identifying the relative importance of individual cases. Because the magnitude of individual fit Model Contrasts was expected to be small and potentially difficult to detect statistically, the rank ordering of the individual fit Model Contrasts was also investigated. Therefore raw and rank-ordered values of the Model Contrasts were used as dependent variables.

Evaluation Criteria

Significance Testing

Pairwise contrasts between known subpopulations are expected to have true differences when the Model Contrast includes the data generation model of *only one* subpopulation. For example, in a Model Contrast between a model with no predictors and a model through Mediator 1, the Subpopulation Contrast between individuals with effects through Mediator 1 and individuals with effects through Mediator 2 would have a true effect. The true effect occurs because the model with Mediator 1 is the data generation model for the subpopulation with effects through Mediator 1, and neither the independent model nor the model with Mediator 1 is the data generation model for the subpopulation with effects through Mediator 2.

There are two scenarios in which Subpopulation Contrasts are not expected to have a true difference. The first is when the generating models for both

subpopulations are included in the Model Contrast. For example, in the Model Contrast between a model through Mediator 1 and a model through Mediator 2, the contrast between individuals with effects through Mediator 1 and individuals with effects through Mediator 2 would not have a true effect because the model with Mediator 1 is the data generating model for the subpopulation with effects through Mediator 1 and the model with Mediator 2 is the data-generating model for the subpopulation with effects through Mediator 2. The second scenario is when neither data-generating model is included in the Model Contrast. For example, there is no true effect for the Subpopulation Contrast between individuals with effects through Mediator 2 only and individuals with effects through both Mediators 1 and 2 when the model with no predictors and a model through Mediator 1 are contrasted. Neither an independent model nor a model through Mediator 1 are data-generating models for subpopulations with effects through Mediator 2 only or with effects through both Mediators 1 and 2.

The logic for determining true and null differences for Model Contrasts and Subpopulations Contrasts is the same. The subtraction of a small value from a large value will result in a larger difference than the subtraction of two large or two small values. Known subpopulations are expected to have smaller residuals when the data generation and analysis models are the same and larger residuals when they are different. Therefore, individual residual differences will be larger when the Model Contrast involves the individual's data generation model since a larger residual and smaller residual will be subtracted from each other. Individual residual differences will be smaller when the data generation model is not included in the Model Contrast because two larger residuals will be subtracted from each other.

Type 1 Error Rate and Power Analogs. Because the study design involves contrasts of subpopulations within contrasts of model residuals, the traditional definition of Type 1 error does not apply. However, the proportion of replications per condition where the mean difference between subpopulations is significant when there is no true difference can be used as an analog for Type 1 error rates. The power analog was computed as the proportion of replications per condition where the mean differences between known subpopulations were statistically significant and only one of the known subpopulations had a true effect.

There is no difference in the dependent variable (i.e., the value of the Model Contrast) between known subpopulations when either both subpopulations have a true difference or neither have a true difference. Each Model Contrast has different Subpopulation Contrasts for which Type 1 error analog applies. Table 6 summarizes select Model Contrast and Subpopulation Contrast combinations that result in true differences (i.e., statistical power) and true null differences (i.e., Type 1 error). For example, there is no true effect between subpopulations $S1$and $S4$ when Model 0 (i.e., no predictors) and Model 1 (i.e., with Mediator 1) are contrasted. However, there is an effect between subpopulations $S1$and $S2$ when Models 0 and 1 are contrasted.

CHAPTER 7

RESULTS

Organization

One-way ANOVAs were conducted to test for significant differences between known subpopulations. A successful method should find significant differences when they are present and not find significant differences when they are not present. Results for the Model Contrast between a model with no predictors (Model 0) and a model with effects through Mediator 1 (Model 1) and the Model Contrast between Model 0 and a model with effects through Mediator 2 (Model 2) are similar; therefore, only the results for the Model Contrast between Model 0 and Model 1 are presented here. The Model Contrasts compared residuals, delta z, and gCd between estimated regression models. Two a priori Subpopulation Contrasts are described: the Subpopulation Contrast between a known subpopulation with no effects ($S4$) and a known subpopulation with effects through Mediator 1 ($S1$) and the Subpopulation Contrast between a known subpopulation with effects through Mediator 1 ($S1$) and a known subpopulation with effects through Mediator 2 ($S2$). A path diagram outlining the subpopulations is in figure 6.

Simulation results are organized into two sections. The first section describes the proportion of significant replications when there is no true difference between $S1$ and $S4$, which is analogous to examining Type 1 error rates. The second section describes the proportion of significant replications when there is a true difference between $S1$ and $S2$, which is analogous to examining statistical power. Within each

section, results are divided by method (i.e., residuals, delta *z*, and gCd). Results for raw differences and rank-ordered differences are presented simultaneously.

Proportion of Significant Replications (S1 – S4)

Residuals

Table 7 shows the proportion of significant replications for the contrast between subpopulations 1 and 4 for residual differences, delta *z*, and gCd. The first six rows of table 7 summarize the proportion of significant replications for raw and rank-ordered residual differences. The proportion of significant replications that occurred when there was no true subpopulation difference was between .01 – .07 for raw residual differences and between .03 – .07 for rank-ordered residual differences. For all parameter combinations, the proportion of significant replications was below the upper bound of the Type 1 error robustness interval (i.e., .075).

Delta z

The proportion of significant replications when there were no true subpopulation differences was between .00 – 1.00 for raw delta *z* differences and between .04 – .97 for rank-ordered delta *z* differences. The middle section (rows 7 to 10) of table 7 summarizes the proportion of significant replications for raw and rank-ordered delta *z* differences. The proportion of significant replications increased as the *S*1 effects through Mediator 1 increased in size, which is unexpected since there is no true subpopulation difference. Similar patterns were seen in both raw and rank-ordered differences.

gCd

The proportion of significant replications when there were no true subpopulation differences was between .00 – .068 for raw gCd differences and between .02 – .033 for rank-ordered gCd differences. The bottom section (last three rows) of table 7 shows that cThe pattern of results was similar for raw and rank-ordered differences, although the proportion of significant rank-ordered differences was smaller than raw differences when the *S*1 effects were large.

Proportion of Significant Replications (S1 – S2)

Residuals

The proportion of significant replications that occurred when there were true subpopulation differences was between .01 – .09 for raw residual differences and between .03 – .08 for rank-ordered residual differences. The first 18 rows of table 8 summarize the proportion of significant replications for raw and rank-ordered residual differences. For the smaller sample size (N=200), there is a slight increase in the proportion of significant results as the size of *S*1 effects through Mediator 1 increases. This pattern occurs for both raw and rank-ordered differences. However, the proportions don't increase with sample size, and when N=1000, the increase in significant proportions doesn't occur until the *S*1 effects are large. There was no noticeable difference in significant proportions between sample sizes, and the overall low proportions suggest that the observed pattern is slight, if present at all; therefore, further research is needed to determine whether mediation effect sizes truly influence the proportion of significant residual differences.

Delta z

The proportion of significant replications when there were true subpopulation differences was between $.03 - 1.00$ for raw delta z differences and between $.03 - .98$ for rank-ordered delta z differences. The middle section of table 8 shows that the proportion of significant replications increased as the $S1$ effects increased, but the was not a noticeable increase as the $S2$ effects increased. Because delta z is computed by systematically deleting single observations, when observations from $S1$ (with effects through Mediator 1) are deleted, there is a greater impact on the mediated effect and, therefore, on the differences in delta z. Therefore, an increase in significant replications would be expected as the effects of $a1$ and $b1$ increase.

gCd

The proportion of significant replications when there were true subpopulation differences was between $.00 - .69$ for raw gCd differences and between $.02 - .36$ for rank-ordered gCd differences. The pattern for delta z was also present for gCd such that the proportion increased as the $S1$ effects through Mediator 1 increased. The impact of the $S2$ effects can be seen in table 8, where the proportion of significant replications was higher when the $S1$ effects were zero and the $S2$ effects were larger compared to when the $S1$ effects were medium and the $S2$ effects were zero (see rows 6 and 7 from the bottom of table 8).

Summary

None of the three methods showed adequate performance for the Type 1 error and power analogs (i.e., proportions of significant results when there was and was not a true difference). For residual differences, all significant proportions were low, which meant the residuals method performed well when there was no true difference

but performed poorly when there was a true difference. In other words, the residual differences method could not distinguish the true difference between known subpopulations. The significant proportions for delta z were small when $S1$ effects were small and larger when $S1$ effects were larger, and this pattern occurred across all parameter combinations and Subpopulation Contrasts (i.e., the contrast of $S1$ vs. $S4$, or $S1$ vs. $S2$). The pattern of significant replications for gCd was small when the $S1$ effects were small or medium and larger when the $S1$ effects were large. This pattern also occurred across all parameter combinations. Therefore, delta z and gCd methods could not distinguish true differences because the pattern of significant replications was the same in both Subpopulation contrasts.

Overall, the proportion of significant replications when there was no true difference was best for residual differences, as both delta z and gCd resulted in proportions greater than the traditional robustness interval for Type 1 errors (i.e., $.025 - .075$). Correspondingly, the proportion of significant replications when there was a true difference was negligible for raw residual differences and higher for delta z, with the proportion of significant gCd differences falling between the other two methods. In all three methods, the significant proportion increased as the $S1$ effects increased. However, for residual differences, the increases in proportion were slight and were not associated with increases in sample size. Delta z and gCd had only one sample size, so no comparison was made for those two methods. Similar results were seen for raw and rank-ordered differences across all three methods.

CHAPTER 8

DISCUSSION

Traditional mediation analysis investigates causal mechanisms of human behavior, assuming that causal processes are the same for everyone. There are few statistical methods available to investigate mediated effects at the individual level. This dissertation aimed to introduce and test a novel method for investigating mediation when the population's causal process is heterogeneous. This method involves taking the difference in individual fit measures between a model with no predictors and a model with a mediated effect. Three individual fit measures were investigated: regression residuals, delta *z*, and gCd (Pek & MacCallum, 2011). Data were generated with 25% of observations having an effect through Mediator 1, 25% with an effect through Mediator 2, 25% with effects through both Mediator 1 and Mediator 2, and 25% of observations with no mediated effects. A simulation study was conducted to test whether there was a significant difference in individual fit measures between these four known subpopulations.

Summary of Simulation Results

The method of differences in individual fit measures was evaluated using analogs of Type 1 error and power, where the proportion of significant replications when there was no true difference corresponds to Type 1 error, and the proportion of significant replications when there was a true difference corresponds to statistical power.

The proportion of significant replications for residual differences was negligible across all parameter combinations, ranging from .01 - .09, and increased

slightly as effects through Mediator 1 became larger. In contrast, the proportion of significant replications for delta *z* and gCd ranged from 0.0 – 1.0, with greater increases as effects through Mediator 1 got larger compared to residual differences. In summary, the method using residual differences resulted in few significant replications, while the method using delta *z* and gCd resulted in a greater proportion of significant replications as effects increased. However, there was no difference in the pattern of significant replications when there was no true difference between known subpopulations compared to when there was a true difference. Several possible explanations for the poor performance of all three individual fit methods are described below.

Limitations

Several limitations are mentioned next, which will inform future studies. This study provides a methodology for exploring Model Contrasts that can evaluate future individual fit methods, such as the individual chi-square (Reise & Widaman, 1999). This study also represents the initial work necessary to understand how this novel methodology functions under reasonable parameters. Future studies should work to either replicate the findings of this study or uncover flaws in the logic of the methodology presented before applying the contrasts method to other individual fit methods.

Although findings suggest that the residual differences individual fit measure performs differently from delta *z* or gCd in the number of significant replications, none of the methods are adequate in their current form. It may be that the size of individual fit measure differences is simply too small to detect or that

heterogeneity of variance between subpopulations washes out any statistically significant group differences. Additionally, unmodeled mediation effects incorporated in the direct effect make distinguishing the residuals between two different mediators impossible. Finally, there may be logical flaws in how true effects were defined for Type 1 error and power analogs. Each of these alternatives are discussed below.

The pilot study results in table 2 show that mean residuals were relatively small for all subpopulations across all models. By extension, the differences in individual fit between known subpopulations for the same model were often small. Similar results apply to differences in individual fit for the same subpopulation across different models. Note that the pilot study used extremely large effect sizes to maximize results as a proof of concept. The main study used effect sizes that are more common in psychological studies and resulted in smaller residuals and residual differences than the pilot study. Given that the magnitudes of the differences are small, it may be that true differences exist between subpopulations but are indistinguishable from noise using the method described in the study, especially considering that the differences in standard deviations (see table 2) were large in comparison to the differences in residuals.

The heterogeneity of variance observed between subpopulations may account for the method's inability to detect mean differences. Returning to table 2, for Models 0, 1, and 2, there is a subpopulation whose standard deviation is noticeably smaller than the other subpopulations. The subpopulation with a smaller standard deviation corresponds to the subpopulation expected to have the smallest residual for that model, suggesting that if the absolute values of individual fit measures and

differences in individual fit measures are too small to detect, perhaps a method focused on the variability between subpopulations would be more efficacious.

