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Latent Difference Score Mediation Analysis in Developmental Research: A Monte Carlo Study and Application

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LATENT DIFFERENCE SCORE MEDIATION ANALYSIS IN DEVELOPMENTAL RESEARCH: A MONTE CARLO STUDY AND APPLICATION

by

Melissa Simone

A dissertation submitted in partial fulfillment of the requirements for the degree of

DOCTOR OF PHILOSOPHY

in

Psychology

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2018
ABSTRACT

Latent Difference Score Mediation Analysis in Developmental Research: A Monte Carlo Study and Application

by

Melissa Simone, Master of Art
Utah State University, 2018

Major Professor: Ginger Lockhart, Ph.D.
Department: Psychology

Developmental and prevention researchers aim to determine how maladaptive health behaviors emerge. Mediation analysis offers a tool to identify the processes through which early risk factors influence later health. Recent quantitative developments offer several longitudinal mediation models through which researchers can examine how these effects unfold over time, rather than modeling all effects simultaneously through the application of traditional mediational models. Among existing longitudinal mediation models, latent difference score mediation stands out due to its unique ability to model changes both within and across individuals, as well as its ability to capture non-linear change over time. However, latent difference mediation lacks empirically supported sample size guidelines which has resulted in few applications of this method. To address
this limitation, the aims of this project were threefold: (1) evaluate the performance of various latent difference score mediation model structures through a Monte Carlo simulation study; (2) use the results from the Monte Carlo study to develop a set of empirical sample size guidelines for future use of latent difference score mediation; and (3) apply one of the latent difference score mediation model structures to real prevention data, to examine the underlying processes between disordered eating among adolescent girls and alcohol misuse among adult women.

First, a Monte Carlo simulation study was conducted in which power, parameter and standard error biases, and coverage were examined across several latent difference score mediational model structures and population models to determine the required sample size for each structural and population model. Empirical sample size guidelines were determined in an iterative fashion, in which models with too much power were reevaluated with a smaller sample size, and those with too little power were reevaluated with a larger sample size. The resulting sample size guidelines represent the smallest possible sample size with adequate power, minimal biases, and adequate coverage for each model. Latent difference score mediation was then applied to real prevention data, to examine how disordered eating among adolescent girls exerts its efforts on alcohol misuse among adult women.
LATENT DIFFERENCE SCORE MEDIATION ANALYSIS IN DEVELOPMENTAL RESEARCH: A MONTE CARLO STUDY AND APPLICATION

Melissa Simone

Developmental and prevention researchers aim to determine how unhealthy behaviors emerge. Mediation analysis offers a statistical tool that allows researchers to describe the processes underlying early risk and later health outcomes. Among existing longitudinal mediation models, latent difference score mediation stands out due to its unique ability to capture variations in changes both within and across individuals, as well as its ability to examine non-linear change over time. However, the literature currently lacks sample size guidelines for latent difference mediation models, which has proven to make the use of these models difficult. The current project addresses this limitation by offering an empirical set of sample guidelines for a variety of latent difference mediation score models through a Monte Carlo simulation study. By offering empirical sample size guidelines for latent difference score mediation models, future developmental and prevention researchers can make informed sampling decisions prior to data collection.

Moreover, women who misuse alcohol have been found to experience more severe medical consequences than men. However, minimal research has evaluated how gender specific risk factors influence its onset. The current project addresses this limitation by applying latent difference score mediation to evaluate how disordered eating behaviors among adolescent girls influence alcohol misuse among adult women.
DEDICATION

This dissertation is dedicated to my Nana, Hope Simone, whose humor, love, support, and guidance has helped me throughout each step I’ve taken towards achieving my academic goals. Her recent and unexpected loss has forever changed me – yet, I can only hope that it has helped me approach life more fearlessly and with greater purpose than ever before. I dedicate this dissertation and all work associated with it to her, to represent a fraction of all that she’s given me throughout life.

Melissa Simone
I would first like to thank Dr. Ginger Lockhart for her guidance and support, both professionally and personally, throughout my doctoral training. Her guidance has helped me to find my passions in quantitative research and mediation analysis. Dr. Lockhart has spent countless hours helping me to develop my professional writing style, and has provided me with the skills and tools necessary to produce this work. She has also taught me how to be a woman in academia, how to achieve a work-life balance, and how to live my life less apologetically. I would also like to thank Dr. Christian Geiser for teaching me the fundamental components of structural equation modeling, mixture modeling, test theory, and Mplus statistical software; all of which will help me greatly throughout my career. Similarly, I would like to thank my committee members, Dr. Michael P. Twohig, Dr. Jamison Fargo, Dr. Scott Bates, and Dr. Lisa Boyce for their ongoing support and reviews of this work.

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of my parents and my two sisters who have supported me in more ways than they know, even while living on opposite sides of the country.

Finally, this research uses data from the National Longitudinal Study of Adolescent Health, a program project directed by Kathleen Mullan Harris and designed by J. Richard Udry, Peter S. Bearman, and Kathleen Mullan Harris at the University of North Carolina at Chapel Hill, and funded by grant P01-HD31921 from the Eunice Kennedy Shriver National Institute of Child Health and Development, with cooperative funding from 23 other federal agencies and foundations. Information on how to obtain the Add Health data files is available on the Add Health website (http://www.cpc.unc.edu/addhealth). No direct support was received from grant P01-HD31921 for this project.
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Developmental and prevention researchers are often interested in uncovering the mechanisms through which early risk factors influence distal health outcomes. Mediation analysis is a statistical tool that allows researchers to explain these processes by examining indirect pathways through which underlying mechanisms, or mediators, exert their efforts (MacKinnon, Krull & Lockwood, 2000; Baron & Kenny, 1986). To this end, mediation analysis includes a product term \((ab)\) to explain the combined effect of a predictor on a mediator \((a)\) and a mediator on an outcome \((b)\). Traditional mediation methods are cross-sectional in the sense that they examine all effects simultaneously. Yet, researchers are often interested in understanding how events take place over time to understand the temporal order of events and the developmental period in which problematic behaviors emerge (Kellam, Koretz, & Mościciki, 1999).

Recent quantitative developments expand upon traditional mediation models by offering longitudinal mediational tools to explain how mediational processes unfold over time (Selig & Preacher, 2009). When planning to use mediational methods, researchers are often interested in how many participants are needed to achieve adequate statistical power (Fritz & MacKinnon, 2007). Yet, sample size guidelines are not always readily available for each form of mediation analysis and sample size estimates vary greatly across mediational methods (Muthén & Muthén, 2002). Thus, the current project focuses on a single form of longitudinal mediation.
While longitudinal mediation builds upon traditional mediation by allowing researchers to model change over time, researchers must be careful to select the appropriate model for any given research question. Specifically, longitudinal researchers may have one of several unique goals. Some common goals in developmental research include: (1) identify the direction of change within an individual overtime; (2) identify differences in change across individuals; (3) analyze predictors of change within an individual; and (4) analyze predictors of differences in change across individuals (Baltes & Nesselroade, 1979). Thus, researchers must identify the goal of their research prior to determining which longitudinal mediation model is best suited to answer their questions.

Among existing longitudinal mediation models, latent difference score mediation is possibly the most flexible model, as change is not assumed to be systematic across measurement occasions and researchers may use this method to examine either changes within individuals and differences in change across individuals over time (Selig & Preacher, 2009; MacKinnon, 2008; McArdle & Hamagami, 2001). Through the application of latent difference score mediation developmental and prevention researchers can evaluate whether changes in an early risk factor influence changes in hypothesized underlying processes, which ultimately influence changes in distal health outcomes. However, little is known about the sample size guidelines with this method, which has proven to cause difficulty when determining how large of a sample to collect. Moreover, previous research that has applied latent difference score mediation has not offered model fit information (e.g., Bryan et al., 2016; Reeve, Paul & Butterworth, 2015), thus calling the power of these studies into question. To address this limitation, the current project seeks to develop a set of empirically supported sample size guidelines for
use of latent difference score mediation. After establishing a set of sample size
guidelines, the current study applies latent difference score mediation to real prevention
science data to examine gender specific factors contributing to the etiological course of
alcohol misuse among women.

Alcohol use disorder is a serious psychiatric illness that diminishes individuals
personal and physical well-being and has extensive consequences for societal functioning
(Whiteford et al., 2008). While recent findings suggest that women suffer greater
consequences as a result alcohol misuse than their male counterparts (Substance Abuse
and Mental Health Services Administration, 2014), little is known about gender specific
predictors and the developmental pathways associated with the emergence of alcohol
misuse among adult women. Recent research suggests that psychiatric vulnerabilities
among adolescent girls, namely disordered eating behaviors, may serve as a gender
specific predictor of alcohol misuse among adult women (Ortega-Luyando et al., 2015;
Micali et al., 2015; Harrop & Marlatt, 2010). However very little is known about how
these processes unfold over time. To address this limitation, the current project applied
latent difference score mediation from an ecological perspective (Bronfenbrenner, 1977)
to determine the extent to which social environmental and internal processes mediate the
relation between disordered eating among adolescent girls and alcohol misuse among
adult women.

In sum, the current project has aimed to address the highlighted limitations by
first developing a set of empirically supported sample size guidelines for the use of
latent difference score mediation, and then applying latent difference score mediation to
examine the mechanisms through which disordered eating among adolescent girls
predicts alcohol misuse among adult women. The aims of this two-study project were threefold: (1) evaluate the performance of various latent difference score mediation model structures through a Monte Carlo simulation study; (2) use the results from the Monte Carlo study to develop a set of empirical sample size guidelines for use of latent difference score mediation model structures; and (3) apply one of the latent difference score mediation model structures to determine the extent to which environmental and internal processes mediate the relation between disordered eating among adolescent girls and alcohol misuse among adult women.
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Substance Abuse and Mental Health Services Administration (SAMHSA; 2014). *Results from the 2013 National Survey on Drug Use and Health: Summary of National Findings*. Rockville, MD: Substance Abuse and Mental Health Services Administration.


ABSTRACT

Study 1: A Set of Guidelines for Use of Latent Difference Score Mediation Models: A Monte Carlo Study

Background. Mediation models are used to identify the mechanisms through which one variable influences another. Recent advancements in quantitative methodology offer several longitudinal mediational tools to examine how mediational effects unfold overtime. Among existing longitudinal mediation models, latent difference score mediation stands out due to its unique ability to capture changes both within and across individuals, as well as its ability to examine non-linear change over time. Yet, latent difference mediation models lack empirically supported sample size guidelines across its many possible structures, resulting in few applications of this method. Objective. The goal of the current study is to address this limitation by developing a set of empirically supported sample size estimates across several model structures and population models. Method. A Monte Carlo simulation study was conducted to determine the smallest possible sample size to detect mediation while retaining adequate statistical power, minimal biases, and adequate coverage across all tested models. Results. The resulting empirical sample size guidelines represent the smallest sample size to retain adequate statistical power, minimal bias, and adequate coverage for each tested model. The b pathway, representing the effect of the mediator on the outcome, was found to have a greater impact on the required sample size to detect mediation. Further, while population models with large effect sizes require smaller sample sizes to achieve adequate statistical
power, these models trade power for inflated parameter and standard error bias. This trade-off resulted in bias-adjusted empirical sample estimates among the final sample estimates for certain effect size pairings. Finally, simplicity was considered a virtue, as more complex models required significantly larger samples than more simple model structures. Conclusions. The current study provides researchers with a reference for determining the required sample size for a wide range of single-mediator latent difference score mediational models. The empirical sample size estimates should be interpreted as a lower bound for sample size, as the current study was simulated with normally distributed data with no missing data across measurement occasions.
CHAPTER II

STUDY 1: A SET OF GUIDELINES FOR USE OF LATENT DIFFERENCE SCORE MEDIATION MODELS: A MONTE CARLO STUDY

Introduction

One of the main goals in prevention research is to reduce rates of specific psychological disorders (Kellam, Kortez & Mośicki, 1999). To this end, prevention researchers are interested in identifying early behavioral and sociocontextual risk factors related to psychological disorders, as well as the developmental processes that underlie early risk and later health and variations among these processes (Kellam & Van Horn, 1997). By identifying early risk factors and the processes through which they exert their impacts on psychological health, prevention scientists can identify groups who are at greatest risk of developing a psychological disorder and identify of potential intervention targets that can ultimately reduce risk among those groups (Kellam et al., 1999).

Mediation analysis is a powerful statistical tool that allows researchers to answer substantive questions about how an early risk factor impacts a distal health outcome (MacKinnon, 2008; MacKinnon, Krull & Lockwood, 2000; MacKinnon & Dwyer, 1993; Hansen, 1992), and thus these methods are commonly used within the field of prevention research. Mediation analyses examine two main pathways (see Figure 1.1): the direct effect of a predictor ($X$) on an outcome ($Y$), and the indirect effect of $X$ on $Y$ through a mediator ($M$) ($X \rightarrow M$ and $M \rightarrow Y$; MacKinnon et al., 2000). For example, family
environment may mediate the relationship between disordered eating and alcohol misuse among girls. In this example, girls who engage in disordered eating behaviors may experience increased parent-child conflict in response to their eating patterns, which in turn may result in alcohol misuse. In this example, prevention scientists may be interested in reducing alcohol misuse by targeting parent-child conflict among girls who are engaging in disordered eating behaviors.

![Figure 1.1: Example of a simple mediation model with a single predictor (X), mediator (M) and outcome (Y) variable](image)

Traditional mediation methods examine all tested effects simultaneously, resulting in a cross-sectional analysis that prevents researchers from examining how processes unfold over time. Yet, often times researchers are interested in understanding the temporal order of events and the developmental period in which problematic behaviors emerge (Kellam, Koretz, & Mościciki, 1999). Recent advances in the field of quantitative methodology offer a range of longitudinal mediational tools that can better describe these developmental processes (Selig & Preacher, 2009). When planning to use mediational methods, researchers are often interested in how many participants are
needed to achieve adequate statistical power (Fritz & MacKinnon, 2007). Yet, sample size guidelines are not always readily available and requirements vary greatly across models (Muthén & Muthén, 2002). Thus the current chapter first introduces several longitudinal mediational methods and the questions they are able to answer. Next, the chapter shifts focus to a single longitudinal mediation method with an overall goal of developing a set of empirically supported sample size guidelines for that method with a Monte Carlo simulation study.

**Longitudinal Mediation**

Longitudinal mediation is superior to traditional mediation methods as longitudinal analyses allow researchers to examine how developmental processes unfold over time (Selig & Preacher, 2009). The ability to model change as it occurs is important because it allows researchers to evaluate whether an effect, or outcome, is dependent upon the amount of time elapsed (Selig & Preacher, 2009), and allows researchers to describe the temporal order of effects. Recent quantitative developments offer several possible longitudinal mediation models, including: latent growth curve mediation (Bollen & Curran, 2006), cross-lagged panel mediation (Cole & Maxwell, 2003) and latent difference score mediation (Selig & Preacher, 2009; MacKinnon, 2008; McArdle & Hamagami, 2001). Below, a brief description of each of model is provided. Because developmental researchers are often interested in describing change as it occurs within an
individual or between individuals, the ability to model each of these processes is discussed for each model.

Latent growth curve mediation examines change over time by evaluating the average rate of change and variability in change between individuals (Selig & Preacher, 2009). While latent growth curve mediation models have the capacity to explain variability between individuals and variability within the individual at the variable level, this method does not have the utility to explain variance within the individual for the indirect path (Selig & Preacher, 2009). Moreover, latent growth curve mediation models assume systematic change among the variables of interest over time, and require a minimum of nine measurement occasions in order to explain the temporal order of events (Selig & Preacher, 2009).

Cross-lagged panel mediation models require fewer measurement occasions than latent growth curve models, with a requirement of at least three measurement occasions. Cross-lagged panel mediation requires the predictor, mediation, and outcome variables be measured at all measurement occasions. In this model, it is assumed that a predictor at the first measurement occasion \( X_1 \) predicts a mediator at the second measurement occasion \( M_2 \), which ultimately affects the outcome at time 3 \( Y_3 \) (Selig & Preacher, 2009). While cross-lagged panel mediation requires fewer measurement occasions to model longitudinal effects than does latent growth curve mediation, it does not have the capacity to capture changes within the individual across measurement occasions.

Latent difference score mediation is the only known longitudinal mediation method that directly models both within and between individual variability (Selig & Preacher, 2009), thus offering greater utility to answer questions related to development.
over time. Latent difference score mediation represents change at the variable level as the difference in between adjacent observations, which allows researchers to explain the temporal order of modeled variables and relaxes the assumption of systematic change over time (Selig & Preacher, 2009; McArdle, 2009; MacKinnon, 2008; McArdle & Hamagami, 2001). Thus, latent difference score mediation models are useful when change is expected to vary across measurement occasions (MacKinnon, 2008). Latent difference score mediation offers great utility to examine developmental processes related to prevention science and thus will be the focus of the remainder of this chapter.

**Latent Difference Score Mediation**

Latent difference score mediation models examine change over time among predictors, mediators, and/or outcomes over time. In these models, change is represented as the difference in scores in adjacent observation periods (Selig & Preacher, 2009). The adjacent observations that make up latent difference scores are included in the model as lower-level latent factors. For example, when a predictor ($X$) is modeled as a change score variable ($\Delta X_{1-2}$), $X_1$ and $X_2$ are modeled as unobserved lower-level latent factors with multiple indicators (see Figure 1.2 for an example).
Figure 1.2: Example of a single lower-level latent factor with three observed indicators

Lower-level latent factors are then used to form the latent change score variables. The latent change scores represent the change between adjacent measurement occasions as well as the effect of the variable at the first time point on the change between occasions. See Figure 1.3 for an example.

Figure 1.3: Example of a latent change score variable derived from two lower-level factors
In a full latent difference score mediation model (see Figure 1.4), the predictor \( X \), mediator \( M \), and outcome \( Y \) variables are measured at four measurement occasions (where \( \Delta X_{1-2} \rightarrow \Delta M_{2-3} \rightarrow \Delta Y_{3-4} \)). In such a model, \( \Delta M_{2-3} \) is predicted by both \( \Delta X_{1-2} \) and \( X_1 \), which are labeled as \( \alpha_1 \) and \( \alpha_2 \) in Figure 1.4, respectively. Similarly, \( \Delta Y_{3-4} \) is predicted by both \( \Delta M_{2-3} \) and \( M_2 \), or \( b_1 \) and \( b_2 \), to form the indirect path, as well as \( c'_1 \) and \( c'_2 \) to capture the direct effect of \( X \) on \( Y \). Often times the effect of \( \Delta X_{1-2} \) and \( X_1 \) on \( M_2 \), or \( \alpha_3 \) and \( \alpha_4 \), are also included in the model, as well as the effect of \( \Delta M_{2-3} \) and \( M_2 \), or \( b_3 \) and \( b_4 \), on \( Y_3 \). Similarly, the effect of both \( \Delta X_{1-2} \) and \( X_1 \) on \( Y_3 \), or \( c'_3 \) and \( c'_4 \), can be tested. In a full latent difference score model, where all \( a, b \) and \( c' \) effects are included

\[
X_2 = X_1 + \Delta X_{1-2} \tag{1}
\]

Whereas the change in \( X \) from time 1 – time 2 (\( \Delta X_{1-2} \)) is represented as a function of \( X \) at time 1, where \( \beta_1 \) is the effect of \( X \) at time 1 on the change \( X \) in at time 2 (Selig & Preacher, 2009), and \( \psi \Delta X_{1-2} \) is the variance in \( \Delta X_{1-2} \).

\[
\Delta X_{1-2} = \beta_1 + \psi \Delta X_{1-2} \tag{2}
\]

Further, \( M \) at time 2 is represented as the sum of: (1) the effect of the change in \( X \) from time 1 – time 2 on \( M \) at time two (\( \alpha_3 \)); (2) the effect of \( X \) at time 1 on \( M \) at time 2 (\( \alpha_4 \)), and (3) the variance in \( M_2 \) (\( \psi M_2 \)).
\[ M_2 = \alpha_3 + \alpha_4 + \psi M_2 \] 
\hspace{1cm} (3)

Similarly, \( M \) at time 3 is represented as the sum of \( M \) at time 2 and the change in \( M \) from time 2 – time 3 (\( \Delta M_{2-3} \)):

\[ M_3 = M_2 + \Delta M_{2-3} \] 
\hspace{1cm} (4)

Where the change in \( M \) (\( \Delta M_{2-3} \)) is represented as the sum of: (1) the effect of the change in \( X \) (\( \Delta X_{1-2} \)) on the change in \( M \) from time 2 – time 3 (\( \alpha_1 \)); (2) the effect of \( X \) at time 1 on the change in \( M \) from time 2 – time 3 (\( \alpha_2 \)); (3) a function of \( M \) at time 2, where \( \beta_2 \) is the effect of \( M \) at time 2 on the change in \( M \) from time 2 – time 3; and (4) the variance in \( \Delta M_{2-3} \) (\( \psi M \Delta_{2-3} \)):

\[ \Delta M_{2-3} = \alpha_1 + \alpha_2 + \beta_2 + \psi \Delta M_{2-3} \] 
\hspace{1cm} (5)

Further, \( Y \) at time 3 is represented as the sum of: (1) a function of the change in \( X \) from time 1 to time 2, where \( c'_3 \) is the effect of the change in \( X \) from time 1 – time 2 on \( Y \) at time 3; (2) a function of \( X \) at time 1, where \( c'_4 \) is the effect of \( X \) at time 1 on \( Y \) at time 3; (3) a function of the change in \( M \) from time 2 – time 3, where \( b_3 \) is the effect of the change in \( M \) from time 2 – time 3 on \( Y \) at time 3; (4) a function of \( M \) at time 2, where \( b_4 \) is the effect of \( M \) at time 2 on \( Y \) at time 3; and (5) the variance in \( Y_3 \) (\( \psi Y_3 \)):
\[ Y_3 = c'_3 + c'_4 + b_3 + b_4 + \psi Y_3 \]  \hspace{1cm} (6)

Finally, \( Y \) at time 4 is represented as the sum of \( Y \) at time 3 and the change in \( Y \) from time 3 – time 4:

\[ Y_4 = Y_3 + \Delta Y_{3-4} \]  \hspace{1cm} (7)

Where the change in \( Y \) from time 3 to time 4 (\( \Delta Y_{3-4} \)) is represented as the sum of: (1) a function of the change in \( X \) from time 1 to time 2, where \( c'_1 \) is the effect of the change in \( X \) from time 1 – time 2 on the change in \( Y \) from time 3 to time 4; (2) a function of \( X \) at time 1, where \( c'_2 \) is the effect of \( X \) at time 1 on the change in \( Y \) from time 3 – time 4; (3) a function of the change in \( M \) from time 2 – time 3, where \( b_1 \) is the effect of the change in \( M \) from time 2 – time 3 on the change in \( Y \) from time 3 – time 4; (4) a function of \( M \) at time 2, where \( b_2 \) is the effect of \( M \) at time 2 on the change in \( Y \) from time 3 – time 4; (5) a function of \( Y \) at time 3, where \( \beta_3 \) is the effect of \( Y \) at time 3 on the change in \( Y \) from time 3 – time 4; and (6) the variance in the \( \Delta Y_{3-4} \) variable (\( \psi \Delta Y_{3-4} \)): 

\[ \Delta Y_{3-4} = c'_1 + c'_2 + b_1 + b_2 + \beta_3 + \psi \Delta Y_{3-4} \]  \hspace{1cm} (8)

Latent difference score mediation models examine change in model predictors, mediators, and outcomes dynamically over time, without assuming systematic change within or across measurement occasions. However, there are several important considerations to address in order to capture the mediated effect of interest.
Considerations with Latent Difference Score Mediation

Due to the complex nature of latent difference score mediation models, a wide range of factors such as the role of time, span of the study, the developmental period of interest, the model assumptions and distribution qualities must be considered. Specifically, the role of time is a critical component in latent difference score mediation because researchers need to collect data at the appropriate developmental periods to capture the mediated effect over the course of the study (Selig & Preacher, 2009). For example, a researcher interested in detecting the mediated effect of adolescent social environment on the relationship between late childhood eating patterns and adult alcohol
misuse, must carefully select the amount of time between measurement occasions in order to make sure enough time elapses for the expected effects to take place. That is to say, while the value of disordered eating at the first measurement occasion may predict the family environment scores at the second measurement occasion, latent difference score mediation captures whether the change in a predictor across measurement occasions predicts change in a mediator. Thus, failure to measure study variables at the appropriate developmental period, or with insufficient time between measurement occasions, may result in insignificant findings, or change, across occasions. Therefore, when considering the role of time, the choice of developmental period, space between data collection, and the span of a study must be determined on a case-by-case basis (Selig & Preacher, 2009), as differences in these components directly impact the interpretation of the change in each construct.