Observed study results may have occurred because unmodeled mediator effects are incorporated into the effect of X, which would reduce the residual for that model. The residuals for models with and without the mediator should be the same because the effect of the unmodeled mediator is absorbed into the direct effect of X. Therefore, contrasted residuals from these two models would not distinguish between the modeled and unmodeled mediator. This alternative explanation represents a potential flaw in the current study's design that can be improved upon in further research. To address the issue, a second pilot study is described under Future Directions and in Appendix F.

Finally, the current study relied on definitions of true effects and analogs of Type 1 error and power that may limit the method's performance. Specifically, true subpopulation differences were defined using a two-step process. The first step assumed that the various individual fit measures would be smaller or larger for each model relative to each subpopulation. The second step assumed that subtracting values of relatively different sizes would result in a true difference while subtracting values of relatively similar sizes would result in no true difference. There are two potential problems with this strategy. First, the magnitudes of the individual fit measures and their differences are ill-defined and rely on relative comparisons (i.e., one is smaller than the other, or one is larger than the other) rather than explicit definitions of small and large magnitudes. Second, there are two qualitatively different scenarios in which two values of similar size are subtracted: when both

individual fit measures are small (or zero), or both values are large. For example, if both residuals in a comparison are small, the data generating process for both subpopulations matches the estimated model. However, if both residuals are large, neither subpopulation's data-generating process matches the estimated model. While the inferences one would draw from each of these scenarios are likely different, the current method of defining no true differences does not distinguish these two scenarios.

Future Directions

The following section discusses future research that addresses the limitations and alternative explanations for the study results discussed above. A secondary pilot data set was generated and analyzed for models that excluded X as a predictor to address the fact that unmodeled mediation effects become incorporated in modeled direct effects. Details and results of the pilot simulation are reported in Appendix F. Initial results suggest that subpopulation differences in delta z and gCd could be distinguished in a one-way ANOVA when mediators alone are used to predict Y.

Originally, the study design contained 36 contrasts between four mediation models and four known subpopulations. However, it became apparent that several contrasts were redundant because residuals for different mediators could not be distinguished. Additionally, it was unclear how some contrasts should be interpreted, such as Subpopulation Contrasts when one subpopulation has mediated effects through both mediators or when mediated effects represented by the Model Contrast do not correspond with mediated effects present in the known subpopulations. Future research can address these, and other limitations related to

the complexity of the original study design by focusing more narrowly on the Model Contrast between a model with one mediator versus a model with no predictors or a model with only a direct effect. Limiting the study design to only two models that compare one mediator to no mediators will make it clearer under which circumstances there are subpopulation differences before extending the research to multiple mediators. Finally, the methods used in this study were designed to detect single influential cases and outliers. However, the current study used subpopulations of equal proportions rather than single observations with differing mediation effects. A future simulation may look at how different proportions in the subpopulations affect results.

Implications

The causal processes that influence human behavior often apply to most individuals in a population. Mediation studies that involve constructs like social norms, subject-matter knowledge, or self-efficacy can describe processes that are relevant to many people. However, some disease processes, such as substance use disorder or rheumatoid arthritis, are thought to be heterogenous and dependent on individual characteristics (Carroll, 2021; Deane & Holers, 2019; Holers et al., 2018; Hsiao et al., 2020). Identifying individual mediation effects could help researchers identify more effective treatment protocols for people who respond differently or have different mediation processes at work.

The results from this study suggest that a method comparing individual fit methods across models with different mediators is limited in its ability to detect differences between people who have effects through one mediator compared to

people who do not. Focusing on the variability of individual fit differences may be more productive than studying raw values; however, this may be difficult if an atypical mediation process only occurs for a small proportion of the population. Heterogeneity of variance was observed in the current study, where each mediation process existed for 25% of the sample.

The current study focused on a method that used comparisons of residuals, delta *z,* and gCd across multiple mediation models. By incorporating improvements discussed in the limitations section, this methodology could be used to test other individual fit or effect size measures to investigate individual mediation effects, such as Individual Chi-square (Reise & Widaman, 1999). Individual fit measures are only one possible approach to individual mediation. Moderation of mediation effects, latent class models, and multi-level models are all statistical methods that incorporate information on individual mediation as part of the model. The method of contrasting mediation models presented in this study could conceivably be applied to these other statistical methods to investigate individual differences in mediation. The current study does not provide conclusive evidence for the viability of the described method or the utility of individual fit measures for investigating individual mediation effects. However, it can provide a framework for future studies to improve upon the method and apply it to other measures of individual effects.

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APPENDIX A

TABLES

Table 1

True Coefficient Values for Pilot Study

Subpopulation	*n*	*a1*	*a2*	*b1*	*b2*	*c'*
S1	500	9	9	0	0	0
S2	500	0	0	9	9	0
S3	500	0	0	0	0	0
Total	1500	-	-	-	-	-
Average	-	3	3	3	3	0

Note. Parameter magnitudes are uncharacteristically large whole numbers to show conceptual viability.

Table 2

Means by Model and Subpopulation

Subpopulation	N	Mean	Std Dev
	Residuals - Model 0		
S1	500	-.322	84.538
S2	500	-1.680	81.483
S3	500	2.002	0.995
Full Sample	1500	.000[a]	67.763
	Residuals - Model 1		
S1	500	0.031	5.033
S2	500	-1.890	41.496
S3	500	1.859	41.361
Full Sample	1500	.000[a]	33.963
	Residuals - Model 2		
S1	500	-1.616	41.366
S2	500	-.078	5.010
S3	500	1.694	42.897
Full Sample	1500	.000[a]	34.531
	Residuals - Model 3		
S1	500	-.520	8.223
S2	500	-.223	8.823
S3	500	.743	13.077
Full Sample	1500	.000[a]	10.278
	Delta z - Mediation Path a_1b_1		
S1	500	.036	.057
S2	500	-.019	.027
	Delta z - Mediation Path a_2b_2		
S1	500	-.020	.029
S2	500	.035	.054
	gCd - Mediation Path a_1b_1		
S1	500	.005	.018
S2	500	.001	.004
	gCd - Mediation Path a_2b_2		
S1	500	.001	.005
S2	500	.004	.016
	IND_{CHI}		
S1	500	2.812	1.015
S2	500	2.845	1.032
S3	500	3.670	2.110

Note. [a]Non-zero values rounded to three decimal places.

Table 3

Summary of Subpopulations

Subpopulation	True Effects
S1	M1
S2	M2
S3	M1 and M2
S4	None

Note. M1 refers to a mediated effect through Mediator 1, while M2 refers to a mediated effect through Mediator 2.

Table 4

Summary of Possible Model Contrasts

Models Compared	Residual Differences	Delta z Differences	gCd Differences
Model 0 – Model 1	$\delta 1 = e_{0i} - e_{1i}$	$\delta deltaz1 = \Delta z_{\hat{Y}_0} - \Delta z_{a1b1}$	$\delta gCd1 = gCd_{\hat{Y}_0} - gCd_{a1b1}$
Model 1 – Model 2	$\delta 2 = e_{1i} - e_{2i}$	$\delta deltaz2 = \Delta z_{a1b1} - \Delta z_{a2b2}$	$\delta gCd2 = gCd_{a1b1} - gCd_{a2b2}$
Model 1 – Model 3	$\delta 3 = e_{1i} - e_{3i}$	$\delta deltaz3 = \Delta z_{a1b1} - \Delta z_{a1b1+a2}$	$\delta gCd3 = gCd_{a1b1} - gCd_{a1b1+a2b}$
Model 0 – Model 2	$\delta 4 = e_{0i} - e_{2i}$	$\delta deltaz4 = \Delta z_{\hat{Y}_0} - \Delta z_{a2b2}$	$\delta gCd4 = gCd_{\hat{Y}_0} - gCd_{a2b2}$
Model 2 – Model 3	$\delta 5 = e_{2i} - e_{3i}$	$\delta deltaz5 = \Delta z_{a2b2} - \Delta z_{a1b1+a2}$	$\delta gCd5 = gCd_{a2b2} - gCd_{a1b1+a2}$
Model 0 – Model 3	$\delta 6 = e_{0i} - e_{3i}$	$\delta deltaz6 = \Delta z_{\hat{Y}_0} - \Delta z_{a1b1+a2b2}$	$\delta gCd6 = gCd_{\hat{Y}_0} - gCd_{a1b1+a2b2}$

Note. This table demonstrates all possible Model Contrasts; however, several of these contrasts are redundant. Simulation results focus on the first row which is the contrast between Models 0 and 1.

Table 5

Summary of Mediators Estimated in each Model

Model	M1	M2
Model 0	NE	NE
Model 1	*E*	NE
Model 2	NE	*E*
Model 3	*E*	*E*

Note. Cells designated with an 'NE' mean the mediator is not estimated in the model. Cells designated with an 'E' mean the mediator is estimated in the model.

Table 6

Effects of Significant Replications

Subpopulation Contrasts	Analysis Model Contrasts		
	$\delta 1 = e_{0i} - e_{1i}$	$\delta 4 = e_{0i} - e_{2i}$	$\delta 6 = e_{0i} - e_{3i}$
S1 - S2	Effect	Effect	No Effect
S1 - S3	Effect	No Effect	Effect
S1 - S4	No Effect	Effect	Effect
S3 - S4	Effect	Effect	No Effect

Note. This table summarizes whether a Subpopulation Contrast is expected to have a true effect for each Model Contrast. Model Contrasts are defined in table 4.

Table 7

Proportion of Significant Replications (S1 – S4)

Parameter combination			Significant Proportion[a]	
N	*a1=b1 (S1)*[b]	*a1=b1 (S4)*[b]	Raw difference	Rank-ordered difference
			Residuals	
200	0	0	0.03	0.04
	0.39	0	0.03	0.05
	0.99	0	0.05	0.06
1000	0	0	0.03	0.04
	0.39	0	0.04	0.05
	0.99	0	0.06	0.06
			Delta *z*	
200	0	0	0.03	0.05
	0.39	0	0.38	0.36
	0.99	0	0.97	0.93
			gCd	
200	0	0	0.02	0.04
	0.39	0	0.05	0.04
	0.99	0	0.34	0.15

Note. Results are from the Model Contrast between a model with no predictors and a model with effects through Mediator 1 and the Subpopulation Contrast between a subpopulation with no mediated effects and a subpopulation with effects through Mediator 1.

[a]Values are analogous to Type 1 error. [b]S1 is the subpopulation with effects through Mediator 1. S4 is the subpopulation with no mediated effects.

Table 8

Proportion of Significant Replications (S1 – S2)

Parameter combination			Significant Proportion[a]	
N	*a1=b1 (S1)*[b]	*a2=b2 (S2)*[b]	Raw difference	Rank-ordered difference
			Residuals	
200	0	0	0.03	0.04
		0.39	0.03	0.04
		0.99	0.04	0.05
	0.39	0	0.04	0.05
		0.39	0.04	0.05
		0.99	0.05	0.06
	0.99	0	0.07	0.07
		0.39	0.07	0.07
		0.99	0.07	0.07
1000	0	0	0.04	0.04
		0.39	0.04	0.04
		0.99	0.04	0.04
	0.39	0	0.04	0.05
		0.39	0.04	0.05
		0.99	0.04	0.05
	0.99	0	0.06	0.06
		0.39	0.05	0.06
		0.99	0.05	0.05
			Delta *z*	
200	0	0	0.04	0.05
		0.39	0.04	0.05
		0.99	0.06	0.07
	0.39	0	0.44	0.40
		0.39	0.45	0.41
		0.99	0.49	0.45
	0.99	0	0.97	0.92
		0.39	0.97	0.92
		0.99	0.99	0.96
			gCd	
200	0	0	0.03	0.04
		0.39	0.02	0.04
		0.99	0.12	0.12
	0.39	0	0.06	0.05
		0.39	0.05	0.05
		0.99	0.11	0.08
	0.99	0	0.31	0.11
		0.39	0.30	0.12
		0.99	0.26	0.22

Note. Results are from the Model Contrast between a model with no predictors and a model with effects through Mediator 1, and the Subpopulation Contrast between a subpopulation with effects through Mediator 1 and a subpopulation with effects through Mediator 2.

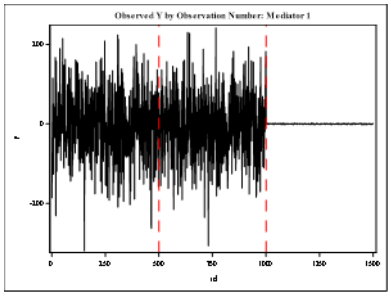
[a]Values are analogous to statistical power. [b]S1 is the subpopulation with effects through Mediator 1. S2 is the subpopulation with effects through Mediator 2.

APPENDIX B

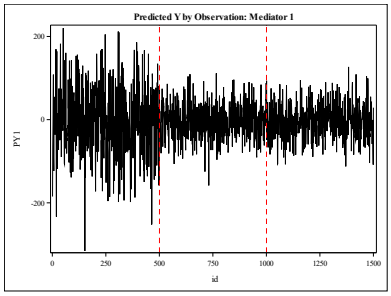
FIGURES

Figure 1

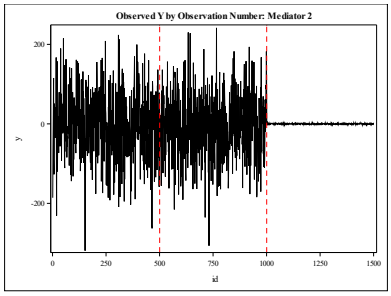
Index Plots of Observed and Predicted Y by Observation



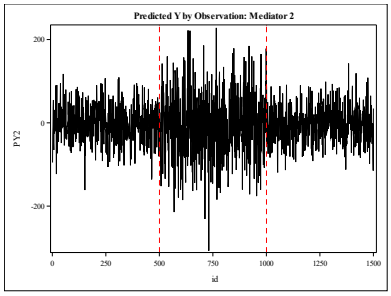
1a.



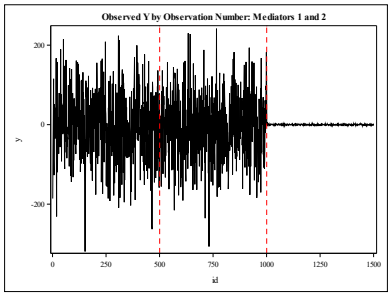
1b.



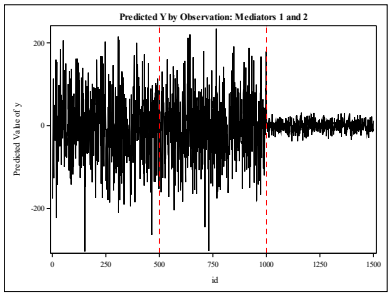
2a.



2b.



3a.

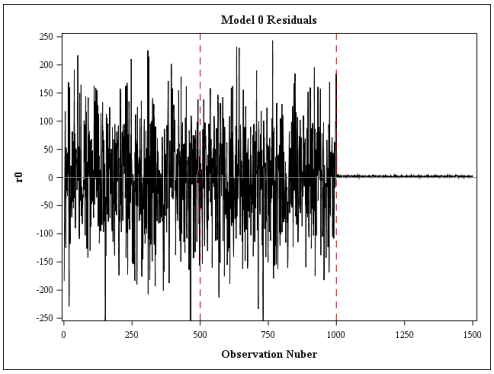


3b.

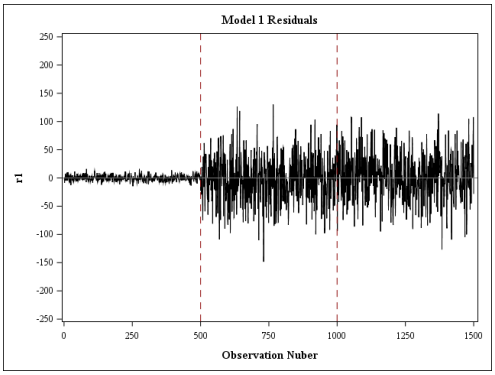
Note. Panel columns are plots of observed (left) and predicted (right) values. Rows are different models. The dashed reference lines show the last replicate representing S1 and the last replicate representing S2 (replicate 500 and 1000).

Figure 2

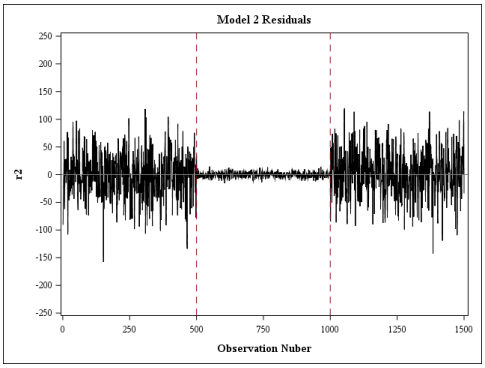
Index Plots of Residuals by Observation



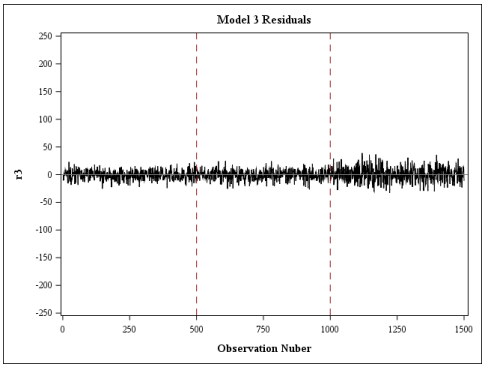
Panel 1



Panel 2



Panel 3

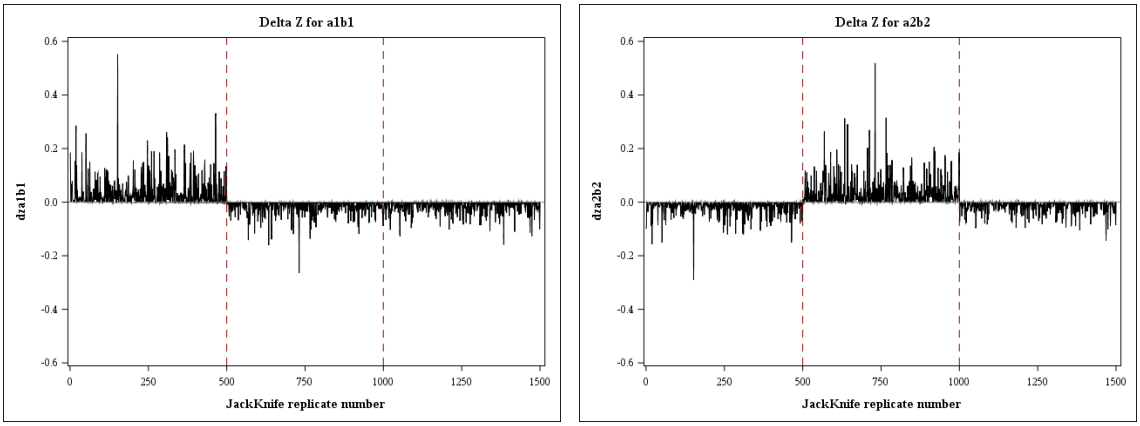


Panel 4

Note. Panel 1 shows a model for Y with no predictors. Panel 2 shows a model through M1 only. Panel 3 shows a model through M2 only. Panel 4 shows a through both M1 and M2.
The dashed reference lines show the last replicate representing S1 and the last replicate representing S2 (replicate 500 and 1000). Observations 1-500 reflect S1, 501-1000 reflect S2, and 1001-1500 reflect S3.

Figure 3

Index Plot of Delta z by Observation and Subpopulation

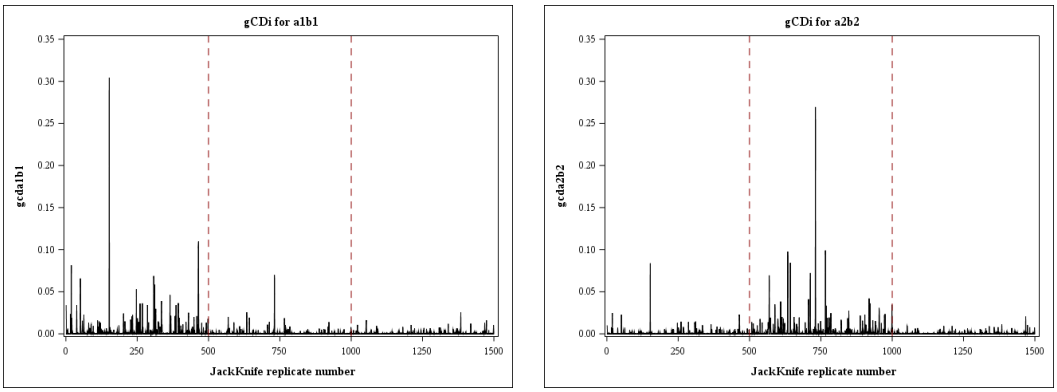


Panel A Panel B

Note. The dashed reference lines show the last replicate representing S1 and the last replicate representing S2 (replicate 500 and 1000).

Figure 4

Index Plots of gCd by Observation and Subpopulation

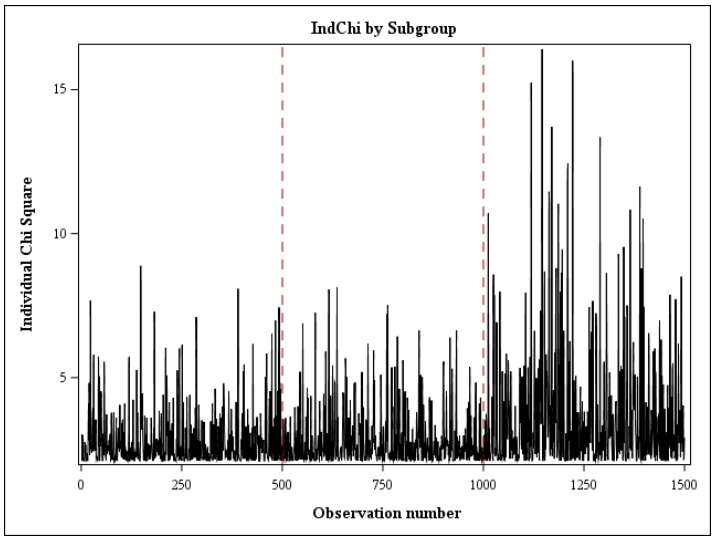


Panel A Panel B

Note. The dashed reference lines show the last replicate representing S1 and the last replicate representing S2 (replicate 500 and 1000).

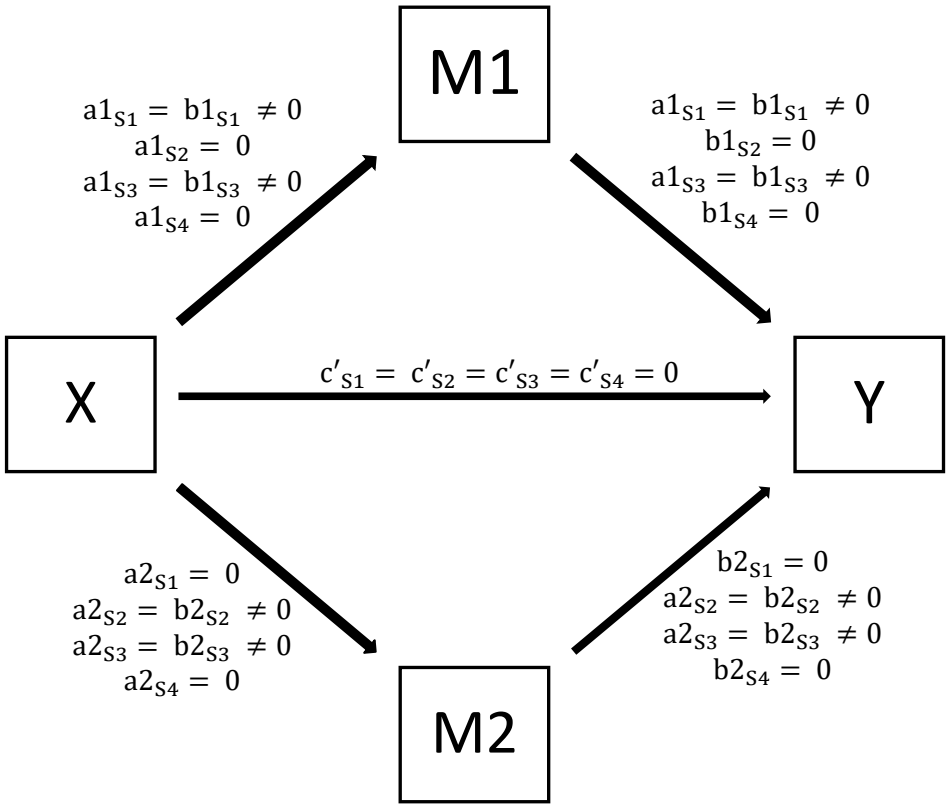
Figure 5

Index Plot of IND_{CHI} by Observation and Subpopulation



Note. The dashed reference lines show the last replicate representing S1 and the last replicate representing S2 (replicate 500 and 1000).