Mediation analyses contain several assumptions (MacKinnon, 2008), which should be checked prior to examining any data. Specifically, latent difference score mediation models assume that there is no interaction between the X and M variables, and that the causal ordering and direction of effects are accurate (e.g. $X \rightarrow M \rightarrow Y$, not $Y \rightarrow M \rightarrow X$; Lockhart, MacKinnon & Ulrich, 2011; MacKinnon, Fairchild & Fritz, 2007). Further, it is assumed that the analyses use valid and reliable measures (Lockhart et al., 2011), that there is no misspecification due to unmeasured variables (Holland, 1988; James & Brett, 1984), and that residual variables are not correlated with the mediated effect (McDonald, 1997).

Mediation analysis involves obtaining at least one product between two coefficients and the underlying distribution of this product is asymmetrical, which
violates the standard regression assumption of normality (MacKinnon, Lockwood & Williams, 2004). Thus, resampling methods (e.g. Jackknife, Percentile Bootstrap and Bias-corrected Bootstrap; MacKinnon et al., 2004; Efron & Tibshirani, 1993; Mosteller & Tukey, 1977) are typically used to adjust for bias. Resampling techniques generate a variety of datasets by resampling observations from the original dataset to test distributional hypotheses and generate confidence intervals (Williams & MacKinnon, 2008). Of current resampling techniques, the bias-corrected bootstrap method provides the most accurate confidence intervals and more power than normal theory and non-corrected methods (MacKinnon et al., 2004).

Types of Indirect Effects

In order to capture change in $X, M$ and $Y$, the best possible latent difference score mediation model includes four measurement occasions ($\Delta X_{1-2} \rightarrow \Delta M_{2-3} \rightarrow \Delta Y_{3-4}$). In this model, there are several different indirect effects that may be calculated (see Table 1.1; Selig & Preacher, 2009). Because each indirect effect includes different forms of the predictor, mediator and outcome variables, resulting in different substantive meanings, the calculation of a total indirect effect is not recommended (Selig & Preacher, 2009). For example, while it is possible to calculate the total indirect effect of the $\alpha_1 b_1$ and $\alpha_4 b_4$, where $\alpha_1 = \Delta X_{1-2} \rightarrow \Delta M_{2-3}, \, b_1 = \Delta M_{2-3} \rightarrow \Delta Y_{3-4}$, $\alpha_4 = X_1 \rightarrow M_2$, and $b_4 = M_2 \rightarrow Y_3$, the total indirect effect would be uninterpretable. Specifically, $\alpha_1 b_1$ explains the total indirect effect of the change in $X$ from time 1 to time 2 on the change in $Y$ from time 3 to
time 4, as mediated by the change in $M$ from time 2 to time 3. Here, the indirect effect explains a mediated effect by examining changes between measurements. Whereas, $\alpha_4 b_4$ explains the extent to which the status of $X$ at time 1 influences the status of $Y$ at time 3, as mediated by the status of $M$ at time 2. In this example, the indirect effect explains the mediated effect by examining individual differences on each construct, rather than changes across measurements. Thus, the meaning of a total indirect effect that combines static effects ($X_1 \rightarrow M_2 \rightarrow Y_3$) with changes across measurement occasions ($\Delta X_{1-2} \rightarrow \Delta M_{2-3} \rightarrow \Delta Y_{3-4}$) cannot be interpreted.

**Table 1.1:** All Possible Indirect Effects Within a Full Single Mediator Latent Difference Score Model

<table>
<thead>
<tr>
<th>Type of Indirect Effect</th>
<th>Parameters Responsible</th>
</tr>
</thead>
<tbody>
<tr>
<td>$X_1 \rightarrow M_2 \rightarrow Y_3$</td>
<td>$\alpha_4 \times b_4$</td>
</tr>
<tr>
<td>$X_1 \rightarrow \Delta M_{2,3} \rightarrow \Delta Y_{3,4}$</td>
<td>$\alpha_2 \times b_1$</td>
</tr>
<tr>
<td>$X_1 \rightarrow M_2 \rightarrow \Delta Y_{3,4}$</td>
<td>$\alpha_4 \times b_2$</td>
</tr>
<tr>
<td>$X_1 \rightarrow \Delta M_{2,3} \rightarrow Y_3$</td>
<td>$\alpha_2 \times b_1$</td>
</tr>
<tr>
<td>$\Delta X_{1,2} \rightarrow \Delta M_{2,3} \rightarrow \Delta Y_{3,4}$</td>
<td>$\alpha_1 \times b_1$</td>
</tr>
<tr>
<td>$\Delta X_{1,2} \rightarrow \Delta M_{2,3} \rightarrow Y_3$</td>
<td>$\alpha_1 \times b_3$</td>
</tr>
<tr>
<td>$\Delta X_{1,2} \rightarrow M_2 \rightarrow Y_3$</td>
<td>$\alpha_3 \times b_4$</td>
</tr>
<tr>
<td>$\Delta X_{1,2} \rightarrow M_2 \rightarrow \Delta Y_{3,4}$</td>
<td>$\alpha_3 \times b_2$</td>
</tr>
</tbody>
</table>

Note: $\Delta =$ difference score, $\alpha =$ paths connecting predictors to mediators, $b =$ paths connecting mediators and the outcomes.

**Utility of Power Analysis**

Though the ideal latent difference score mediation structural model includes four measurement occasions, other forms of the model may be used to examine change over
time. Specifically, it is not required that all variables included in the study be represented by a latent change score. For example, a randomized controlled trial with 3 measurement occasions and a categorical $X$ variable (e.g. type of treatment) a research may use a model where $X_d \rightarrow \Delta M_{2-3} \rightarrow \Delta Y_{3-4}$. To date, no known power analyses have been conducted to determine the sample size requirements for the complete latent difference score mediation model ($\Delta X_{1-2} \rightarrow \Delta M_{2-3} \rightarrow \Delta Y_{3-4}$) or its variants. Thus, the current study seeks to conduct a comprehensive Monte Carlo simulation study to detect the required sample size for a wide range of latent difference score mediation structural models, based on several commonly used study designs.

Monte Carlo simulations offer a powerful tool to evaluate the performance of statistical models under various conditions (Muthén & Muthén, 2002). Monte Carlo studies offer an empirical method for evaluating statistical methods and power (Paxton, Curran, Bollen, Kirby & Chen, 2001) and are often used to determine the required sample size to detect effects within a wide range of statistical models. In Monte Carlo studies, data are simulated from a predetermined population model with known population values (Muthén & Muthén, 2002; Paxton et al., 2001). Population values are determined either through a review of the literature that has applied the statistical model of interest, or through covariance algebra calculations based on effect size selections (MacKinnon, 2008; Muthén & Muthén, 2002). A large number of samples are then drawn from the population model, for which all model parameters are estimated. Parameter values and standard errors reflect the average value over the samples drawn from the population model (Muthén & Muthén, 2002). These estimates can then be compared to the predefined population model to evaluate statistical power, bias, and coverage.
In order to determine the required sample size for any given effect, a model must meet four criteria: (1) parameter and standard error biases do not exceed 10% for any parameter in the model; (2) the standard error bias for the parameter being examined, in this case the mediated path, does not exceed 5%; (3) coverage remains between .91 - .98; and (4) statistical power is close to .80 (Muthén & Muthén, 2002). Parameter bias captures how close the average parameter estimate across all replications for any given parameter is to the specified population parameter. Similarly, standard error bias captures how close the average standard error estimate across all replications for any given parameter is to the population standard error. The method to calculate parameter and standard error bias are displayed in Equations 9 and 10, respectively.

\[
\frac{\text{Parameter}_{avg} - \text{Parameter}_{pop}}{\text{Parameter}_{pop}}
\]

\[
\frac{\text{Standard Error}_{avg} - \text{Standard Error}_{pop}}{\text{Standard Error}_{pop}}
\]

Coverage accounts for the proportion of replications for which the 95% confidence interval contains the true parameter value. Finally, statistical power is defined as the probability of accurately rejecting or accepting the null hypothesis (Wilson VanVoorhis & Morgan, 2007; Sedlmaier & Gigerenzer, 1989). A power of .80 has been widely accepted as adequate (Murphy, Myors & Wolach, 2014; Muthén & Muthén, 2002),
meaning that rejecting the null is four times as likely as failure (Murphy et al., 2014).

Though greater power may represent a more acceptable level of risk (e.g. .90 power), it is incredibly difficult to achieve power much greater than .80 (Murphy et al., 2014).

The required sample size to detect any given effect varies greatly by the specific model of interest and expected effect size (Murphy et al., 2014; Muthén & Muthén, 2002). Monte Carlo power analyses identify the required sample size for specific population models, which researchers can then refer to in order to ensure that they collect a large enough sample to detect their hypothesized effects (Thoemmes, MacKinnon & Reiser, 2010; Muthén & Muthén, 2004; Paxton et al., 2001).

**Purpose**

Latent difference score mediation allows researchers to examine change over time, variability in change within and between individuals, and offers flexibility in how change is modeled over time. The lack of empirically supported sample size requirements for latent difference score mediation has resulted in few applications of the model. Monte Carlo studies offer a powerful tool to develop an empirically supported set of sample size guidelines for statistical methods that applied researchers can use to inform their sampling procedures for any study. To address this gap in the literature, the current study includes a Monte Carlo simulation study to develop an empirically supported set of sample size guidelines for ten possible latent difference score mediation structural models.
with nine unique population models. The structural and population models were selected to offer a generalizable set of sample size requirements.

**Statistical Analysis**

A Monte Carlo simulation was conducted to examine the required sample size to detect mediation among several variations of the latent difference score mediation model using Mplus statistical software (Muthén & Muthén, 1998-2011). Ten structural latent difference score mediation models and nine population models were included in the study. Each structural model represents a possible single mediator model, in which \( X, M, \) and \( Y \) may be either a latent change score or latent variable. Models with a dichotomous \( X \) were also tested to offer sample estimates for those interested in conducting randomized controlled trials or experimental studies. The population models were selected to offer a generalizable set of guidelines among a wide range of effect size pairings. Thus, all possible effect size pairings for the indirect, or \( ab \), paths were tested. Structural and population models tested in the Monte Carlo study are displayed in Table 1.2. In Table 1.2, small, medium, and large effect size estimates are labeled S, M, and L, respectively. For example, models with a small \( a \) path and large \( b \) path are labeled SL.
Table 1.2: All Structural and Parameter Models Tested in the Monte Carlo Study

<table>
<thead>
<tr>
<th>Structural Models</th>
<th>Population model effect size pairings</th>
</tr>
</thead>
<tbody>
<tr>
<td>$X_{d1} \rightarrow \Delta M_{1,2} \rightarrow \Delta Y_{2,3}$</td>
<td>SS, SM, SL, MS, MM, ML, LM, LL</td>
</tr>
<tr>
<td>$X_{d1} \rightarrow \Delta M_{1,2} \rightarrow Y_3$</td>
<td>SS, SM, SL, MS, MM, ML, LM, LL</td>
</tr>
<tr>
<td>$X_{d1} \rightarrow \Delta M_{1,2} \rightarrow \Delta Y_3$</td>
<td>SS, SM, SL, MS, MM, ML, LM, LL</td>
</tr>
<tr>
<td>$X_1 \rightarrow \Delta M_{1,2} \rightarrow \Delta Y_{2,3}$</td>
<td>SS, SM, SL, MS, MM, ML, LM, LL</td>
</tr>
<tr>
<td>$X_1 \rightarrow \Delta M_{1,2} \rightarrow Y_3$</td>
<td>SS, SM, SL, MS, MM, ML, LM, LL</td>
</tr>
<tr>
<td>$X_1 \rightarrow M_2 \rightarrow \Delta Y_{2,3}$</td>
<td>SS, SM, SL, MS, MM, ML, LM, LL</td>
</tr>
<tr>
<td>$\Delta X_{1,2} \rightarrow \Delta M_{2,3} \rightarrow \Delta Y_{3,4}$</td>
<td>SS, SM, SL, MS, MM, ML, LM, LL</td>
</tr>
<tr>
<td>$\Delta X_{1,2} \rightarrow \Delta M_{2,3} \rightarrow Y_3$</td>
<td>SS, SM, SL, MS, MM, ML, LM, LL</td>
</tr>
<tr>
<td>$\Delta X_{1,2} \rightarrow M_2 \rightarrow \Delta Y_{2,3}$</td>
<td>SS, SM, SL, MS, MM, ML, LM, LL</td>
</tr>
<tr>
<td>$\Delta X_{1,2} \rightarrow M_2 \rightarrow Y_3$</td>
<td>SS, SM, SL, MS, MM, ML, LM, LL</td>
</tr>
</tbody>
</table>

Parameter and Model Specifications

To offer a wide range of generalizable empirical sample estimates, (Paxton et al., 2001), each of the ten structural models were tested with the six possible effect size combinations for the paths representing the relationship between $X \rightarrow M$ ($a$) and $M \rightarrow Y$ ($b$). Although the latent difference score mediation model has been previously applied (Bryan et al., 2016; Reeve, Paul & Butterworth, 2015; Infurna & Mayer, 2015; Bryan et al., 2015), no known study to date has presented model fit information and therefore, the population parameter values could not be estimated from existing literature.

Corresponding unstandardized path coefficients were calculated using covariance algebra from a set of predefined effect size estimates. Complete details regarding the covariance algebra needed to calculate these values has been outlined in MacKinnon (2008).

Effect size estimates used to develop the population model parameters followed Cohen’s (1988) guidelines for $R^2$ estimates, which describes the amount of explained
variance in the outcome. Cohen (1988) defined $R^2$ effect sizes as small (0.14), medium (0.39), and large (0.59) effects. The $R^2$ effect size statistic was selected to provide empirical sample estimates that compare to those from other Monte Carlo studies examining power among complex mediation models (e.g., Thoemmes, MacKinnon, & Reiser, 2010; Ma & Zeng, 2014; Fritz & MacKinnon, 2007). All unstandardized path values are provided in Table 1.3. Unstandardized path coefficients of .14, .36, and .51 were used to represent small, medium, and large effect sizes for the $a$ and $b$ paths when $X$ was a latent or latent change score variable. Whereas, unstandardized path coefficients of .28, .72, and 1.02 were used to represent small, medium, and large effect sizes for the $a$ path when $X$ was dichotomous. The $c'$ pathway, or the path representing the direct effect of $X$ on $Y$ while controlling for $M$, was fixed to a small effect size, resulting in an path coefficient of .14 when $X$ was a latent or latent change score, and .28 when dichotomous.

**Table 1.3:** Unstandardized Parameter Values for Each Structural Model Representing Small, Medium, and Large $R^2$ Values

<table>
<thead>
<tr>
<th>Structural Models</th>
<th>Unstandardized paths</th>
<th>$\alpha$</th>
<th>$b$</th>
</tr>
</thead>
<tbody>
<tr>
<td>$X_{d1} \rightarrow \Delta M_{1,2} \rightarrow \Delta Y_{2,3}$</td>
<td>0.28, 0.72, 1.02</td>
<td>0.14, 0.36 and 0.51</td>
<td></td>
</tr>
<tr>
<td>$X_{d1} \rightarrow \Delta M_{1,2} \rightarrow Y_3$</td>
<td>0.28, 0.72, 1.02</td>
<td>0.14, 0.36 and 0.51</td>
<td></td>
</tr>
<tr>
<td>$X_{d1} \rightarrow \Delta M_{1,2} \rightarrow \Delta Y_3$</td>
<td>0.28, 0.71, 1.02</td>
<td>0.14, 0.36 and 0.51</td>
<td></td>
</tr>
<tr>
<td>$X_1 \rightarrow \Delta M_{1,2} \rightarrow \Delta Y_{2,3}$</td>
<td>0.14, 0.36, 0.51</td>
<td>0.14, 0.36 and 0.51</td>
<td></td>
</tr>
<tr>
<td>$X_1 \rightarrow \Delta M_{1,2} \rightarrow Y_3$</td>
<td>0.14, 0.36, 0.51</td>
<td>0.14, 0.36 and 0.51</td>
<td></td>
</tr>
<tr>
<td>$X_1 \rightarrow M_2 \rightarrow \Delta Y_{2,3}$</td>
<td>0.14, 0.36, 0.51</td>
<td>0.14, 0.36 and 0.51</td>
<td></td>
</tr>
<tr>
<td>$\Delta X_{1,2} \rightarrow \Delta M_{2,3} \rightarrow \Delta Y_{3,4}$</td>
<td>0.14, 0.36, 0.51</td>
<td>0.14, 0.36 and 0.51</td>
<td></td>
</tr>
<tr>
<td>$\Delta X_{1,2} \rightarrow \Delta M_{2,3} \rightarrow Y_3$</td>
<td>0.14, 0.36, 0.51</td>
<td>0.14, 0.36 and 0.51</td>
<td></td>
</tr>
<tr>
<td>$\Delta X_{1,2} \rightarrow M_2 \rightarrow \Delta Y_{2,3}$</td>
<td>0.14, 0.36, 0.51</td>
<td>0.14, 0.36 and 0.51</td>
<td></td>
</tr>
<tr>
<td>$\Delta X_{1,2} \rightarrow M_2 \rightarrow Y_3$</td>
<td>0.14, 0.36, 0.51</td>
<td>0.14, 0.36 and 0.51</td>
<td></td>
</tr>
</tbody>
</table>
For simplicity purposes, and consistent with other studies examining the required sample size to detect mediation among complex mediation models (e.g., Thoemmes et al., 2010), all continuous variables were generated to have a mean of 0 and a variance of 1. Distributions were assumed to be normal, and the data were generated with no missing data. Factor loadings were randomized between 0.90 – 1.10 to ensure that the tested models attained adequate measurement invariance, without requiring strict measurement invariance (equal loadings set to 1.00). Residual variances were fixed so that the total variance was 1.0. The covariance algebra that was used to determine the unstandardized path estimates and residual variances is presented in Appendix A.

Several preliminary analyses were conducted to determine the most appropriate beta value for a latent $X$, $M$, and $Y$ variable onto its respective change score variable. These preliminary analyses revealed an average unstandardized path value of -.30, and thus when applicable, the $X$, $M$, and $Y$ loadings onto their respective change score variables were fixed to -.30. Consistent with previous Monte Carlo simulation studies, the residual variances for the indicators for any $M$ variable were fixed to the value of the $b$ path, and the residual variances for the indicators of any $X$ and $Y$ variables were fixed to the residual variables of their respective latent factors.

Given the complex nature of the indirect effects tested within the full latent difference score mediation model, only the indirect path of interest for each model was tested. For example, in a $\Delta X_{1-2} \rightarrow \Delta M_{2-3} \rightarrow \Delta Y_{3-4}$ only the $\Delta X_{1-2} \rightarrow \Delta M_{2-3} \rightarrow \Delta Y_{3-4}$ indirect path were tested. See Figure 1.5 for an example.
Each model was tested with 1,000 Monte Carlo replications with a random seed value of 11787 across models. Bias-corrected bootstrap tests were used to adjust for the asymmetrical distribution inherent in mediation analysis, as this method provides the most accurate confidence intervals of current resampling techniques (MacKinnon et al., 2004). Each model included 1,000 bootstrap draws. A sample Mplus Monte Carlo input file has been provided in Appendix B.

**Model Selection**

The primary goal of this study was to generate a set of guidelines for the required sample size to detect mediation among several latent difference score mediation model variants. The four criteria for sample size selection outlined by Muthén and Muthén (2002) were followed to select the final sample sizes for each model: (1) parameter and
standard error biases do not exceed 10% for any parameter in the model; (2) the standard error bias for the parameter being examined, in this case the mediated path, does not exceed 5%; (3) coverage remains between .91 - .98; and (4) statistical power is close to .80. Thus, the final sample size estimates have met all four of these criteria.

Sample sizes were adjusted in an iterative process, accordingly, until the empirical power estimate is within 0.001 of 0.80, coverage ranged between .91 - .98, and both parameter and standard error bias were at an acceptable level. For example, if power was greater than 0.80, the sample size was decreased, and if the power estimate was smaller than 0.80, the sample size was increased (Fritz & MacKinnon, 2007). This iterative process was conducted until all criteria were met for each of the tested models.

Results

The final empirical sample estimates for each structural and population model are shown in Table 1.4. Empirical sample estimates ranged from 120 – 1015, with sample requirements varying greatly across each population model and model structure. In general, population models with larger effect sizes tended to require smaller sample sizes than those with smaller effect size pairings. The smallest sample sizes requirements were achieved among population models with MM and LL effect size pairings. Moreover, the magnitude of effect for the $b$ path tended to influence the sample size estimates more strongly than the effect size of the $a$ path. Specifically, population models in which the $b$
path was specified as small required larger samples than any other models. Sample size requirements were highest among the population model with a LS effect size pairing ranging from 775 – 1015.

Model structures also had a large impact on the empirical sample size estimates. To this end, model structures in which \( X \) was included as a dichotomous variable tended to require smaller samples than models in which \( X \) was modeled as a continuous or latent change score variable. Similarly, model structures in which \( X \) was modeled as a continuous \( X \) variable required fewer participants than models in which \( X \) was modeled as a latent change score.

Table 1.4: Empirical Sample Estimates for Each Model After Adjusting for Standard Error and Parameter Biases

<table>
<thead>
<tr>
<th>Models</th>
<th>Effect Size Combinations</th>
<th>SS</th>
<th>SM</th>
<th>SL</th>
<th>MS</th>
<th>MM</th>
<th>ML</th>
<th>LS</th>
<th>LM</th>
<th>LL</th>
</tr>
</thead>
<tbody>
<tr>
<td>( X_d \rightarrow \Delta M_{1,2} \rightarrow \Delta Y_{2,3} )</td>
<td></td>
<td>550</td>
<td>435</td>
<td>483</td>
<td>625</td>
<td>150</td>
<td>155</td>
<td>775</td>
<td>145</td>
<td>150</td>
</tr>
<tr>
<td>( X_d \rightarrow \Delta M_{1,2} \rightarrow Y_3 )</td>
<td></td>
<td>515</td>
<td>380</td>
<td>430</td>
<td>675</td>
<td>120</td>
<td>165</td>
<td>800</td>
<td>148</td>
<td>170</td>
</tr>
<tr>
<td>( X_d \rightarrow \Delta M_{1,2} \rightarrow \Delta Y_3 )</td>
<td></td>
<td>597</td>
<td>413</td>
<td>435</td>
<td>650</td>
<td>120</td>
<td>150</td>
<td>787</td>
<td>140</td>
<td>155</td>
</tr>
<tr>
<td>( X_1 \rightarrow \Delta M_{1,2} \rightarrow \Delta Y_{2,3} )</td>
<td></td>
<td>647</td>
<td>600</td>
<td>638</td>
<td>680</td>
<td>270</td>
<td>230</td>
<td>862</td>
<td>260</td>
<td>250</td>
</tr>
<tr>
<td>( X_1 \rightarrow \Delta M_{1,2} \rightarrow Y_3 )</td>
<td></td>
<td>595</td>
<td>565</td>
<td>632</td>
<td>636</td>
<td>250</td>
<td>210</td>
<td>880</td>
<td>250</td>
<td>250</td>
</tr>
<tr>
<td>( X_1 \rightarrow M_2 \rightarrow \Delta Y_{2,3} )</td>
<td></td>
<td>640</td>
<td>580</td>
<td>648</td>
<td>640</td>
<td>240</td>
<td>200</td>
<td>845</td>
<td>180</td>
<td>200</td>
</tr>
<tr>
<td>( \Delta X_{1,2} \rightarrow \Delta M_{2,3} \rightarrow \Delta Y_{3,4} )</td>
<td></td>
<td>670</td>
<td>645</td>
<td>715</td>
<td>750</td>
<td>275</td>
<td>280</td>
<td>1000</td>
<td>470</td>
<td>350</td>
</tr>
<tr>
<td>( \Delta X_{1,2} \rightarrow \Delta M_{2,3} \rightarrow Y_3 )</td>
<td></td>
<td>670</td>
<td>635</td>
<td>715</td>
<td>750</td>
<td>250</td>
<td>260</td>
<td>1050</td>
<td>400</td>
<td>390</td>
</tr>
<tr>
<td>( \Delta X_{1,2} \rightarrow M_2 \rightarrow \Delta Y_{2,3} )</td>
<td></td>
<td>715</td>
<td>656</td>
<td>710</td>
<td>753</td>
<td>265</td>
<td>250</td>
<td>1020</td>
<td>320</td>
<td>250</td>
</tr>
<tr>
<td>( \Delta X_{1,2} \rightarrow M_2 \rightarrow Y_3 )</td>
<td></td>
<td>690</td>
<td>650</td>
<td>704</td>
<td>730</td>
<td>250</td>
<td>275</td>
<td>1015</td>
<td>290</td>
<td>248</td>
</tr>
</tbody>
</table>

Note. \( X = \) predictor, \( d = \) dichotomous variable, \( M = \) mediator, \( Y = \) outcome. \( S = \) small effect, \( M = \) medium effect, \( L = \) large effect
After .80 power was achieved, parameter and standard error biases for population models MM, ML, LM, and LL exceeded 10%, with bias linearly increasing with the size of the effect pairings. To adjust for inflated bias, iterations with larger sample sizes were tested until the smallest sample size necessary to achieve minimal bias was achieved. The unadjusted sample size for each structural and population models are provided in Table 1.5 to display the impact of bias on the required sample size for each of these models.