Figure 6



Note. S1 has effects through M1 only, S2 has effects through M2 only, S3 has effects through M1 and M2, and S4 has no effects

APPENDIX C

SAS PROGRAM FOR DATA GENERATION

```
libname a "D: ";

*imports csv file with true values for the simulation conditions;

PROC IMPORT OUT= WORK.conds
DATAFILE= "D: \conditions.csv"
    DBMS=CSV REPLACE;
    GETNAMES=YES;
    DATAROW=2;
RUN;

/* reads through csv file and creates a macro variable for each simulation parameter
and appends a number to each value corresponding to the condition it is associated with. */

data conds; length ii $8; set conds;
i+1;
ii=left(put(i, 8.));
call symput ('FILE'||ii, FILE);
call symput ('NSIM'||ii, NSIM);
call symput ('NOBS'||ii, NOBS);
call symput ('TNOBS'||ii,TNOBS);
call symput ('S1A1'||ii, S1A1);
call symput ('S1B1'||ii, S1B1);
call symput ('S1A2'||ii, S1A2);
call symput ('S1B2'||ii, S1B2);
call symput ('S1CP'||ii, S1CP);
call symput ('S2A1'||ii, S2A1);
call symput ('S2B1'||ii, S2B1);
call symput ('S2A2'||ii, S2A2);
call symput ('S2B2'||ii, S2B2);
call symput ('S2CP'||ii, S2CP);
call symput ('S3A1'||ii, S3A1);
call symput ('S3B1'||ii, S3B1);
call symput ('S3A2'||ii, S3A2);
call symput ('S3B2'||ii, S3B2);
call symput ('S3CP'||ii, S3CP);
call symput ('S4A1'||ii, S4A1);
call symput ('S4B1'||ii, S4B1);
call symput ('S4A2'||ii, S4A2);
call symput ('S4B2'||ii, S4B2);
call symput ('S4CP'||ii, S4CP);
call symput ('n', _n_);
drop i;
run;


DATA SUMMARY; SET _NULL_;
%MACRO
SIMULATE(NSIM,NOBS,TNOBS,FILE,S1A1,S1B1,S1A2,S1B2,S1CP,S2A1,S2B1,S2A2,S2B
2,S2CP,S3A1,S3B1,S3A2,S3B2,S3CP,S4A1,S4B1,S4A2,S4B2,S4CP);

TITLE 'SIMULATION OF MEDIATION RESIDUALS';
```

```
/*SUBPOP 1 EFFECTS THROUGH M1*/
DATA SIM1;
totaln=&NSIM*&NOBS;
DO I=1 TO totaln;
        sgrp=1;
        N=&NOBS;
        xmean = 0;
        x_std = 1;

        intM1 = 0;
        bM1X = &S1A1;

        intM2 = 0;
        bM2X = &S1A2;

        intY = 0;
        bYX = &S1CP;
        bYM1 = &S1B1;
        bYM2 = &S1B2;

        e_stdM1 = 1;
        e_stdM2 = 1;
        e_stdY = 1;

call streaminit( 19800303 );
                x = RAND('Normal', xmean, x_std);
                m1 = intM1 + bM1X*x + e_stdM1*RAND('Normal', 0,1);
                m2 = intM2 + bM2X*x + e_stdM2*RAND('Normal',0,1);
                y = intY + bYX*x + bYM1*m1 + bYM2*m2 + e_stdY*RAND('Normal',0,1);
                OUTPUT;
        END;

/*ASSIGNING REPLICATION NUMBERS TO OBSERVATIONS*/
DATA SIM1; set SIM1;
J=&nobs;
DO J=0 to totaln by &nobs;
IF 1+J<=I<=&NOBS+J then rep=1+(J/&nobs);
end;

/*SUBPOP 2 EFFECTS THROUGH M2*/
DATA SIM2;
totaln=&NSIM*&NOBS;
DO I=1 TO totaln;
        sgrp=2;
        N=&NOBS;
        xmean = 0;
        x_std = 1;

        intM1 = 0;
        bM1X = &S2A1;
```

```
        intM2 = 0;
        bM2X = &S2A2;

        intY = 0;
        bYX = &S2CP;
        bYM1 = &S2B1;
        bYM2 = &S2B2;

        e_stdM1 = 1;
        e_stdM2 = 1;
        e_stdY = 1;

call streaminit( 19960303 );
                x = RAND('Normal', xmean, x_std);
                m1 = intM1 + bM1X*x + e_stdM1*RAND('Normal', 0,1);
                m2 = intM2 + bM2X*x + e_stdM2*RAND('Normal',0,1);
                y = intY + bYX*x + bYM1*m1 + bYM2*m2 + e_stdY*RAND('Normal',0,1);
                OUTPUT;
        END;
/*ASSIGNING REPLICATION NUMBERS TO OBSERVATIONS*/
DATA SIM2; set SIM2;
J=&nobs;
DO J=0 to totaln by &nobs;
IF 1+J<=I<=&NOBS+J then rep=1+(J/&nobs);
end;

/*SUBPOP 3 EFFECTS THROUGH BOTH */
DATA SIM3;
totaln=&NSIM*&NOBS;
DO I=1 TO totaln;
        sgrp=3;
        N=&NOBS;
        xmean = 0;
        x_std = 1;

        intM1 = 0;
        bM1X = &S3A1;

        intM2 = 0;
        bM2X = &S3A2;

        intY = 0;
        bYX = &S3CP;
        bYM1 = &S3B1;
        bYM2 = &S3B2;

        e_stdM1 = 1;
        e_stdM2 = 1;
        e_stdY = 1;

call streaminit( 20010303 );
                x = RAND('Normal', xmean, x_std);
```

```
                m1 = intM1 + bM1X*x + e_stdM1*RAND('Normal', 0,1);
                m2 = intM2 + bM2X*x + e_stdM2*RAND('Normal',0,1);
                y = intY + bYX*x + bYM1*m1 + bYM2*m2 + e_stdY*RAND('Normal',0,1);
                OUTPUT;
        END;

/*ASSIGNING REPLICATION NUMBERS TO OBSERVATIONS*/
DATA SIM3; set SIM3;
J=&nobs;
DO J=0 to totaln by &nobs;
IF 1+J<=I<=&NOBS+J then rep=1+(J/&nobs);
end;

/*SUBPOP 4 EFFECTS THROUGH NEITHER*/
DATA SIM4;
totaln=&NSIM*&NOBS;
DO I=1 TO totaln;
        sgrp=4;
        N=&NOBS;
        xmean = 0;
        x_std = 1;

        intM1 = 0;
        bM1X = &S4A1;

        intM2 = 0;
        bM2X = &S4A2;

        intY = 0;
        bYX = &S4CP;
        bYM1 = &S4B1;
        bYM2 = &S4B2;

        e_stdM1 = 1;
        e_stdM2 = 1;
        e_stdY = 1;

call streaminit( 20220303 );
                x = RAND('Normal', xmean, x_std);
                m1 = intM1 + bM1X*x + e_stdM1*RAND('Normal', 0,1);
                m2 = intM2 + bM2X*x + e_stdM2*RAND('Normal',0,1);
                y = intY + bYX*x + bYM1*m1 + bYM2*m2 + e_stdY*RAND('Normal',0,1);
                OUTPUT;
        END;

/*ASSIGNING REPLICATION NUMBERS TO OBSERVATIONS*/
DATA SIM4; set SIM4;
J=&nobs;
DO J=0 to totaln by &nobs;
IF 1+J<=I<=&NOBS+J then rep=1+(J/&nobs);
end;
```

```
/*COMBINING SUBPOPULATIONS INTO SINGLE POPULATION*/
DATA FULL; SET SIM1 SIM2 SIM3 SIM4;
NOBS=&NOBS;
FILE="&FILE";
drop J totaln;
run;

/*Save each condition to a new file*/
Data a.&file; set FULL;
run;

%MEND simulate;
%MACRO Condloop;
        %do i=1 %to &n;
%SIMULATE(NSIM=&&NSIM&i,NOBS=&&NOBS&i,TNOBS=&&TNOBS&i,FILE=&&FIL
E&i,S1A1=&&S1A1&i,S1B1=&&S1B1&i,S1A2=&&S1A2&i,S1B2=&&S1B2&i,S1CP=&&S1C
P&i,S2A1=&&S2A1&i,
S2B1=&&S2B1&i,S2A2=&&S2A2&i,S2B2=&&S2B2&i,S2CP=&&S2CP&i,S3A1=&&S3A1&i,
S3B1=&&S3B1&i,S3A2=&&S3A2&i,S3B2=&&S3B2&i,S3CP=&&S3CP&i,
S4A1=&&S4A1&i,S4B1=&&S4B1&i,S4A2=&&S4A2&i,S4B2=&&S4B2&i,S4CP=&&S4CP&i)
;
        %end;
%mend condloop;
%condloop; run;
```