**Table 1.5: Sample Size Estimates Prior to Adjusting for Standard Error and Parameter Biases**

<table>
<thead>
<tr>
<th>Models</th>
<th>Effect Size Combinations</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>SS</td>
</tr>
<tr>
<td>$X_{d1} \rightarrow \Delta M_{1,2} \rightarrow \Delta Y_{2,3}$</td>
<td>550</td>
</tr>
<tr>
<td>$X_{d1} \rightarrow \Delta M_{1,2} \rightarrow \Delta Y_{3}$</td>
<td>515</td>
</tr>
<tr>
<td>$X_{d1} \rightarrow \Delta M_{1,2} \rightarrow \Delta Y_{3}$</td>
<td>597</td>
</tr>
<tr>
<td>$X_1 \rightarrow \Delta M_{1,2} \rightarrow \Delta Y_{2,3}$</td>
<td>647</td>
</tr>
<tr>
<td>$X_1 \rightarrow \Delta M_{1,2} \rightarrow \Delta Y_{3}$</td>
<td>595</td>
</tr>
<tr>
<td>$X_1 \rightarrow M_2 \rightarrow \Delta Y_{2,3}$</td>
<td>640</td>
</tr>
<tr>
<td>$\Delta X_{1,2} \rightarrow \Delta M_{2,3} \rightarrow \Delta Y_{3,4}$</td>
<td>670</td>
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<tr>
<td>$\Delta X_{1,2} \rightarrow \Delta M_{2,3} \rightarrow \Delta Y_{3}$</td>
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<tr>
<td>$\Delta X_{1,2} \rightarrow M_2 \rightarrow \Delta Y_{2,3}$</td>
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<tr>
<td>$\Delta X_{1,2} \rightarrow M_2 \rightarrow Y_3$</td>
<td>690</td>
</tr>
</tbody>
</table>

*Note.* $X =$ predictor variable, $d =$ dichotomous variable, $M =$ mediator variable, $Y =$ outcome variable. $S =$ small effect, $M =$ medium effect, $L =$ large effect
The purpose of this paper was to develop a set of empirically supported sample size guidelines for ten unique latent difference score mediation models and nine population models. There are four key findings from the current Monte Carlo analysis: (1) population models with larger effect size pairings generally require smaller sample sizes than those with smaller effect size pairings; (2) the effect size of the $b$ path has a greater impact on the required sample size than that of the $a$ path; (3) population models with larger effect size pairings show inflated parameter and standard error biases, resulting in the need for bias-adjusted sample size requirements; and (4) simple structural models require fewer participants than more complex structural models. Each of the main findings are discussed in more detail below.

The magnitude of effect for both the $a$ and $b$ paths strongly influenced the empirical sample estimates across population models. More specifically, population models with larger effect size pairings generally required smaller samples than population models with smaller effect size pairings. This finding is consistent with findings from other Monte Carlo simulations with mediation analysis (e.g., Fritz & Mackinnon, 2007).

Moreover, the magnitude of effect of the $b$ path has a greater impact on the required sample size than that of the $a$ path. Models with a small effect size for the $b$ path required larger sample estimates than models with small, medium, or large effect sizes the $a$ path, and models with medium or large effect sizes for the $b$ path. This finding
suggests that the $b$ path is more important in determining the required sample size to
detect mediation than the $a$ path. While these findings are consistent with other
mediational power analyses (e.g., Fritz & Mackinnon, 2007), the discrepancies in the
sample size requirements among models with a small $b$ path effect size were much larger
than those seen in previous power analyses. For example, the results of the current study
identified a required sample size of 862 with a LS effect size pairing and 638 with a SL
effect size pairing in the $X_{1-2} \rightarrow \Delta M_{1-2} \rightarrow \Delta Y_{2-3}$ structural model. In this case, the
population model with a LS effect size pairing requires a sample size with more than 200
more participants than the SL effect size pairing. Findings from a previous Monte Carlo
simulation study on mediation report a required sample size of 396 for a LS effect size
pairing and 385 for a SL effect size pairing (Fritz & MacKinnon, 2007), which is a much
smaller discrepancy than those seen in the current study.

The results from the current Monte Carlo simulation found that population models
MM, ML, LM, and LL effect size pairings with adequate statistical power and coverage
violated the parameter and standard error bias guidelines set forth by Muthén and Muthén
(2002). As a result, the final sample size requirements reflect sample sizes with greater
than .80 power. Moreover, parameter and standard error biases increased linearly with the
effect size pairings, with the exception of the LS effect size pairing that did not encounter
any issues with parameter or standard error bias. This finding indicates that latent
difference score mediation models require no less than 120 participants, as a result of
parameter and standard error bias. Overall, the general trends in the required sample size
to detect mediation compare to those seen in previous mediational power analyses (e.g.,
Fritz & MacKinnon, 2007), however latent difference score mediation models were found to require more participants than mediational path analysis.

Model structure also played a large role in determining the size of the empirical sample estimates. To this end, less complicated models, such as those with a dichotomous $X$, required fewer participants across population models than those with multiple change score variables. This finding highlights the benefits of simplicity, and suggests that researchers should only include change scores for the variables in which modeling change across adjacent measurement occasions is necessary to answer the question of interest. For example, consider a researcher who is interested in whether disordered eating among adolescents predicts changes in family environment, which ultimately predicts alcohol misuse among adult women. In this example, family environment would be modeled as a latent change score, but disordered eating and alcohol misuse can be modeled as latent variables.

On the basis of the results from the empirical sample estimates, there are four key recommendations for researchers interested in using latent difference score mediation models: (1) researchers must thoroughly evaluate the literature to determine the expected effect sizes before choosing their sample size; (2) sample estimates should be viewed as a lower bound; (3) simplicity is considered a virtue and thus, change scores should be included as necessary within any given model; and (4) the number of mediators and kinds of indirect effects to be tested must be determined before selecting a sample size. Each of the key recommendations are discussed in detail below.

First, given the impact of effect size pairings on the required sample size to detect mediation, it is suggested that researchers thoroughly review the literature in their
research area of interest to determine their hypothesized effect size. Because the $b$ pathway more strongly influences the required sample size to detect mediation, researchers should seek out the results of other mediational models in their substantive area to inform their decision on the effect size estimates for the $b$ pathway.

Second, because the simulated data included in this study were normally distributed and contained no missing data, researchers should view the empirical sample estimates from the current study as a lower limit of the number of participants needed to achieve .8 power. Consider a researcher who is interested in testing whether changes in self-esteem mediate the relationship between changes in disordered eating and changes in alcohol abuse. This researcher hypothesizes that the effect of changes in disordered eating on self-esteem will be of medium size and the effect of changes in self-esteem on changes in alcohol abuse to be of small size. Using the results from the current power analysis, the researcher can identify a lower sample size bound of 750 for their study to achieve .8 power.

Third, because simplicity is considered a virtue of latent difference score mediation models, researchers should strongly consider which variables will be modeled as latent change scores. This consideration is important because sample size requirements drastically increase with the addition of each change score variable. Moreover, if change across measurement occasions is not a key component of the research question at hand, modeling a variable as such may result in no change overtime and thus the findings will be rendered insignificant.

Finally, researchers must consider how many mediators they would like to include and which specific indirect paths they hope to test prior to selecting a sample
size. The current study examined the required sample size to detect mediation among ten structural latent difference score mediation models and nine population models, all with a single mediator and one indirect effect. Researchers looking to include more than one mediator or test more than one indirect path will likely require more participants to achieve adequate power than those identified in the empirical sample size estimates within the current study.

Power estimates that offer empirical estimates for complex statistical models are becoming a common requirement when applying for grants and other funding mechanisms (Fritz & MacKinnon, 2007). The current study aimed to offer a generalizable set of sample size guidelines for researchers interested in applying latent difference score mediation models. There is no general rule of thumb for determining the required sample size for any given model, thus the sample size needed for a study depends on many factors (e.g., effect size; Muthén & Muthén, 2002). Monte Carlo simulations are common empirical method to evaluate structural equation models (Paxton et al., 2001), thus Monte Carlo models were used to general a set of empirical sample estimates across ten model structures and nine population models. As a result, the current study offers future researchers a reference for determining sample size requirements for a wide range of single-mediator latent difference score mediational models.
REFERENCES


Fritz, M.S., & MacKinnon, D.P. (2007). Required sample size to detect the mediated


Carlo simulation. *American Journal of Applied Psychology, 3*(3), 72-79. DOI: 10.11648/j.ajap.20140303.15


ABSTRACT

Study 2: Ecodevelopmental Processes of Disordered Eating and Alcohol Misuse Among Girls and Women

Background. Alcohol misuse has extensive consequences for societal functioning, physical health and well-being. Overall, women experience more severe consequences as a result of alcohol misuse, yet little is known about gender-specific predictors of alcohol misuse among women. To this end, disordered eating behaviors have been shown to reliably predict onset of alcohol misuse among women, yet little is known about how disordered eating behaviors exert their efforts on later alcohol misuse. Because ecological theories of human development suggest that adolescents share a reciprocal relationship with their environment, in which behavior changes social environment, and social environment changes behavior, environmental and internal processes were hypothesized as potential mechanisms through which alcohol misuse emerges. Objective. The purpose of the current study was to evaluate the extent to which social environmental and internal processes mediate the relationship between disordered eating behaviors among adolescent girls and alcohol misuse among adult women. Method. Data from 10,480 girls and women from waves I-III within the National Study of Adolescent to Adult Health dataset were examined through the application of latent difference score mediation. The analyses tested the extent to which family environment, parent-child relationships, and self-esteem mediated the relationship between adolescent disordered eating and alcohol misuse among adult women. Results. The results of the study revealed that none of the
hypothesized environmental or internal processes of interest mediate the relationship between disordered eating behaviors and alcohol misuse. *Conclusions.* The current study did not identify significant evidence that changes in perceived family environment, parent-child relationships, and self-esteem as a result of adolescent disordered eating predict subsequent changes in alcohol misuse among girls and women. It is possible that the adolescent girls who use substances to control weight are more likely to experience later alcohol misuse, yet the current study did not have enough power to examine the impact of substance-based disordered eating on alcohol misuse, thus highlighting the importance of disordered eating subtypes when examining their impacts on later alcohol misuse and their associated underlying environmental and internal processes.
CHAPTER III

STUDY 2: ECODEVELOPMENTAL PROCESSES OF DISORDERED EATING AND ALCOHOL MISUSE AMONG GIRLS AND WOMEN

Introduction

Alcohol misuse is a serious issue that greatly diminishes individuals’ personal and physical well-being and has extensive consequences for societal functioning. Though men have higher rates of drinking related problems than women (Substance Abuse and Mental Health Services Administration, 2014), health-related consequences of alcohol misuse are stronger for women than men (Nolan-Hoeksema, 2004), as women experience more severe medical and psychiatric consequences with lower volumes of drinking. Additionally, fetal alcohol syndrome and fetal alcohol effects are transmitted by women’s drinking behaviors during pregnancy, resulting in intergenerational effects on health and well-being (Brady & Randall, 1999; Rehm et al., 2003). As a result of these substantial costs, researchers are justifiably interested in uncovering the etiological course of alcohol misuse among women through longitudinal studies.

Over the past two decades, a large body of work has accumulated in the interest of explaining differences in the prevalence of alcohol misuse between women and men (Schulte et al., 2009). Of special interest to developmental researchers is the discontinuity of gender differences in alcohol problems from adolescence to adulthood: girls and boys drink at approximately the same rate in adolescence but diverge substantially as they
transition into adulthood, with men showing elevated levels of alcohol use from age 18 onward (Young et al., 2002). The presence of psychiatric vulnerabilities in adolescence has emerged as a possible mechanism through which alcohol misuse develops over time (Schulte et al., 2009), and there is evidence that boys’ and girls’ drinking behaviors are affected differently across different developmental stages. However, the specific long-term nature of these developmentally linked changes is currently unclear. Moreover, the field is in the early stages of undertaking long-term investigations of the role of other psychiatric problems in the progression of alcohol misuse that may yield important information for tailoring alcohol prevention programs for girls and women (e.g., Micali et al, 2015).