APPENDIX D

SAS PROGRAM FOR ANALYSIS OF RESIDUALS

```
libname a "D:\Sync\Science Binders\028-dissertation\028-data";

OPTIONS PS=59 LS=80 REPLACE NONOTES;
FILENAME NULLOG DUMMY 'C:\NULL';
PROC PRINTTO LOG=NULLOG;

PROC IMPORT OUT= WORK.conds
        DATAFILE= "D:\Sync\Science Binders\028-dissertation\028-code\memo
run\CONDITIONS.csv"
        DBMS=CSV REPLACE;
   GETNAMES=YES;
   DATAROW=2;
        GUESSINGROWS=500;
RUN;

data conds; length ii $8; set conds;
i+1;
ii=left(put(i, 8.));
call symput ('FILE'||ii, FILE);
call symput ('NSIM'||ii, NSIM);
call symput ('NOBS'||ii, NOBS);
call symput ('TNOBS'||ii,TNOBS);

call symput ('S1A1'||ii, S1A1);
call symput ('S1B1'||ii, S1B1);
call symput ('S1A2'||ii, S1A2);
call symput ('S1B2'||ii, S1B2);
call symput ('S1CP'||ii, S1CP);

call symput ('S2A1'||ii, S2A1);
call symput ('S2B1'||ii, S2B1);
call symput ('S2A2'||ii, S2A2);
call symput ('S2B2'||ii, S2B2);
call symput ('S2CP'||ii, S2CP);

call symput ('S3A1'||ii, S3A1);
call symput ('S3B1'||ii, S3B1);
call symput ('S3A2'||ii, S3A2);
call symput ('S3B2'||ii, S3B2);
call symput ('S3CP'||ii, S3CP);

call symput ('S4A1'||ii, S4A1);
call symput ('S4B1'||ii, S4B1);
call symput ('S4A2'||ii, S4A2);
call symput ('S4B2'||ii, S4B2);
call symput ('S4CP'||ii, S4CP);

call symput ('n', _n_);
drop i;
run;

data SUMMARY2; set _null_;
```

```
data SUMMARY2b; set _null_;

%macro
RESIDS(NSIM,NOBS,TNOBS,FILE,S1A1,S1B1,S1A2,S1B2,S1CP,S2A1,S2B1,S2A2,S2B2,S2
CP,S3A1,S3B1,S3A2,S3B2,S3CP,S4A1,S4B1,S4A2,S4B2,S4CP);

data SUMMARY; set _null_;
data SUMMARYB; set _null_;

DATA SIMALL; SET A.&FILE;
run;


%split(nobs=&&nobs&i,TNOBS=&&Tnobs&i, NSIM=&&nSIM&i);
run;

data new2; set SUMMARY2;
run;

data SUMMARY2; set new2 SUMMARY;
run;

data new2b; set SUMMARY2b;
run;

data SUMMARY2b; set new2b SUMMARYb;
run;

%mend RESIDS;
run;
quit;

%MACRO split (nobs, TNOBS, NSIM);
%do k=1 %to &&nsim&i;

Data SIM; set SIMALL(where=(Rep=&k)); run;

/*ESTIMATE FOUR MODELS IN ORIGINAL DATA*/
/*Model 0*/
title;
proc reg data=SIM noprint;
Model y= /p r;
output out=pr0 p=p0 r=r0 stdr=SEr0;
run;QUIT;

/*Model 1*/
proc reg data=SIM noprint;
ModelM: model m1=x;
ModelY: model y=x m1/p r;
output out=pr1 p=p1 r=r1 stdr=SEr1;
run;QUIT;
```

```
/*Model 2*/
proc reg data=SIM noprint;
ModelM: model m2=x;
ModelY: model y=x m2/p r;
output out=pr2 p=p2 r=r2 stdr=SEr2;
run;QUIT;

/*Model 3*/
proc reg data=SIM noprint;
ModelM1: model m1=x;
ModelM2: model m2=x;
ModelY: model y=x m1 m2/p r;
output out=pr3 p=p3 r=r3 stdr=SEr3;
run;
quit;

/*MERGE RESIDUALS INTO DATASET */
DATA FOURMODRES;
merge pr0 pr1 pr2 pr3;
label p0 = 'p0' r0 = 'r0' p1 = 'p1' r1 = 'r1' p2 = 'p2' r2 = 'r2' p3 = 'p3' r3 = 'r3' SEr0 = 'SEr0'
SEr1 = 'SEr1' SEr2 = 'Ser2' Ser3 = 'Ser3' ;
run;

/*COMPUTE RESIDUAL DIFFERENCES*/
data FOURMODRES; set FOURMODRES;
d1 = R0-R1;
d2 = R1-R2;
d3 = R1-R3;
d4 = R0-R2;
d5 = R2-R3;
d6 = R0-R3;
resvar0 = SEr0**2;
resvar1 = SEr1**2;
resvar2 = SEr2**2;
resvar3 = SEr3**2;
run;

/*ITERATIVELY SAVING REPLICATION RESULTS*/
data new; set summary;
run;

data summary; set new FOURMODRES;
run;

/*BOOTSTRAP RESAMPLING*/;
%LET NBOOT=1000;
proc surveyselect data=A.RESIDS noprint out=RESBOOT  method=urs sampsize=&TNOBS
rep=&NBOOT outhits;
BY REP;
run; quit;

/*ESTIMATE FOUR MODELS IN BOOTSTRAP SAMPLES*/
```

```
/*Model 0*/
title;
proc reg data=RESBOOT noprint;
BY REP REPLICATE;
Model y= /p r;
output out=PR0B p=p0b r=r0b stdr=SEr0b;
run;QUIT;

data PR0B; set PR0B;
label p0b = 'p0b' r0b = 'r0b' SEr0b = 'SEr0b';
run;

/*Model 1*/
proc reg data=RESBOOT noprint;
BY REP REPLICATE;
ModelM: model m1=x;
ModelY: model y=x m1/p r;
output out=PR1B p=p1b r=r1b stdr=SEr1b;
run;QUIT;

data PR1B; set PR1B;
label p1b = 'p1b' r1b = 'r1b'  SEr1b = 'SEr1b' ;
run;

/*Model 2*/
proc reg data=RESBOOT noprint;
BY REP REPLICATE;
ModelM: model m2=x;
ModelY: model y=x m2/p r;
output out=PR2B p=p2b r=r2b stdr=SEr2b;
run;QUIT;

data PR2B; set PR2B;
label p2b = 'p2b' r2b = 'r2b'  SEr2b = 'SEr2b';
run;

/*Model 3*/
proc reg data=RESBOOT noprint;
BY REP REPLICATE;
ModelM1: model m1=x;
ModelM2: model m2=x;
ModelY: model y=x m1 m2/p r;
output out=PR3B p=p3b r=r3b stdr=SEr3b;
run;
quit;

data PR3B; set PR3B;
label p3b = 'p3b' r3b = 'r3b' SEr3b = 'SEr3b';
run;

DATA FOURMRBOOT;
merge RESBOOT pr0B pr1B pr2B pr3B;
```

```
run;

/*COMPUTE RESIDUAL DIFFERENCES IN BOOTSTRAP SAMPLES*/
data FOURMRBOOT; set FOURMRBOOT;
d1b = R0b-R1b;
d2b = R1b-R2b;
d3b = R1b-R3b;
d4b = R0b-R2b;
d5b = R2b-R3b;
d6b = R0b-R3b;
resvar0b = SEr0b**2;
resvar1b = SEr1b**2;
resvar2b = SEr2b**2;
resvar3b = SEr3b**2;
run;

/*COMPUTING LCL AND UCL*/
proc univariate data=FOURMRBOOT noprint;
var d1b;
output out=bootd1
pctlpts = 2.5, 97.5
pctlpre = Pd1_;
run;

proc univariate data=FOURMRBOOT noprint;
var d2b;
output out=bootd2
pctlpts = 2.5, 97.5
pctlpre = Pd2_;
run;

proc univariate data=FOURMRBOOT noprint;
var d3b;
output out=bootd3
pctlpts = 2.5, 97.5
pctlpre = Pd3_;
run;

proc univariate data=FOURMRBOOT noprint;
var d4b;
output out=bootd4
pctlpts = 2.5, 97.5
pctlpre = Pd4_;
run;

proc univariate data=FOURMRBOOT noprint;
var d5b;
output out=bootd5
pctlpts = 2.5, 97.5
pctlpre = Pd5_;
run;
```

```
proc univariate data=FOURMRBOOT noprint;
var d6b;
output out=bootd6
pctlpts = 2.5, 97.5
pctlpre = Pd6_;
run;

data cisa; merge bootd1-bootd6;run;

DATA FOURMRBOOT; MERGE FOURMRBOOT CISA;RUN;

/*COPY CIS DOWN ROWS*/
DATA FOURMRBOOT
(DROP = XPd1_2_5 XPd1_97_5 XPd2_2_5 XPd2_97_5 XPd3_2_5 XPd3_97_5 XPd4_2_5
XPd4_97_5 XPd5_2_5 XPd5_97_5 XPd6_2_5 XPd6_97_5) ;
SET FOURMRBOOT ;
RETAIN XPd1_2_5 ;
IF NOT MISSING(Pd1_2_5) THEN XPd1_2_5 = Pd1_2_5 ;
Pd1_2_5 = XPd1_2_5 ;
RETAIN XPd1_97_5 ;
IF NOT MISSING(Pd1_97_5) THEN XPd1_97_5 = Pd1_97_5 ;
Pd1_97_5 = XPd1_97_5 ;
RETAIN XPd2_2_5 ;
IF NOT MISSING(Pd2_2_5) THEN XPd2_2_5 = Pd2_2_5 ;
Pd2_2_5 = XPd2_2_5 ;
RETAIN XPd2_97_5 ;
IF NOT MISSING(Pd2_97_5) THEN XPd2_97_5 = Pd2_97_5 ;
Pd2_97_5 = XPd2_97_5 ;
RETAIN XPd3_2_5 ;
IF NOT MISSING(Pd3_2_5) THEN XPd3_2_5 = Pd3_2_5 ;
Pd3_2_5 = XPd3_2_5 ;
RETAIN XPd3_97_5 ;
IF NOT MISSING(Pd3_97_5) THEN XPd3_97_5 = Pd3_97_5 ;
Pd3_97_5 = XPd3_97_5 ;
RETAIN XPd4_2_5 ;
IF NOT MISSING(Pd4_2_5) THEN XPd4_2_5 = Pd4_2_5 ;
Pd4_2_5 = XPd4_2_5 ;
RETAIN XPd4_97_5 ;
IF NOT MISSING(Pd4_97_5) THEN XPd4_97_5 = Pd4_97_5 ;
Pd4_97_5 = XPd4_97_5 ;
RETAIN XPd5_2_5 ;
IF NOT MISSING(Pd5_2_5) THEN XPd5_2_5 = Pd5_2_5 ;
Pd5_2_5 = XPd5_2_5 ;
RETAIN XPd5_97_5 ;
IF NOT MISSING(Pd5_97_5) THEN XPd5_97_5 = Pd5_97_5 ;
Pd5_97_5 = XPd5_97_5 ;
RETAIN XPd6_2_5 ;
IF NOT MISSING(Pd6_2_5) THEN XPd6_2_5 = Pd6_2_5 ;
Pd6_2_5 = XPd6_2_5 ;
RETAIN XPd6_97_5 ;
IF NOT MISSING(Pd6_97_5) THEN XPd6_97_5 = Pd6_97_5 ;
Pd6_97_5 = XPd6_97_5 ;
```

```
RUN ;


/*variable to code if residual differences are in CIs*/
data FOURMRBOOT; set FOURMRBOOT;
d1sig=0; d2sig=0; d3sig=0; d4sig=0; d5sig=0; d6sig=0;
IF d1 lt Pd1_2_5 then d1sig=1; IF d1 gt Pd1_97_5 then d1sig=1;
IF d2 lt Pd2_2_5 then d2sig=1; IF d2 gt Pd2_97_5 then d2sig=1;
IF d3 lt Pd3_2_5 then d3sig=1; IF d3 gt Pd3_97_5 then d3sig=1;
IF d4 lt Pd4_2_5 then d4sig=1; IF d4 gt Pd4_97_5 then d4sig=1;
IF d5 lt Pd5_2_5 then d5sig=1; IF d5 gt Pd5_97_5 then d5sig=1;
IF d6 lt Pd6_2_5 then d6sig=1; IF d6 gt Pd6_97_5 then d6sig=1;
run;

/*COMPUTING SIGNIFICANT FREQUENCIES */
proc freq data=FOURMRBOOT NOPRINT;
tables d1sig /OUT=F1;
tables d2sig /OUT=F2;
tables d3sig /OUT=F3;
tables d4sig /OUT=F4;
tables d5sig /OUT=F5;
tables d6sig /OUT=F6;
run;

DATA F1A; SET F1; IF D1SIG=1; RENAME COUNT = D1_COUNT; LABEL COUNT =
'D1_COUNT'; DROP PERCENT D1SIG;RUN;
DATA F2A; SET F2; IF D2SIG=1; RENAME COUNT = D2_COUNT; LABEL COUNT =
'D2_COUNT'; DROP PERCENT D2SIG;RUN;
DATA F3A; SET F3; IF D3SIG=1; RENAME COUNT = D3_COUNT; LABEL COUNT =
'D3_COUNT'; DROP PERCENT D3SIG;RUN;
DATA F4A; SET F4; IF D4SIG=1; RENAME COUNT = D4_COUNT; LABEL COUNT =
'D4_COUNT'; DROP PERCENT D4SIG;RUN;
DATA F5A; SET F5; IF D5SIG=1; RENAME COUNT = D5_COUNT; LABEL COUNT =
'D5_COUNT'; DROP PERCENT D5SIG;RUN;
DATA F6A; SET F6; IF D6SIG=1; RENAME COUNT = D6_COUNT; LABEL COUNT =
'D6_COUNT'; DROP PERCENT D6SIG;RUN;

DATA FREQ; MERGE F1A F2A F3A F4A F5A F6A;
RUN;

DATA FOURMRBOOT; MERGE FOURMRBOOT FREQ; RUN;

DATA FOURMRBOOT
(DROP = XD1_COUNT XD2_COUNT XD3_COUNT XD4_COUNT XD5_COUNT
XD6_COUNT) ;
SET FOURMRBOOT ;
RETAIN XD1_COUNT  ;
IF NOT MISSING(D1_COUNT) THEN XD1_COUNT = D1_COUNT ;
D1_COUNT = XD1_COUNT ;
RETAIN XD2_COUNT  ;
IF NOT MISSING(D2_COUNT) THEN XD2_COUNT = D2_COUNT ;
D2_COUNT = XD2_COUNT ;
```

```
RETAIN XD3_COUNT  ;
IF NOT MISSING(D3_COUNT) THEN XD3_COUNT = D3_COUNT ;
D3_COUNT = XD3_COUNT ;
RETAIN XD4_COUNT  ;
IF NOT MISSING(D4_COUNT) THEN XD4_COUNT = D4_COUNT ;
D4_COUNT = XD4_COUNT ;
RETAIN XD5_COUNT  ;
IF NOT MISSING(D5_COUNT) THEN XD5_COUNT = D5_COUNT ;
D5_COUNT = XD5_COUNT ;
RETAIN XD6_COUNT  ;
IF NOT MISSING(D6_COUNT) THEN XD6_COUNT = D6_COUNT ;
D6_COUNT = XD6_COUNT ;
RUN ;


DATA FOURMRBOOT; SET FOURMRBOOT;
CN = &TNOBS*&NBOOT;
D1SIGRAT = ((D1_COUNT)/CN);
D2SIGRAT = ((D2_COUNT)/CN);
D3SIGRAT = ((D3_COUNT)/CN);
D4SIGRAT = ((D4_COUNT)/CN);
D5SIGRAT = ((D5_COUNT)/CN);
D6SIGRAT = ((D6_COUNT)/CN);
RUN;

DATA FOURMRBOOT
(DROP = XD1SIGRAT XD2SIGRAT XD3SIGRAT XD4SIGRAT XD5SIGRAT XD6SIGRAT) ;
SET FOURMRBOOT ;
RETAIN XD1SIGRAT  ;
IF NOT MISSING(D1SIGRAT) THEN XD1SIGRAT = D1SIGRAT ;
D1SIGRAT = XD1SIGRAT ;
RETAIN XD2SIGRAT  ;
IF NOT MISSING(D2SIGRAT) THEN XD2SIGRAT = D2SIGRAT ;
D2SIGRAT = XD2SIGRAT ;
RETAIN XD3SIGRAT  ;
IF NOT MISSING(D3SIGRAT) THEN XD3SIGRAT = D3SIGRAT ;
D3SIGRAT = XD3SIGRAT ;
RETAIN XD4SIGRAT  ;
IF NOT MISSING(D4SIGRAT) THEN XD4SIGRAT = D4SIGRAT ;
D4SIGRAT = XD4SIGRAT ;
RETAIN XD5SIGRAT  ;
IF NOT MISSING(D5SIGRAT) THEN XD5SIGRAT = D5SIGRAT ;
D5SIGRAT = XD5SIGRAT ;
RETAIN XD6SIGRAT  ;
IF NOT MISSING(D6SIGRAT) THEN XD6SIGRAT = D6SIGRAT ;
D6SIGRAT = XD6SIGRAT ;
RUN ;


/*ITERATIVELY SAVE BOOTSTRAP RESULTS*/
data newB; set summaryB;
run;
```

```
data summaryB; set newB FOURMRBOOT;
run;


%end;
%mend split;
%MACRO Condloop;

        %do i=1 %to &n;
%RESIDS(NSIM=&&NSIM&i,NOBS=&&NOBS&i,TNOBS=&&Tnobs&i,FILE=&&FILE&i,S
1A1=&&S1A1&i,S1B1=&&S1B1&i,S1A2=&&S1A2&i,S1B2=&&S1B2&i,S1CP=&&S1CP&i,S
2A1=&&S2A1&i,
S2B1=&&S2B1&i,S2A2=&&S2A2&i,S2B2=&&S2B2&i,S2CP=&&S2CP&i,S3A1=&&S3A1&i,
S3B1=&&S3B1&i,S3A2=&&S3A2&i,S3B2=&&S3B2&i,S3CP=&&S3CP&i,
S4A1=&&S4A1&i,S4B1=&&S4B1&i,S4A2=&&S4A2&i,S4B2=&&S4B2&i,S4CP=&&S4CP&i)
;
        %end;
        %mend condloop;
%condloop; run;

DATA A.RESIDS; SET SUMMARY2; RUN;
DATA A.RESIDSB; SET SUMMARY2B; RUN;
```