One notable challenge in understanding the complexities underlying the development of alcohol misuse concerns the substantial gender differences in alcohol risk factors (Pisetsky, Chao & Dierker, 2008; Walitzer & Dearing, 2006), a topic in which studies are still in high demand. Gender specific predictors of alcohol misuse may be identified by evaluating the predictive effects of adolescent psychiatric vulnerabilities that are more common among either girls or boys. To this end, the role of disordered eating behaviors among adolescent girls on later alcohol misuse has recently gained interest (Harrop & Marlatt, 2010). Disordered eating behaviors disproportionately impact girls (Eichen, Conner, Daly & Fauber, 2012) throughout adolescence and adulthood (Vartanian & Porter, 2016; Neumark-Sztainer et al., 2011) and are often found to co-occur with alcohol misuse (Ortega-Luyando et al., 2015; Micali et al., 2015; Harrop & Marlatt, 2010; Gadalla & Piran, 2007). Moreover, women with co-occurring disordered eating and alcohol misuse often report more psychotic symptoms (Gilchrist, Smith &
Dawson, 2007), more alcohol-related health consequences (Dunn, Larimer, & Neighbors, 2002), and higher mortality rates than women with only disordered eating or alcohol misuse (Franko, Dorer, Keel, Jackson, Manzo & Herzog, 2005). Because disordered eating behaviors result in long-term negative psychiatric consequences (Micali et al., 2015) and alcohol misuse is associated with the highest mortality rates of all psychiatric disorders (Whiteford et al., 2013), researchers are justifiably interested in uncovering the etiological course of the co-occurring disordered eating and alcohol misuse (Harrop & Marlatt, 2010).

The results of a systematic review of the literature conducted to examine the etiology of co-occurring alcohol misuse and disordered eating revealed that disordered eating behaviors reliably predict future alcohol misuse, where as alcohol misuse does not reliably predict the emergence of disordered eating (Harrop & Marlatt, 2010). Moreover, developmental research suggests that disordered eating tends to emerge in middle childhood to early adolescence (Kurz et al., 2016; Rohde, Stice, & Marti, 2015), whereas alcohol misuse tends to emerge somewhat later in adolescence (Chassin, Flora, & King, 2004; Foster, Hicks, Iacono, & McGue, 2015). This unidirectional relation has been further supported by additional empirical evidence (Lange & Fields, 2015; Gregorowski, Seedat & Jordan, 2013; Franko et al., 2005). Thus, there is substantial evidence that disordered eating behaviors among adolescent girls predicts alcohol misuse. However, the underlying processes through which adolescent disordered eating behavior exerts its efforts on alcohol misuse remain unexplained.

In sum, alcohol misuse has extensive consequences for societal functioning and physical health and well-being, with women experiencing more severe consequences.
Disordered eating among adolescent girls has been identified as a possible gender specific predictor of alcohol misuse among adult women. Because disordered eating behaviors impact girls at a younger age than alcohol misuse and reliably predicts subsequent alcohol misuse (Harrop & Marlatt, 2010; Hudson, Hiripi, Pope, & Kessler, 2007), the developmental role of disordered eating on the etiology of alcohol misuse and the underlying time-dependent processes that operate between the two behaviors must be examined holistically. Specifically, guided by the ecological theory of human development (Bronfenbrenner, 1977), the goal of the current study was to determine the processes through adolescent disordered eating behaviors influence social environmental and internal processes which in turn influence alcohol misuse.

**Disordered Eating as an Influence on Development into Adulthood**

Though substantial empirical evidence supports the direct influence of adolescent disordered eating behaviors on alcohol misuse among adult women (Lange & Fields, 2015; Measelle et al., 2006; Franko et al., 2005), evidence explaining the underlying developmental processes through which disordered eating exerts its effects on alcohol misuse remain unclear. To explore potential processes through which these behaviors occur, an ecological perspective was examined. Specifically, Bronfenbrenner’s (1977) ecological theory of human development suggests that adolescents are not passive recipients of their environment, yet the person-environment relationship is reciprocal. Put simply, adolescent behaviors influence their social environment, which in turn influences
later behavior. As active participants in their environment, when adolescent girls change their behaviors (e.g. initiate disordered eating) the behavioral change impacts their social environment (e.g. parental relations; Spanos et al., 2010) and internal processes (e.g. self-esteem; Stephen et al., 2014; Lo, Gullo & Salerno, 2011). These environmental and internal changes significantly contribute to later ecological transitions (e.g. the transition to adulthood; Bronfenbrenner, 1977) and shape the course of girls’ development and influence later health behaviors. Thus, an examination of social ecological pathways underlying adolescent disordered eating to alcohol misuse in adulthood can inform prevention scientists of when and how to intervene upon girls at risk and identify specific environmental and internal factors related to unhealthy transitions in to adulthood.

Disordered Eating, Alcohol Misuse and their Shared Processes

Given the reciprocal relationship between adolescents’ environments and their behaviors, changes in the social environment and internal processes often influence developmental transitions and distal health outcomes (Bronfenbrenner, 1977). Thus social environment and internal processes are often viewed as the processes underlying early predictors and distal outcomes, and ultimately serve as malleable targets for prevention effort. To this end, in order to identify potential processes underlying the relationship between disordered eating and alcohol misuse, shared social environmental and internal processes were examined.
Developmental theorists have come to widely accept that family processes operate as varying reciprocal processes over time, in which the behaviors of family members regularly influence family dynamics (Maccoby, 2003). Thus, the influence of family environment in the relation between disordered eating and alcohol misuse was examined. To this end, while family environment has traditionally been viewed as a risk factor for disordered eating behavior (e.g., Jacobi, Hayward, de Zwaan, Karemer, & Agras, 2004), a recent study (Spanos et al., 2010) has identified family conflict as an environmental product of disordered eating behavior, rather than a risk factor. While these findings stand in contrast to previous works (e.g., Builk, Wade, & Kendler, 2001), older studies have relied on cross-sectional data and thus have been unable to evaluate directional effects. The study conducted by Spanos et al. (2010) was the first of its kind to evaluate prospective effects, and thus the current study examines family environment and parent-child relationships as a product of disordered eating among adolescent girls. These findings have since been replicated (Wallace, 2015), which further supports the notion that family conflict is an environmental product of disordered eating behavior, rather than a risk factor.

The current study aims to build upon previous work by examining the behavioral repercussions of family environments impacted by disordered eating among adolescents. Specifically, family environment and parent-child relationships have also been found to be predictive of future alcohol misuse (Tarantino et al., 2015; Umberson & Montez, 2010; Repetti, Taylor & Seeman, 2002; Griffin et al., 2000). Thus, the current study aims to evaluate the role of family environment and parent-child relationships in the relationship between disordered eating behaviors and alcohol misuse among women. To
this end, it was hypothesized that disordered eating among adolescent girls predicts changes in family environments and parent-child relationships, which in turn predict alcohol misuse among women.

Internal processes have also been shown to influence adolescent development and distal health outcomes. Among these processes, self-esteem is of particular interest. Specifically, adolescent girls who struggle with disordered eating behaviors are more likely to experience low self-esteem (Rodgers, Paxton & McLean, 2014; Santos, Richards & Bleckley, 2007). Similarly, girls with lower self-esteem are likely to develop alcohol misuse issues later in life (Trzesniewski et al., 2006; Flory et al., 2004; Wild et al., 2004). To this end, poor self-esteem as a result of adolescent disordered eating behaviors may be predictive of alcohol misuse among adult women.

In sum, guided by the ecological theory of human development (Bronfenbrenner, 1977), it was predicted that the reciprocal nature of adolescent behavior and their social environment, as well as internal processes, would explain the underlying processes between disordered eating and alcohol misuse. To this end, changes in perceived family environment, parent-child relationships, and self-esteem were identified as potential environmental and internal processes that underlie the relationship between disordered eating among adolescent girls and alcohol misuse among women. Thus, the goal of the current study was to determine the extent to which changes in perceived family environment, parent-child relationships, and self-esteem mediate the relationship between disordered eating among adolescent girls and alcohol misuse among adult women.
The Current Study

Co-occurring disordered eating and alcohol misuse has a broad range of medical and psychiatric consequences for girls and women. While previous work has reliably found disordered eating to predict the onset of alcohol misuse (see Harrop & Marlatt, 2010 for a review), the processes through which disordered eating exerts its efforts onto alcohol misuse are not yet understood. Guided by the ecological theory of human development (Bronfenbrenner, 1977), the goal of the current study was to examine the extent to which social environmental and internal processes mediate the relationship between disordered eating among adolescent girls and alcohol misuse among adult women. Because of the known relationships among disordered eating behaviors perceived family environment, parent-child relationships, self-esteem, and alcohol misuse, these common environmental and internal factors were examined as the mediators through which adolescent disordered eating exerts its impacts on alcohol misuse among women transitioning to adulthood. The current study used extant data from the National Longitudinal Study of Adolescent to Adult Health (Add Health; Harris, 2012) to identify time-dependent processes through which disordered eating behaviors and alcohol misuse operate as girls transition from adolescence to adulthood.
Methods

Data Source

The current study involves data from three measurement periods from the National Longitudinal Study of Adolescent to Adult Health (Add Health; Harris, 2012). The sample consists of all participants who self-identified as girls/women at any wave (N=10,480). Briefly, Add Health is a comprehensive, nationally representative study of adolescent health and risk behaviors, personal traits, families, relationships, social groups, neighborhoods and communities (Harris, 2012). The data are stratified by census region, clustered by school identification, and weighted to control for selection-based oversampling procedures of specific populations of interest. Adolescent data were collected from in-home visits, beginning when the youth were ages 10-18, with a follow-up visit 12 months later. Emerging adulthood data (ages 18-26) were collected at year 7. Thus, the longitudinal analyses consist of the entire span of adolescence (ages 10-17) and emerging adulthood (ages 18-26). The complete study methods for all waves of Add Health have previously been described in detail (Harris, 2012). All variables included in the current study were collected during in-home interviews, unless otherwise noted, and are provided in Appendices C-F.
Measures

Demographics. All analyses account for race, ethnicity, age, family socioeconomic status (e.g., parental education or public assistance), and alcohol history of parental figures. All demographics were collected at wave I.

Disordered Eating Behaviors. Disordered eating behaviors reports from wave I were included in the study. Participants responded “yes” or “no” to the use of 1) dietary restriction, 2) diet pills, 3) self-induced vomiting, and 4) laxative use to control weight. Due to the limited number of participants endorsing many of the items in the scale, the responses were computed into a single sum variable that ranged from 0 – 4.

Alcohol Misuse. Alcohol misuse data from waves II and III were included in the analyses. To this end, participant responses to a series of three items, which captured frequency of alcohol consumption within the past 12 months.

Parental-Child Relationships. Both mother-child and father-child reports form waves I and II were included in the analyses. Adolescents were asked eight questions about the parent-child relationship, such as: “Your mother encourages you to be independent.” The internal consistency for the mother subscale ranges from $\alpha = .96-.97$ and the father subscale ranges from $\alpha = .98-.99$. 
Perceived Family Environment. Perceived family environment was assessed at waves I and II. Participants responded to a 5-item scale, measuring family support, and environment. Items include “How much do you feel like your family pays attention to you?” and “How much do you feel that you and your family have fun together?” The scale has an internal consistency of $\alpha = .93-.97$ across waves I and II.

Self-Esteem. Self-esteem was measured at waves I and II. Participants responded to a 4-item scale, measuring general self-esteem, such as “I feel I have many good qualities,” and “I have a lot to be proud of.” The four-item scale reflects questions from the longer Rosenberg (1965) scale. Though the scale is much shorter, research has shown that self-esteem may be adequately measured with fewer items (Robins, Hendin & Trzesniewski, 2001). The internal consistency ranges from $\alpha = .84-.87$ across waves I and II.

Statistical Analysis

Prior to testing the hypotheses, all measures were formed into latent factors through the use of confirmatory factor analysis to address any problem items. Multiple indices of fit such as the chi-square test, Root Means Square Error Approximation, Comparative Fit Index, and the Tucker-Lewis Index (Hu & Bentler, 1999) were examined to ensure that the hypothesized model fit the data. All analyses were completed using Mplus Statistical Software (Muthén & Muthén, 1998-2016). Each of the latent
difference score variables were fit individually, to ensure that the difference score variables converged. In a second step, a latent difference score mediation model (Selig & Preacher, 2009; MacKinnon, 2008), as shown in Figure 2.1, was applied to the data in order to examine the extent to which changes in perceived family environment, parent-child relationships, and self-esteem mediate the relationship between disordered eating and alcohol misuse.