APPENDIX E

SAS PROGRAM FOR ANALYSIS OF DELTA Z AND GCD

```
OPTIONS PS=59 LS=80 REPLACE NONOTES;
FILENAME NULLOG DUMMY 'C:\NULL';
PROC PRINTTO LOG=NULLOG;

/*/*CREATE DATA SET OF CONDITIONS FROM CSV*/*/;
PROC IMPORT OUT= WORK.conds
      DATAFILE= "D:\Sync\Science Binders\028-dissertation\028-
code\DZGCDRUN\condlg2.csv"
      DBMS=CSV REPLACE;
    GETNAMES=YES;
    DATAROW=2;
      GUESSINGROWS=500;
RUN;


/*/*CREATE MACRO VARIABLES FROM CONDITIONS LIST*/*/;
data conds; length ii $8; set conds;
i+1;
ii=left(put(i, 8.));
call symput ('FILE'||ii, FILE);
call symput ('NSIM'||ii, NSIM);
call symput ('NOBS'||ii, NOBS);
call symput ('TNOBS'||ii,TNOBS);
call symput ('JKNOBS'||ii,JKNOBS);

call symput ('S1A1'||ii, S1A1);
call symput ('S1B1'||ii, S1B1);
call symput ('S1A2'||ii, S1A2);
call symput ('S1B2'||ii, S1B2);
call symput ('S1CP'||ii, S1CP);

call symput ('S2A1'||ii, S2A1);
call symput ('S2B1'||ii, S2B1);
call symput ('S2A2'||ii, S2A2);
call symput ('S2B2'||ii, S2B2);
call symput ('S2CP'||ii, S2CP);

call symput ('S3A1'||ii, S3A1);
call symput ('S3B1'||ii, S3B1);
call symput ('S3A2'||ii, S3A2);
call symput ('S3B2'||ii, S3B2);
call symput ('S3CP'||ii, S3CP);

call symput ('S4A1'||ii, S4A1);
call symput ('S4B1'||ii, S4B1);
call symput ('S4A2'||ii, S4A2);
call symput ('S4B2'||ii, S4B2);
call symput ('S4CP'||ii, S4CP);

call symput ('n', _n_);
drop i;
run;


/*/*READ IN CONDITION DATA FILE AND INVOKE SPLIT MACRO*/*/;
```

```
%macro
JACKDZ(NSIM,NOBS,TNOBS,JKNOBS,FILE,S1A1,S1B1,S1A2,S1B2,S1CP,S2A1,S2B1,S
2A2,S2B2,S2CP,S3A1,S3B1,S3A2,S3B2,S3CP,S4A1,S4B1,S4A2,S4B2,S4CP);


DATA SIMALL; SET a.&file;
run;

%split(nobs=&&nobs&i,TNOBS=&&Tnobs&i, NSIM=&&nSIM&i,
JKNOBS=&&JKNOBS&i);
run;

/*/*/*CLEAR WORK LIBRARY*/*/;
PROC DATASETS LIB=WORK KILL NOLIST;
RUN;
QUIT;
;
%mend JACKDZ;
run;
quit;


/*/*ANALYZE CONDITION BY SIMULATION REPLICATION*/*/;
%MACRO split (nobs, TNOBS, NSIM, JKNOBS);
%do k=1 %to &&nsim&i;

Data SIM; set SIMALL(where=(Rep=&k)); run;


data jack;
      do replicate = 1 to numrecs;
            do rec = 1 to numrecs;
                  set SIM nobs=numrecs point=rec;
                  if replicate ^= rec then output;
            end;
      end;
stop;
run;

/*Model 0*/
PROC CALIS data=JACK method=ML cov outest=F0 NOPRINT ;
BY REPLICATE;
LINEQS
Y= C0 INTERCEPT+E0;
VARIANCE
E0=EE0;
run; quit;

/*Model 1*/
PROC CALIS data=JACK method=ML cov outest=F1 noprint;
BY REPLICATE;
LINEQS
M1=a1 X + E1,
Y=c X + b1 M1 + E2;
VARIANCE
E1=EE1,
```

```
E2=EE2;
run; quit;


/*Model 2*/
PROC CALIS data=JACK method=ML cov outest=F2 noprint;
BY REPLICATE;
LINEQS
M2=a2 X + E3,
Y=c X + b2 M2 + E4;
VARIANCE
E3=EE3,
E4=EE4;
run; quit;

/*Model 3*/
PROC CALIS data=JACK method=ML cov outest=F3 noprint;
BY REPLICATE;
LINEQS
M1=a1 X + E5,
M2=a2 X + E6,
Y=c X + b1 M1 + b2 M2 + E7;
VARIANCE
E5=EE5,
E6=EE6,
E7=EE7;
run; quit;

/*ZERO*/
data a;
set F0;
if _TYPE_='PARMS'; C0i=C0; EE0i=EE0;
DROP _TYPE_ _NAME_ _RHS_ ;
KEEP REPLICATE C0i EE0i;
RUN;

data b;
set F0;
if _TYPE_ = 'STDERR'; sdC0i=C0; varC0i= C0*C0;
DROP _TYPE_ _NAME_ _RHS_;
KEEP REPLICATE sdC0i varC0i;
RUN;

/*ONE*/
data C;
set F1;
if _TYPE_='PARMS'; a1i=a1;  b1i=b1; CP1i=C; EE1i=EE1; EE2i=EE2;
DROP _TYPE_ _NAME_ _RHS_ _Add1;
KEEP REPLICATE a1i b1i CP1i EE1i EE2i;
RUN;

data D;
set F1;
if _TYPE_ = 'STDERR'; sda1i=a1; vara1i= a1*a1;
sdb1i=b1; varb1i = b1*b1; SDCP1i=C; VARCP1i=C*C;
DROP _TYPE_ _NAME_ _RHS_ _Add1;
```

```
KEEP REPLICATE sda1i sdb1i sdCP1i vara1i varb1i varCP1i;
RUN;

/*TWO*/
data E;
set F2;
if _TYPE_='PARMS'; a2i=a2;  b2i=b2; CP2i=C; EE3i=EE3; EE4i=EE4;
DROP _TYPE_ _NAME_ _RHS_ _Add1 ;
KEEP REPLICATE a2i b2i CP2i EE3i EE4i;
RUN;

data F;
set F2;
if _TYPE_ = 'STDERR'; sda2i=a2; vara2i= a2*a2;
sdb2i=b2; varb2i = b2*b2; SDCP2i=C; VARCP2i=C*C;
DROP _TYPE_ _NAME_ _RHS_  _Add1;
KEEP REPLICATE sda2i sdb2i sdCP2i vara2i varb2i varCP2i;
RUN;

/*THREE*/
data G;
set F3;
if _TYPE_='PARMS'; a13i=a1; a23i=a2; b13i=b1; b23i=b2; CP3i=C;
EE5i=EE5; EE6i=EE6; EE7i=EE7;
DROP _TYPE_ _NAME_ _RHS_ _Add1;
KEEP REPLICATE a13i a23i b13i b23i CP3i EE5i EE6i EE7i;
RUN;

data H;
set F3;
if _TYPE_ = 'STDERR'; sda13i=a1; vara13i= a1*a1; sda23i=a2;
vara23i=a2*a2;
sdb13i=b1; varb13i = b1*b1; sdb23i=b2; varb23i=b2*b2; SDCP3i=C;
VARCP3i=C*C;
DROP _TYPE_ _NAME_ _RHS_ _Add1;
KEEP REPLICATE sda13i sda23i sdb13i sdb23i SDCP3i vara13i vara23i
varb13i varb23i VARCP3i;
RUN;

data I;
set F3;
if _TYPE_ = "COV" AND _NAME_ = "a1"; cova13a23i=a2;
DROP _TYPE_ _NAME_ _RHS_ _Add1;
KEEP REPLICATE cova13a23i;
RUN;

data J;
set F3;
if _TYPE_ = "COV" AND _NAME_ = "b1"; covb13b23i=b2;
DROP _TYPE_ _NAME_ _RHS_  _Add1;
KEEP REPLICATE covb13b23i;
RUN;

data K; merge A B C D E F G H I J; run;

DATA K; SET K;
```

```
/*mediation point estimates*/
a1b1i=a1i*b1i;
a2b2i=a2i*b2i;
a13b13i=a13i*b13i;
a23b23i=a23i*b23i;
totmedi=a13b13i+a23b23i;
/*standard error components*/
a1a1i = a1i*a1i;
b1b1i = b1i*b1i;
a13a13i = a13i*a13i;
b13b13i = b13i*b13i;
a1sqvarb1i = a1a1i*varb1i;
b1sqvara1i = b1b1i*vara1i;
a13sqvarb13i = a13a13i*varb13i;
b13sqvara13i = b13b13i*vara13i;

a2a2i=a2i*a2i;
b2b2i=b2i*b2i;
a23a23i=a23i*a23i;
b23b23i=b23i*b23i;
a2sqvarb2i= a2a2i*varb2i;
b2sqvara2i = b2b2i*vara2i;
a23sqvarb23i= a23a23i*varb23i;
b23sqvara23i = b23b23i*vara23i;


/*standard errors*/
sa1b1i = sqrt(a1sqvarb1i+b1sqvara1i);
sa2b2i = sqrt(a2sqvarb2i+b2sqvara2i);
sa13b13i = sqrt(a13sqvarb13i+b13sqvara13i);
sa23b23i = sqrt(a23sqvarb23i+b23sqvara23i);
stotmi =
sqrt(a13sqvarb13i+b13sqvara13i+a23sqvarb23i+b23sqvara23i+2*a13i*a23i*co
vb13b23i+2*b13i*b23i*cova13a23i);

z0i = C0i / sdc0i;
z1a1b1i = a1b1i / sa1b1i;
z2a2b2i = a2b2i / sa2b2i;
z3totmi = totmedi / stotmi;

za13b13i = a13b13i / sa13b13i;
za23b23i = a23b23i / sa23b23i;
run;

DATA JACK; MERGE JACK K; BY REPLICATE; RUN;

/*Analyzing original sample*/
/*Model 0*/
PROC CALIS data=SIM method=ML cov outest=F0O NOPRINT ;
BY FILE REP;
LINEQS
Y= C0 INTERCEPT+E0;
VARIANCE
E0=EE0;
run; quit;
```

```
/*Model 1*/
PROC CALIS data=SIM method=ML cov outest=F1O noprint;
BY FILE REP;
LINEQS
M1=a1 X + E1,
Y=c X + b1 M1 + E2;
VARIANCE
E1=EE1,
E2=EE2;
run; quit;


/*Model 2*/
PROC CALIS data=SIM method=ML cov outest=F2O noprint;
BY FILE REP;
LINEQS
M2=a2 X + E3,
Y=c X + b2 M2 + E4;
VARIANCE
E3=EE3,
E4=EE4;
run; quit;

/*Model 3*/
PROC CALIS data=SIM method=ML cov outest=F3O noprint;
BY FILE REP;
LINEQS
M1=a1 X + E5,
M2=a2 X + E6,
Y=c X + b1 M1 + b2 M2 + E7;
VARIANCE
E5=EE5,
E6=EE6,
E7=EE7;
run; quit;

/*ZERO*/
data L;
set F0O;
if _TYPE_='PARMS'; C0=C0; EE0=EE0;
/*REPLICATE = 0;*/
DROP _TYPE_ _NAME_ _RHS_ ;
KEEP REP FILE C0 EE0 ;
RUN;

data M;
set F0O;
if _TYPE_ = 'STDERR'; sdC0=C0; varC0= C0*C0;
/*REPLICATE = 0;*/
DROP _TYPE_ _NAME_ _RHS_;
KEEP REP FILE sdC0 varC0 ;
RUN;

/*ONE*/
data N;
set F1O;
```

```
if _TYPE_='PARMS'; a1=a1;  b1=b1; CP1=C; EE1=EE1; EE2=EE2;
/*REPLICATE = 0;*/
DROP _TYPE_ _NAME_ _RHS_ _Add1;
KEEP REP FILE A1 b1 CP1 EE1 EE2 ;
RUN;

data O;
set F1O;
if _TYPE_ = 'STDERR'; sda1=a1; vara1= a1*a1;
sdb1=b1; varb1 = b1*b1; SDCP1=C; VARCP1=C*C;
/*REPLICATE = 0;*/
DROP _TYPE_ _NAME_ _RHS_ _Add1;
KEEP REP FILE sda1 sdb1 sdCP1 vara1 varb1 varCP1 ;
RUN;

/*TWO*/
data P;
set F2O;
if _TYPE_='PARMS'; a2=a2;  b2=b2; CP2=C; EE3=EE3; EE4=EE4;
/*REPLICATE = 0;*/
DROP _TYPE_ _NAME_ _RHS_ _Add1 ;
KEEP REP FILE  a2 b2 CP2 EE3 EE4 ;
RUN;

data Q;
set F2O;
if _TYPE_ = 'STDERR'; sda2=a2; vara2= a2*a2;
sdb2=b2; varb2 = b2*b2; SDCP2=C; VARCP2=C*C;
/*REPLICATE = 0;*/
DROP _TYPE_ _NAME_ _RHS_  _Add1;
KEEP REP FILE sda2 sdb2 sdCP2 vara2 varb2 varCP2 ;
RUN;

/*THREE*/
data R;
set F3O;
if _TYPE_='PARMS'; a13=a1; a23=a2; b13=b1; b23=b2; CP3=C; EE5=EE5;
EE6=EE6; EE7=EE7;
/*REPLICATE = 0;*/
DROP _TYPE_ _NAME_ _RHS_ _Add1;
KEEP  REP FILE a13 a23 b13 b23 CP3 EE5 EE6 EE7 ;
RUN;

data S;
set F3O;
if _TYPE_ = 'STDERR'; sda13=a1; vara13= a1*a1; sda23=a2; vara23=a2*a2;
sdb13=b1; varb13 = b1*b1; sdb23=b2; varb23=b2*b2; SDCP3=C; VARCP3=C*C;
/*REPLICATE = 0;*/
DROP _TYPE_ _NAME_ _RHS_ _Add1;
KEEP  REP FILE sda13 sda23 sdb13 sdb23 SDCP3 vara13 vara23 varb13
varb23 VARCP3 ;
RUN;

data T;
set F3O;
if _TYPE_ = "COV" AND _NAME_ = "a1"; cova13a23=a2;
```

```
/*REPLICATE = 0;*/
DROP _TYPE_ _NAME_ _RHS_ _Add1;
KEEP REP  FILE cova13a23 ;
RUN;

data U;
set F3O;
if _TYPE_ = "COV" AND _NAME_ = "b1"; covb13b23=b2;
/*REPLICATE = 0;*/
DROP _TYPE_ _NAME_ _RHS_  _Add1;
KEEP REP FILE covb13b23 ;
RUN;

data V; merge L M N O P Q R S T U; BY REP;run;

DATA V; SET V;
/*mediation point estimates*/
a1b1=a1*b1;
a2b2=a2*b2;
a13b13=a13*b13;
a23b23=a23*b23;