**Figure 2.1:** Latent difference mediation model testing the mediational effects of changes in family environment, parent-child relationships, and self-esteem on changes in alcohol misuse
Because mediation involves an asymmetrical distribution and violates assumptions of normality, bias-corrected bootstrapping was used to test for mediation. Bias-corrected bootstrapping was selected because it has been shown to provide the most accurate confidence intervals and greater power when compared to other resampling methods (MacKinnon et al., 2004). To best handle missing data, the model was estimated using the full information maximum likelihood method (FIML). FIML was selected because it includes all available information from participants with incomplete cases, which increases power and retains more information than other methods of handling missing data (e.g., listwise or pairwise deletion), and has been shown to produce parameter estimates with less bias than other methods of handling missing data (e.g., mean imputation; Enders, 2010). The Mplus input file has been provided in Appendix G.

**Results**

Complete descriptive information for the current sample is displayed in Table 2.1. Among the sample, disordered eating scores were relatively low, child reports of parental-rejection were lower among mother figures than father figures. Family environment scores and self-esteem scores were moderate, specifically, they tended to fall close to the middle in the range of all possible scores. Whereas alcohol misuse scores were low across waves. Among the control variables, few parents received public assistance, were unable to afford their bills or did not graduate from high school or complete a GED.
Table 2.1: Sample Demographics and Study Variable Descriptive Statistics

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<tr>
<th>Characteristic</th>
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<tr>
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<tr>
<td>Wave II</td>
<td>1.86 (0.76)</td>
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<td>Father-rejection</td>
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<td>Wave II</td>
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<td>Wave II</td>
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<td>Wave I</td>
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<tr>
<td>Wave II</td>
<td>1.61 (1.19)</td>
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<td>Public assistance (Dad), No. (%)</td>
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<td>Financial hardship, No. (%)</td>
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<td>Did not graduate high school, No. (%)</td>
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<td>Parent 1 Alcohol Use, mean (SD)(^c)</td>
<td>1.20 (0.62)</td>
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<td>Parent 2 Alcohol Use, mean (SD)(^c)</td>
<td>2.30 (1.28)</td>
<td>1 – 5</td>
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Note. \(N = 10,480\)

\(^a\) Unless otherwise noted, data represent the Mean (SD) of the sample

\(^b\) Participants were able to respond with more than one racial category, resulting in a total percentage greater than 100

\(^c\) Parent alcohol use data were measured in Add Health as parent 1 and parent 2, rather than as Mother and/or Father
The results from the full latent difference score mediational model are displayed in Figure 2.2. Specifically, the results from the latent difference score mediational model suggest that changes in the environmental and internal processes of interest do not mediate the relationship between total disordered eating and changes in alcohol misuse among girls and women. More specifically, the results suggest that adolescent disordered eating does not predict changes in mother-child relationship ($p = .53$), father-child relationship ($p = .40$), perceived family environment ($p = .67$), or self-esteem ($p = .70$), which in turn do not predict changes in alcohol misuse ($ps = .29 - .77$). These findings indicate that the environmental and internal processes included in the study do not serve as the processes through which disordered eating behaviors exerts its impacts on alcohol misuse.

**Figure 2.2**: Results from the latent difference score mediation analysis including standardized path coefficients
Discussion

Alcohol misuse greatly diminishes personal and psychological well-being. Among those who abuse alcohol, women experience more severe medical and psychiatric consequences, which has resulted in a strong interest in uncovering the etiological course of alcohol misuse among women through longitudinal studies. Of particular interest within the present study, disordered eating behaviors impact girls and women at a substantially higher rate than boys and men (Neumark-Sztainer et al., 2011) and have been shown to predict future alcohol misuse, among other substances (Gadalla & Piran, 2007; Ortega-Luyando et al., 2015; Micali et al., 2015), and tend to emerge earlier than alcohol misuse (Kurz et al., 2016; Chassin et al., 2004). Thus, it was hypothesized that disordered eating among adolescent girls serves as an early risk factor for alcohol misuse among adult women.

To test the long-term impacts of disordered eating on alcohol misuse, the ecological theory of human development was applied (Bronfenbrenner, 1977). Specifically, it was hypothesized that adolescent girls have a reciprocal relationship with their environment, in which disordered eating influences their family environment and internal processes which in turn influence later health behaviors, namely, alcohol misuse. To this end, perceived family environment, parent-child relationships, and self-esteem were identified as social environmental and internal processes related to both disordered eating (Sandler et al., 2014; Conklin et al., 2014; Johnson, Pratt & Wardle, 2012) and alcohol misuse (Tarantino et al., 2015; Chou, Liang & Sareen, 2011) and were thus
examined as potential processes underlying this relation. Specifically, a latent difference score mediational model was applied to examine the extent to which changes in perceived family environment, parent-child relationships, and self-esteem mediated the relationship between disordered eating and changes in alcohol misuse.

The results of the current study did not identify significant evidence that changes in perceived family environment, parent-child relationships, and self-esteem as a result of adolescent disordered eating predict subsequent changes in alcohol misuse among girls and women. Though previous research supports the negative impact of disordered eating behaviors on perceived family environments (Sandler et al., 2014), parent-child relationships (Spanos et al., 2010), and self-esteem (Conklin et al., 2014), the current study included only a single measure of disordered eating. It is possible that the addition of questions regarding frequency of disordered eating or a wider range of questions regarding disordered eating symptomology (e.g., extreme exercising and bingeing) would impact the results of the current study, highlighting the importance of test validity. Further, very few participants within the current study endorsed laxative use ($N = 29$) or diet pill use ($N = 126$). It is possible that the adolescent girls who use substances to control weight are more likely to experience later alcohol misuse, yet the current study did not have enough power to examine the impact of substance-based disordered eating on alcohol misuse, thus highlighting the importance of disordered eating subtypes when examining their impacts on later alcohol misuse and their associated underlying environmental and internal processes.

The current study contains several limitations. Perhaps most importantly, the measure of disordered eating is quite limited in that it only captures whether participants
engage in four disordered eating behaviors. To this end, the frequency of the behaviors cannot be capture and as a result, neither can the severity of disordered eating. Further the construct coverage of the disordered eating behavior measure included in the current study is limited. In addition, other environmental constructs (i.e. social environment) were not included in the current study, and thus a complete picture of social environment is not captured in the current study. Moreover, the current study included data from the Add Health dataset (Harris, 2012) for which wave I was collected between 1994-1995. Because disordered eating behaviors have steadily increased in prevalence among girls ages 15-19 over the last two decades (Smink, van Hoeken, & Hoek, 2012), it is possible that the Add Health data does not represent the frequency of disordered eating among the current US population. Lastly, the current study is limited to adolescent girls. Future research would benefit from a longitudinal examination into etiological differences in alcohol misuse emergence by gender.

Overall, the current study contributes to the literature on the etiology of alcohol misuse among girls and women by applying a novel mediational approach to examine the underlying role of changes in family environmental processes and self-esteem in the longitudinal relationship between disordered eating and alcohol misuse among adolescent girls and adult women. Specifically, guided by both the ecological theory of human development and previous empirical work, the main goal of the study was to determine the extent to which changes in family environment, parent-child relationships, and self-esteem mediate the relationship between disordered eating and changes in alcohol misuse. The lack of significant findings indicates that the current measure of disordered eating does not predict changes in the environmental and internal constructs of interest,
thus the results indicate that changes in perceived family environment, parent-child relationships, and self-esteem do not serve as the processes through which disordered eating exerts its impacts on changes in alcohol misuse, but likely through alternative social environmental factors (e.g., friendships) and internal processes (e.g., decision making).
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CHAPTER 4
SUMMARY AND CONCLUSIONS

Summary

Developmental and prevention researchers are often interested in not only identifying predictors of maladaptive health behaviors, but uncovering how early predictors exert their impacts on distal health outcomes. Mediation analysis is a statistical tool that allows researchers to examine the processes through which early risk factors impact distal health factors. Latent difference score mediation models extend the bounds of traditional mediation by allowing researchers to evaluate change within individuals and variation in change across individuals without constraining change to be systematic over time (Selig & Preacher, 2009; MacKinnon, 2008). As described throughout this project, adequate power, minimal bias, and acceptable coverage are necessary to accurately detect effects within any given model (Muthén & Muthén, 2002). When applied, latent difference score mediation examines the extent to which changes within a predictor variable predict changes in a mediating variable, which in turn predict changes within an outcome variable. From an ecological developmental perspective these mediators, or underlying processes, are often accounted for by various social environmental and internal processes (Bronfenbrenner, 1977).

The current project includes a series of two studies in which: (1) a Monte Carlo study was conducted to develop a set of empirical sample guidelines for the use of latent...
difference score mediation; and (2) latent difference score mediation was applied to examine the underlying gender-specific processes that explain the etiology of alcohol misuse among women. The aims of this two-study project were three-fold: (1) evaluate the performance of various latent difference score mediation model structures through a Monte Carlo simulation study; (2) use the results from the Monte Carlo study to develop a set of empirical sample size guidelines for use of latent difference score mediation model structures; and (3) apply one of the latent difference score mediation model structures to determine the extent to which environmental and internal processes mediate the relation between disordered eating among adolescent girls and alcohol misuse among adult women. The key findings from each of two studies are described below.

**Key Findings from Study 1**

The goals of Chapter II were to evaluate the performance of latent difference score mediation across population models and model structures, and to develop a generalizable set of guidelines for the use of latent difference score mediation models. In order to adequately establish a set of empirical guidelines for the use of any statistical method, several conditions must be met. Specifically, parameter and standard error biases must not exceed 10% for any parameter in the model, nor shall they exceed 5% for the parameters being tested, which refers to the indirect path in the current study (Muthén & Muthén, 2002). Moreover, coverage must range from .91 - .98, and statistical power
should be close to .80 (Muthén & Muthén, 2002). Chapter II included a Monte Carlo simulation study with the goal of developing a set of guidelines for the use of latent difference score mediation, for which all aforementioned conditions were met.

The results from Chapter II offer empirical sample estimates among ten model structures and nine population models across each structure, resulting in a total of ninety empirical sample estimates that range from 120 – 1050. The empirical sample estimates follow similar trends to those among other mediational models (e.g., Fritz & MacKinnon, 2007), thought latent difference score mediation models require larger sample sizes than less complex mediational designs. Monte Carlo simulations revealed that the required sample size to detect mediation varied greatly by model structure and population model estimates.

The applied findings from Chapter II are fourfold. First, population models with larger effect size pairings generally required smaller sample sizes than those with smaller effect size pairings. Which suggests that researchers must carefully consider their hypothesized effect sizes for each pathway prior to determining their sample size. Second, the magnitude of effect for the $b$ pathway was found to contribute more to estimates of statistical power than the effect size of the $a$ pathway, highlighting the importance of determining the hypothesized $b$ pathway for any study planning to use latent difference score mediation. Third, while population models with larger effect size product terms required smaller sample sizes to achieve adequate power than those with smaller effect size product terms, these models exchange statistical power for inflated parameter and standard error biases. Thus, the final empirical estimates among models with MM, ML, LM, and LL effect size pairings reflect sample sizes with much greater
than .80 power. Finally, less complicated model structures (e.g., dichotomous versus change score predictor) were found to require smaller sample sizes than more complicated model structures. Sample size estimates increased with the addition of each difference score variable, highlighting the virtue of simplicity in meditational analysis. With this in mind, it is suggested that difference score variables be included when modeling change is necessary to answer the research question at hand, rather than modeling a difference score factor for each variable in a study.

On the basis of the three key findings from Chapter II, there are four main recommendations for researchers interested in using latent difference score mediation models: (1) researchers must thoroughly evaluate the literature to determine the expected effect sizes before choosing their sample size; (2) empirical sample estimates should be viewed as a lower bound; (3) change scores should be included as necessary within any given model; and (4) the number of mediators and kinds of indirect effects to be tested must be determined before selecting a sample size.

Chapter II contributes to the literature by providing a wide range of empirical sample size estimates for single mediator latent difference score mediation models. The results from the Monte Carlo study provide researchers with a starting point for sample size estimates when using latent difference score models. Taken together, the results from the Monte Carlo simulation study indicate that both model structures and population models contribute to empirical sample size estimates. In general, future researchers should view the empirical sample estimates from Chapter II as a lower bound, as the Monte Carlo simulation included normally distributed data with no missing data. Future researchers should also develop a set of empirical sample guidelines for latent difference
score mediation models with non-normally distributed data, multiple mediators, and missing data among the predictors, mediators, and outcomes.

**Key Findings from Study 2**

Alcohol misuse has extensive consequences for psychiatric and physical health (Substance Abuse and Mental Health Services Administration, 2014), which more strongly impact women when compared to men (Nolan-Hoeksema, 2004). One notable challenge in understanding the complexities underlying the development of alcohol misuse concerns the substantial gender differences in alcohol risk factors (Pisetsky, Chao & Dierker, 2008; Walitzer & Dearing, 2006), a topic in which studies are still in high demand. While disordered eating behaviors have been identified as an early predictor of alcohol misuse among women, the processes through which disordered eating impacts alcohol misuse remain unclear.