totmed=a13b13+a23b23;
/*standard error components*/
a1a1 = a1*a1;
b1b1 = b1*b1;
a13a13 = a13*a13;
b13b13 = b13*b13;
a1sqvarb1 = a1a1*varb1;
b1sqvara1 = b1b1*vara1;
a13sqvarb13 = a13a13*varb13;
b13sqvara13 = b13b13*vara13;

a2a2=a2*a2;
b2b2=b2*b2;
a23a23=a23*a23;
b23b23=b23*b23;
a2sqvarb2= a2a2*varb2;
b2sqvara2 = b2b2*vara2;
a23sqvarb23= a23a23*varb23;
b23sqvara23 = b23b23*vara23;


/*standard errors*/

sa1b1 = sqrt(a1sqvarb1+b1sqvara1);
sa2b2 = sqrt(a2sqvarb2+b2sqvara2);
sa13b13 = sqrt(a13sqvarb13+b13sqvara13);
sa23b23 = sqrt(a23sqvarb23+b23sqvara23);

stotm =
sqrt(a13sqvarb13+b13sqvara13+a23sqvarb23+b23sqvara23+2*a13*a23*covb13b2
3+2*b13*b23*cova13a23);

z0 = C0 / sdc0;
z1a1b1 = a1b1 / sa1b1;
z2a2b2 = a2b2 / sa2b2;
```

```
z3totm = totmed / stotm;

za13b13 = a13b13 / sa13b13;
za23b23 = a23b23 / sa23b23;
run;

DATA JACK; MERGE JACK V; RUN;


DATA JACK
(DROP = XC0 XEE0 XsdC0 XvarC0 Xa1 Xb1 XEE1 XEE2 XCP1 Xsda1 Xvara1 Xsdb1
Xvarb1 XSDCP1 XVARCP1 Xa2 Xb2 XEE3 Xee4 XCP2 Xsda2 Xvara2
Xsdb2 Xvarb2 XSDCP2 XVARCP2 XEE5 XEE6 XEE7 XA13 XA23 XB13 Xb23 XCP3
Xsda13 xvara13
xsda23 xvara23 xsdb13 xvarb13 xsdb23 xvarb23 XSDCP3 XVARCP3 Xcova13a23
Xcovb13b23
Xa1b1 Xa2b2 Xa13b13 Xa23b23 Xtotmed Xa1a1 Xb1b1 Xa13a13 Xb13b13
Xa1sqvarb1 Xb1sqvara1
Xa13sqvarb13 Xb13sqvara13 Xa2a2 Xb2b2 Xa23a23 Xb23b23 Xa2sqvarb2
Xb2sqvara2
Xa23sqvarb23 Xb23sqvara23 Xsa1b1 Xsa2b2 Xsa13b13 Xsa23b23 Xstotm Xz0
Xz1a1b1
Xz2a2b2 Xz3totm Xza13b13 Xza23b23) ;
SET JACK;
RETAIN XC0 ; IF NOT MISSING(C0) THEN XC0 = C0 ; C0 = XC0 ;
RETAIN XEE0 ; IF NOT MISSING(EE0) THEN XEE0 = EE0 ; EE0 = XEE0 ;
RETAIN XsdC0 ; IF NOT MISSING(sdC0) THEN XsdC0 = sdC0 ; sdC0 = XsdC0 ;
RETAIN XvarC0 ; IF NOT MISSING(varC0) THEN XvarC0 = varC0 ; varC0 =
XvarC0 ;
RETAIN Xa1 ; IF NOT MISSING(a1) THEN Xa1 = a1 ; a1 = Xa1 ;
RETAIN Xb1 ;IF NOT MISSING(b1) THEN Xb1 = b1 ; b1 = Xb1 ;
RETAIN XEE1 ; IF NOT MISSING(EE1) THEN XEE1 = EE1 ; EE1 = XEE1 ;
RETAIN XEE2 ; IF NOT MISSING(EE2) THEN XEE2 = EE2 ; EE2 = XEE2 ;
RETAIN XCP1 ; IF NOT MISSING(CP1) THEN XCP1 = CP1 ; CP1 = XCP1 ;
RETAIN Xsda1 ; IF NOT MISSING(sda1) THEN Xsda1 = sda1 ; sda1 = Xsda1 ;
RETAIN Xvara1 ; IF NOT MISSING(vara1) THEN Xvara1 = vara1 ; vara1 =
Xvara1 ;
RETAIN Xsdb1 ; IF NOT MISSING(sdb1) THEN Xsdb1 = sdb1 ; sdb1 = Xsdb1 ;
RETAIN Xvarb1 ; IF NOT MISSING(varb1) THEN Xvarb1 = varb1 ; varb1 =
Xvarb1 ;
RETAIN XSDCP1 ; IF NOT MISSING(SDCP1) THEN XSDCP1 = SDCP1 ; SDCP1 =
XSDCP1 ;
RETAIN XVARCP1 ; IF NOT MISSING(VARCP1) THEN XVARCP1 = VARCP1 ; VARCP1
= XVARCP1 ;
RETAIN Xa2 ; IF NOT MISSING(a2) THEN Xa2 = a2 ; a2 = Xa2 ;
RETAIN Xb2 ; IF NOT MISSING(b2) THEN Xb2 = b2 ; b2 = Xb2 ;
RETAIN XEE3 ; IF NOT MISSING(EE3) THEN XEE3 = EE3 ; EE3 = XEE3 ;
RETAIN Xee4 ; IF NOT MISSING(ee4) THEN Xee4 = ee4 ; ee4 = Xee4 ;
RETAIN XCP2 ; IF NOT MISSING(CP2) THEN XCP2 = CP2 ;CP2 = XCP2 ;
RETAIN Xsda2 ; IF NOT MISSING(sda2) THEN Xsda2 = sda2 ; sda2 = Xsda2 ;
RETAIN Xvara2 ; IF NOT MISSING(vara2) THEN Xvara2 = vara2 ; vara2 =
Xvara2 ;
RETAIN Xsdb2 ; IF NOT MISSING(sdb2) THEN Xsdb2 = sdb2 ; sdb2 = Xsdb2 ;
RETAIN Xvarb2 ; IF NOT MISSING(varb2) THEN Xvarb2 = varb2 ; varb2 =
Xvarb2 ;
```

```
RETAIN XSDCP2 ; IF NOT MISSING(SDCP2) THEN XSDCP2 = SDCP2 ; SDCP2 =
XSDCP2 ;
RETAIN XVARCP2 ;IF NOT MISSING(VARCP2) THEN XVARCP2 = VARCP2 ; VARCP2 =
XVARCP2 ;
RETAIN XEE5 ; IF NOT MISSING(EE5) THEN XEE5 = EE5 ;EE5 = XEE5 ;
RETAIN XEE6 ; IF NOT MISSING(EE6) THEN XEE6 = EE6 ; EE6 = XEE6 ;
RETAIN XEE7 ; IF NOT MISSING(EE7) THEN XEE7 = EE7 ; EE7 = XEE7 ;
RETAIN XA13 ; IF NOT MISSING(A13) THEN XA13 = A13 ; A13 = XA13 ;
RETAIN XA23 ; IF NOT MISSING(A23) THEN XA23 = A23 ; A23 = XA23 ;
RETAIN XB13 ; IF NOT MISSING(B13) THEN XB13 = B13 ; B13 = XB13 ;
RETAIN Xb23 ;IF NOT MISSING(b23) THEN Xb23 = b23 ;b23 = Xb23 ;
RETAIN XCP3 ; IF NOT MISSING(CP3) THEN XCP3 = CP3 ; CP3 = XCP3 ;
RETAIN Xsda13 ; IF NOT MISSING(sda13) THEN Xsda13 = sda13 ;sda13 =
Xsda13 ;
RETAIN Xvara13 ;IF NOT MISSING(vara13) THEN Xvara13 = vara13 ;vara13 =
Xvara13 ;
RETAIN Xsda23 ; IF NOT MISSING(sda23) THEN Xsda23 = sda23; sda23 =
Xsda23;
RETAIN Xvara23; IF NOT MISSING(vara23) THEN Xvara23 = vara23;vara23 =
Xvara23;
RETAIN Xsdb13 ; IF NOT MISSING(sdb13) THEN Xsdb13 = sdb13; sdb13 =
Xsdb13;
RETAIN Xvarb13; IF NOT MISSING(varb13) THEN Xvarb13 = varb13;varb13 =
Xvarb13;
RETAIN Xsdb23; IF NOT MISSING(sdb23) THEN Xsdb23 = sdb23; sdb23 =
Xsdb23;
RETAIN Xvarb23; IF NOT MISSING(varb23) THEN Xvarb23 = varb23; varb23 =
Xvarb23;
RETAIN XSDCP3 ;IF NOT MISSING(SDCP3) THEN XSDCP3 = SDCP3;SDCP3 =
XSDCP3;
RETAIN XVARCP3; IF NOT MISSING(VARCP3) THEN XVARCP3 = VARCP3;VARCP3 =
XVARCP3;
RETAIN Xcova13a23; IF NOT MISSING(cova13a23) THEN Xcova13a23 =
cova13a23;cova13a23 = Xcova13a23;
RETAIN Xcovb13b23;IF NOT MISSING(covb13b23) THEN Xcovb13b23 =
covb13b23; covb13b23 = Xcovb13b23;
RETAIN Xa1b1; IF NOT MISSING(a1b1) THEN Xa1b1 = a1b1; a1b1 = Xa1b1;
RETAIN Xa2b2; IF NOT MISSING(a2b2) THEN Xa2b2 = a2b2; a2b2 = Xa2b2;
RETAIN Xa13b13; IF NOT MISSING(a13b13) THEN Xa13b13 = a13b13; a13b13 =
Xa13b13;
RETAIN Xa23b23; IF NOT MISSING(a23b23) THEN Xa23b23 = a23b23; a23b23 =
Xa23b23;
RETAIN Xtotmed; IF NOT MISSING(totmed) THEN Xtotmed = totmed; totmed =
Xtotmed;
RETAIN Xa1a1; IF NOT MISSING(a1a1) THEN Xa1a1 = a1a1; a1a1 = Xa1a1;
RETAIN Xb1b1;IF NOT MISSING(b1b1) THEN Xb1b1 = b1b1; b1b1 = Xb1b1;
RETAIN Xa13a13; IF NOT MISSING(a13a13) THEN Xa13a13 = a13a13 ; a13a13 =
Xa13a13;
RETAIN Xb13b13; IF NOT MISSING(b13b13) THEN Xb13b13 = b13b13;b13b13 =
Xb13b13;
RETAIN Xa1sqvarb1;IF NOT MISSING(a1sqvarb1) THEN Xa1sqvarb1 =
a1sqvarb1; a1sqvarb1 = Xa1sqvarb1;
RETAIN Xb1sqvara1;IF NOT MISSING(b1sqvara1) THEN Xb1sqvara1 =
b1sqvara1; b1sqvara1 = Xb1sqvara1;
RETAIN Xa13sqvarb13; IF NOT MISSING(a13sqvarb13) THEN Xa13sqvarb13 =
a13sqvarb13;a13sqvarb13 = Xa13sqvarb13;
```

```
RETAIN Xb13sqvara13; IF NOT MISSING(b13sqvara13) THEN Xb13sqvara13 =
b13sqvara13;b13sqvara13 = Xb13sqvara13;
RETAIN Xa2a2; IF NOT MISSING(a2a2) THEN Xa2a2 = a2a2;a2a2 = Xa2a2;
RETAIN Xb2b2; IF NOT MISSING(b2b2) THEN Xb2b2 = b2b2 ; b2b2 = Xb2b2;
RETAIN Xa23a23; IF NOT MISSING(a23a23) THEN Xa23a23 = a23a23;a23a23 =
Xa23a23;
RETAIN Xb23b23; IF NOT MISSING(b23b23) THEN Xb23b23 = b23b23;b23b23 =
Xb23b23;
RETAIN Xa2sqvarb2; IF NOT MISSING(a2sqvarb2) THEN Xa2sqvarb2 =
a2sqvarb2; a2sqvarb2 = Xa2sqvarb2;
RETAIN Xb2sqvara2; IF NOT MISSING(b2sqvara2) THEN Xb2sqvara2 =
b2sqvara2; b2sqvara2 = Xb2sqvara2;
RETAIN Xa23sqvarb23;IF NOT MISSING(a23sqvarb23) THEN Xa23sqvarb23 =
a23sqvarb23;a23sqvarb23 = Xa23sqvarb23;
RETAIN Xb23sqvara23; IF NOT MISSING(b23sqvara23) THEN Xb23sqvara23 =
b23sqvara23; b23sqvara23 = Xb23sqvara23;
RETAIN Xsa1b1; IF NOT MISSING(sa1b1) THEN Xsa1b1 = sa1b1;sa1b1 =
Xsa1b1;
RETAIN Xsa2b2; IF NOT MISSING(sa2b2) THEN Xsa2b2 = sa2b2; sa2b2 =
Xsa2b2;
RETAIN Xsa13b13; IF NOT MISSING(sa13b13) THEN Xsa13b13 = sa13b13;
sa13b13 = Xsa13b13;
RETAIN Xsa23b23; IF NOT MISSING(sa23b23) THEN Xsa23b23 =
sa23b23;sa23b23 = Xsa23b23;
RETAIN Xstotm; IF NOT MISSING(stotm) THEN Xstotm = stotm;stotm =
Xstotm;
RETAIN Xz0; IF NOT MISSING(z0) THEN Xz0 = z0; z0 = Xz0;
RETAIN Xz1a1b1; IF NOT MISSING(z1a1b1) THEN Xz1a1b1 = z1a1b1; z1a1b1 =
Xz1a1b1;
RETAIN Xz2a2b2; IF NOT MISSING(z2a2b2) THEN Xz2a2b2 = z2a2b2; z2a2b2 =
Xz2a2b2;
RETAIN Xz3totm;IF NOT MISSING(z3totm) THEN Xz3totm = z3totm; z3totm =
Xz3totm;
RETAIN Xza13b13; IF NOT MISSING(za13b13) THEN Xza13b13 = za13b13;
za13b13 = Xza13b13;
RETAIN Xza23b23; IF NOT MISSING(za23b23) THEN Xza23b23 = za23b23;
za23b23 = Xza23b23;
RUN ;


/*compute delta zs and comparisons*/
DATA JACK; SET JACK;
DZ0 =(z0-z0i)/sdc0i;
DZ1 =(z1a1b1-z1a1b1i)/sa1b1i;
DZ2 =(z2a2b2-z2a2b2i)/sa2b2i;
DZ3 =(z3totm-z3totmi)/stotmi;
gcd0 = dz0**2;
gcd1 = dz1**2;
gcd2 = dz2**2;
gcd3 = dz3**2;

/*compute comparisons*/
deltadz1 = DZ0-DZ1;
deltadz2 = DZ1-DZ2;
deltadz3 = DZ1-DZ3;
deltadz4 = DZ0-DZ2;
```

```
deltadz5 = DZ2-DZ3;
deltadz6 = DZ0-DZ3;
deltagcd1 = gcd0-gcd1;
deltagcd2 = gcd1-gcd2;
deltagcd3 = gcd1-gcd3;
deltagcd4 = gcd0-gcd2;
deltagcd5 = gcd2-gcd3;
deltagcd6 = gcd0-gcd3;
RUN;

data b.DZ&file&k; set jack;
run;

%end;
%mend split;


%MACRO Condloop;

      %do i=1 %to &n;
%JACKDZ(NSIM=&&NSIM&i,NOBS=&&NOBS&i,TNOBS=&&Tnobs&i,JKNOBS=&&JKNOBS&i,
FILE=&&FILE&i,S1A1=&&S1A1&i,S1B1=&&S1B1&i,S1A2=&&S1A2&i,S1B2=&&S1B2&i,S
1CP=&&S1CP&i,S2A1=&&S2A1&i,
S2B1=&&S2B1&i,S2A2=&&S2A2&i,S2B2=&&S2B2&i,S2CP=&&S2CP&i,S3A1=&&S3A1&i,S
3B1=&&S3B1&i,S3A2=&&S3A2&i,S3B2=&&S3B2&i,S3CP=&&S3CP&i,
S4A1=&&S4A1&i,S4B1=&&S4B1&i,S4A2=&&S4A2&i,S4B2=&&S4B2&i,S4CP=&&S4CP&i);
      %end;
      %mend condloop;
%condloop; run;
```

APPENDIX F

SECONDARY PILOT WITH A SINGLE DATASET

A single dataset was generated to demonstrate how residual differences or delta z could differentiate mediators in individual observations. For simplicity and illustration of the concept, data were generated with very large effects through Mediator 1 for only one subpopulation ($a1 = b1 = 9$). All other effects were zero for all subpopulations. The sample size was N=200. Additionally, because the direct effects of X absorb unmodeled mediator effects, analysis of residual differences uses residuals from a regression model where X has been excluded as a predictor of Y to test differences between the two mediators directly. The analyses of delta z and gCd include X as a predictor of Y.

The example compares residuals for a model with no predictors of Y, and a model where Mediator 1 is the only predictor of Y. Differences in delta z and gCd for the mediated effect (i.e., $a1b1$) are also tested. Dependent variables are the differences in residuals, delta z and gCd, the absolute value of the differences, and the rank ordering of the differences. The independent variable is known subpopulation.

Results

Residual Differences. The mean residual difference for known subpopulations was not significant, $F\,(3, 196) = .65, p = .59$. However, Levene's test showed a significant difference in variance between subpopulations, $F\,(3, 196) = 29.64, p < .0001$. The mean residual difference for $S1$ was $(M = 7.12, SD = 83.90)$. For $S2$, it was $(M = -1.13, SD = 9.05)$. The mean residual difference for $S3$ was $(M = -2.52, SD = 9.12)$ and for $S4$ was $(M = -3.47, SD = 6.86)$.

Residual Differences (Absolute value). A second analysis found a significant effect of subpopulation on the absolute value of the residual difference, $F\,(3, 196) =$

$89.73, p < .0001$, such the mean absolute value residual difference of $S1$ was significantly different from the other three subpopulations, $p < .0001$. The mean absolute value residual difference for $S1$ was $(M = 69.83, SD = 45.99)$. For $S2$, it was $(M = 7.70, SD = 4.77)$. The mean absolute value residual difference for $S3$ was $(M = 7.64, SD = 5.49)$ and for $S4$ was $(M = 6.23, SD = 4.43)$. Levene's test was also significant, $F(3, 196) = 20.25, p < .0001$.

Residual Differences (Ranked absolute value). There was a significant effect of subpopulation predicting the rank ordering of the absolute value residual difference, $F(3, 196) = 54.51, p < .0001$, such that observations from $S1$ were more likely to have larger residual differences than the other subpopulations. The mean ranking for $S1$ was $(M = 167.46, SD = 37.98)$. For $S2$, it was $(M = 82.98, SD = 42.40)$. The mean ranking for $S3$ was $(M = 80.85, SD = 46.90)$ and for S4 was $(M = 70.71, SD = 44.47)$. Levene's test was not significant.

Delta z. There was a significant effect of subpopulation predicting delta z for the mediated effect, $a1b1$, $F(3, 196) = 12.83, p < .0001$. $S1$ differed from the other three subpopulations in that the delta z difference was negative, of a greater magnitude, and had more variance than the other three subpopulations. The mean delta z difference for $S1$ was $(M = -.04, SD = .07)$. For $S2$, it was $(M = .00, SD = .01)$. The mean delta z difference for $S3$ was $(M = .00, SD = .02)$ and for $S4$ was $(M = .00, SD = .02)$. Levene's test was also significant, $F(3, 196) = 4.58, p = .004$.

Delta z *(Absolute value)*. There was also a significant effect of subpopulation on the absolute value of the delta z difference, $F(3, 196) = 9.55, p < .0001$, such the mean absolute value delta z difference of $S1$ was significantly different from the other three subpopulations, $p < .0001$. The mean absolute value delta z difference

for $S1$ was ($M = 0.4, SD = .07$). For $S2$, it was ($M = .01, SD = .01$). The mean absolute value delta z difference for $S3$ was ($M = .01, SD = .01$) and for $S4$ was ($M = .01, SD = .01$). Levene's test was also significant, $F\ (3, 196) = 4.31, p = .006$.

Delta *z (Ranked)*. There was a significant effect of subpopulation predicting the rank ordering of the delta z difference, $F\ (3, 196) = 4.42, p = .005$, such that observations from $S1$ were more likely to have smaller delta z differences than the other subpopulations. The mean ranking for $S1$ was ($M = 76.16\ SD = 67.67$). For $S2$, it was ($M = 102.73, SD = 50.98$). The mean ranking for $S3$ was ($M = 112.74, SD = 55.23$) and for $S4$ was ($M = 110.37, SD = 50.15$). Levene's test was also significant, $F\ (3, 196) = 8.14, p < .0001$.

Delta *z (Ranked absolute value)*. There was a significant effect of subpopulation predicting the rank ordering of the absolute value of the delta z difference, $F\ (3, 196) = 4.49, p = .005$, such that $S1$ was more likely to have larger absolute value delta z differences than the other subpopulations. The mean ranking for $S1$ was ($M = 124.91\ SD = 64.78$). For $S2$, it was ($M = 86.73, SD = 54.60$). The mean ranking for $S3$ was ($M = 97.90, SD = 55.07$) and for $S4$ was ($M = 92.46, SD = 50.20$). Levene's test was also significant, $F\ (3, 196) = 2.85, p = .04$.

gCd. The effect of subpopulation on gCd difference was not significant.

gCd *(Ranked)*. There was a significant effect of subpopulation predicting the rank ordering of the gCd difference, $F\ (3, 196) = 11.19, p < .0001$, such that $S1$ was more likely to have larger gCd differences than the other subpopulations. The mean ranking for S1 was ($M = 138.15\ SD = 70.16$). For $S2$, it was ($M = 93.00, SD = 48.38$). The mean ranking for $S3$ was ($M = 82.14, SD = 48.61$) and for $S4$ was ($M = 88.71, SD = 44.59$). Levene's test was also significant, $F\ (3, 196) = 7.99, p < .0001$.

Summary

This example illustrates how Model Contrasts in a single dataset can differentiate subpopulations with different mediating effects. In this example, the absolute value of residual differences showed that the subpopulation with an effect through Mediator 1 had significantly larger residual differences with more variance than the subpopulations with no effect through Mediator 1. Similar results were obtained for delta *z* and its absolute value. Subpopulation predicted rank order of the differences, which suggests differences between a subpopulation with a mediated effect and subpopulations without a mediated effect.