Guided by the ecological theory of human development (Bronfenbrenner, 1977), social environmental and internal processes related to both disordered eating and alcohol misuse were evaluated as potential mediators. More specifically, the mediating role of perceived family environment, parent-child relationships, and self-esteem were examined. Latent difference score mediation was applied to model the effect of disordered eating among adolescent girls on changes in the social environmental and internal processes of interest, which in turn predicted changes in alcohol misuse.
Overall, the current study contributes to the literature on the etiology of alcohol misuse among girls and women by applying a novel mediational approach to examine the extent to which changes in social environmental and internal processes mediate the relation between disordered eating and changes in alcohol misuse. Though previous research supports the direct effect of disordered eating on the hypothesized mediators (Conklin et al., 2014; Johnson, Pratt & Wardle, 2012; Spanos et al., 2010) and the hypothesized mediators on alcohol misuse (Tarantino et al., 2015; Chou, Liang & Sareen, 2011), the results from Chapter III did not find significant evidence for mediating role of perceived family environment, parent-child relationships, or self-esteem within the disordered eating and alcohol misuse relation. The results from Chapter III highlight the need to further evaluate the social environmental and internal processes through which disordered eating exerts its impacts on alcohol misuse.

The lack of significant findings suggest that unmeasured social environmental processes may serve as the processes through which disordered eating exerts its impact on alcohol misuse. Moreover, the lack of significant findings may be a result of the specific measure of disordered eating behaviors included in the study. Future researchers should evaluate the role of friendships and social networks in the relation between disordered eating and alcohol misuse, as well as the predictive role of varying subtypes of disordered eating behaviors.
Conclusions

Grounded in developmental and prevention research, the current project contributes to the field from both quantitative and applied perspectives. Specifically, the two studies included in this project test the quantitative properties of latent difference score mediation and apply the novel methodology to evaluate the underlying processes related to alcohol misuse among women. The current project contributes to the quantitative area of developmental and prevention research by offering a set of guidelines for researchers interested in examining change both within and across people over time through the use of latent difference score mediation models. From an applied perspective, the current project applies advanced mediation methods to answer complex questions related to the gender-specific etiology of alcohol misuse. In sum, this project evaluates and applies complex mediational models to expand the current understanding of developmental methodology and the emergence of maladaptive health behaviors.
REFERENCES


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APPENDICES
APPENDIX A. Covariance Algebra for Chapter II

The equations below were used to calculate the parameter values included in the Chapter II. Within the following equations, \( \text{Var} \) represents the variance of a variable, \( \text{Cov} \) is a covariance, \( \alpha, b \) and \( c' \) are unstandardized path estimates, and \( \text{Var}(e) \) is a residual variance term. More detailed accounts of the covariance algebra used to calculate parameter values for a Monte Carlo simulation are described in MacKinnon (2008).

Unstandardized \( \alpha \) path estimates:

\[
\alpha = \frac{\text{Cov}(X, M)}{\text{Var}(X)}
\]

Unstandardized \( b \) path estimates:

\[
b = \sqrt{R^2}
\]

Unstandardized \( c' \) path estimates:

\[
c' = \frac{\sqrt{R^2}}{\sqrt{\text{Var}(X)}}
\]

Covariance between \( X \) and \( M \) variables:

\[
\text{Cov}(X, M) = \alpha \cdot \text{Var}(X)
\]
Variance of $X$:

$$Cov(X, X) = E[(X - E(X))^2]$$

Equation used to solve for $Var(e_M)$, when $Var(M)$ is known:

$$Var(M) = a^2 \cdot Var(X) + Var(e_M)$$

Equation used to solve for $Var(e_Y)$, when $Var(Y)$ is known:

$$Var(Y) = c'^2 \cdot Var(X) + b^2 \cdot Var(M) + 2 \cdot bc' \cdot Cov(X, M) + Var(e)$$

When the equation included a change score the following terms were added to their respective equation:

$$\beta^2 \cdot Var(X)$$

$$\beta^2 \cdot Var(M)$$

$$\beta^2 \cdot Var(Y)$$
APPENDIX B. Sample Mplus Code for Chapter II

**TITLE:** MONTE CARLO WITH Xc AND MEDIUM M AND Yc

**ANALYSIS:** Estimator = ML;
Bootstrap is 1000;

**MONTECARLO:**

names are
x1A X1B X1C
X2A X2B X2C
M2A M2B M2C
y3a y3b y3c
Y4A Y4B Y4C;
nobs = 265;
nreps = 1000;
seed = 17721;

**MODEL POPULATION:**

!structure for X

[X1A - X1C @ 0];
X1A - X1C@.91;

[X2A - X2C @ 0];
X2A - X2C@.91;

X1 BY X1A@1 X1B@.98 X1C@.92;
X2 BY X2A@1 X2B@.98 X2C@.92;
X1@1;
X2@0;

!structure of mediator

[M2a - M2c @ 0];
M2a - M2c @ .36; !this is the b path value
M2 BY M2a@1.0 M2b@.95 M2c@.89;

[M2@0]; !SETS LATENT MEAN TO 0
M2@.78; !SETS RESIDUAL TO .87

!structure for outcome

[Y3a - Y3C @ 0];
Y3A - Y3C @ .72; !this is the residual Y
[Y4a - Y4C @ 0];
Y4A - Y4C @ .72;

Y4 BY Y4A@1 Y4B@.93 Y4C@.88;

Y3 BY Y3A@1 Y3B@.93 Y3C@.88;

[Y3@0];
[Y4@0];

Y3@.72;
Y4@0;

!STRUCTURE OF THE CHANGE SCORE VARIABLES

!BETA
X2 ON X1@1;
XDIFF BY X2@1;
XDIFF ON X1@-.3;
!PSI
XDIFF@.91;
!BETA
Y4 ON Y3 @1;
YDIFF BY Y4 @1;
YDIFF ON Y3*-.3;
!PSI
YDIFF@.63; !residual after accounting for Xc and Yc
M2 ON XDIFF*.36;
YDIFF ON M2*.36 XDIFF*.14;

MODEL INDIRECT:
YDIFF IND XDIFF;

MODEL:

!structure for X

[X1A - X1C @ 0];
X1A - X1C*.91;

[X2A - X2C @ 0];
X2A - X2C*.91;

X1 BY X1A@1 X1B*.98 X1C*.92;
X2 BY X2A@1 X2B*.98 X2C*.92;

X1*1;
X2@0;

!structure of mediator

[M2a - M2c @ 0];
M2a = M2c* .36; !this is the b path value
M2 BY M2a@1.0 M2b*.95 M2c*.89;

[M2@0];
M2*.78; !SETS RESIDUAL
\[ [Y3a - Y3C @ 0]; \]
\[ Y3A = Y3C* .72; \]
\[ [Y4a - Y4C @ 0]; \]
\[ Y4A = Y4C* .72; \]

\[ Y4 \text{ BY } Y4A@1 \ Y4B* .93 \ Y4C*.88; \]
\[ Y3 \text{ BY } Y3A@1 \ Y3B*.93 \ Y3C*.88; \]
\[ [Y3@0]; \]
\[ [Y4@0]; \]
\[ Y3*.72; \]
\[ Y4@0; \]

!STRUCTURE OF THE CHANGE SCORE VARIABLES

!BETA
X2 ON X1@1;
XDIF\(F\) BY X2@1;
XDIF\(F\) ON X1*-.3;
!PSI
XDIF\(F\)*.91;

!BETA
Y4 ON Y3@1;
YDIF\(F\) BY Y4@1;
YDIF\(F\) ON Y3*-.3;
!PSI
YDIF\(F\)*.63;

M2 ON XDIF\(F\)*.36;
YDIF\(F\) ON M2*.36 XDIF\(F\)*.14;

MODEL INDIRECT:
YDIF\(F\) IND XDIF\(F\);

output: tech9 CINTERVAL(BCBOOTSTRAP);
APPENDIX C. Disordered Eating Behavior Questionnaire for Chapter III

Prompt:

“During the past seven days, have you done the following things in order to lose weight or to keep from gaining weight”

Response categories:

Yes or No

Items:

1. Dieted
2. Made yourself vomit
3. Took diet pills
4. Took laxatives
APPENDIX D. Alcohol Misuse Questionnaire for Chapter III

Response categories:

Everyday or almost everyday, 3 to 5 days a week, 1 or 2 days a week, 2 or 3 days a month, once a month or less (3-12 times in the past twelve months, 1 or 2 days in the past 12 months, never

Items:

1. During the past 12 months, on how many days did you drink alcohol?
2. Over the past 12 months, on how many days did you drink 5 or more drinks in a row?
3. During the past 12 months how often did you get drunk?
APPENDIX E. Perceived Family Environment Questionnaire for Chapter III

Response categories:

Not at all, Very little, Somewhat, Quite a bit, Very much

Items:

1. How much do you feel that your parents care about you?
2. How much do you feel that people in your family understand you?
3. How much do you feel that you want to leave home?
4. How much do you feel that you and your family have fun together?
5. How much do you feel that your family pays attention to you?
APPENDIX F. Mother-Child Relationship Questionnaire for Chapter III

Prompt:

“How strongly do you agree or disagree with the following statements?”

Response categories:

Strongly agree, Agree, Neither agree nor disagree, Disagree, Strongly disagree

Items:

1. Most of the time, your mother is warm and loving toward you
2. Your mother encourages you to be independent
3. When you do something wrong that is important, your mother talks about it with you and helps you understand why it is wrong
4. You are satisfied with the way your mother and you communicate with each other
5. Overall, you are satisfied with your relationship with your mother
APPENDIX G. Father-Child Relationship Questionnaire for Chapter III

Prompt:

“How strongly do you agree or disagree with the following statements?”

Response categories:

Strongly agree, Agree, Neither agree nor disagree, Disagree, Strongly disagree

Items:

1. Most of the time, your father is warm and loving toward you
2. You are satisfied with the way your father and you communicate with each other
3. Overall, you are satisfied with your relationship with your father
APPENDIX H. Self-Esteem Questionnaire for Chapter III

Prompt:

“How strongly do you agree or disagree with the following statements?”

Response categories:

Strongly agree, Agree, Neither agree nor disagree, Disagree, Strongly disagree

Items:

1. I feel I have many good qualities
2. I have a lot to be proud of
3. I like myself just the way I am
4. I am doing things just about right
APPENDIX F. Demographic Items Included in Chapter III Analyses

Child questionnaire

1. What is your birthdate?

2. What is your sex?

3. Are you of Spanish/Latino origin?

4. What is your race?

5. Does your resident father receive public assistance, such as welfare?

6. Does your resident mother receive public assistance, such as welfare?

Parental Questionnaire:

7. Do you have enough money to pay your bills?

8. How far did you go in school?

9. How far did your current spouse/partner go in school?

10. How often in the last month, have you had five or more drinks on one occasion?

11. During the past 12 months, about how often did your current partner drink alcohol?
APPENDIX G. Mplus Code for Chapter III

TITLE: Mediation with FIML and Covs

DATA:
FILE IS Diss_women_112.dat;

VARIABLE: NAMES ARE
IMONTH SCID SSCID COMMID
SCH YR BIO SEX VERSION BIRYR
LOC1 HIS1 WHITE AAMER AMIND
ASIAN OTHER RACE LANG USBORN
USCIT GRADE MOMAST DADAST PAED
BILLS PAAU PAED2 PAAU2 DEB1A
DEB1B DEB1C DEB1D MOM1A MOM1B
MOM1C MOM1D MOM1E DAD1A DAD1B
DAD1C FE1A FE1B FE1C FE1D
FE1E DM1A DM1B DM1C SE1A
SE1B SE1C SE1D SE1E DEB2A
DEB2B DEB2C DEB2D MOM2A MOM2B
MOM2C MOM2D MOM2E DAD2A DAD2B
DAD2C FE2A FE2B FE2C FE2D
FE2E SE2A SE2B SE2C SE2D SE2E
DM2A DM2B DM2C ALC2A ALC2B ALC2C
ALC3A ALC3B ALC3C WC_tot WCT_dct;

USEVARIABLES
MOM1A MOM1B MOM1C MOM1D MOM1E
MOM2A MOM2B MOM2C MOM2D MOM2E
FE2A FE2B FE2C FE2D FE2E
FE1A FE1B FE1C FE1D FE1E
DAD1A DAD1B DAD1C DAD2A DAD2B
DAD2C SE1A SE1B SE1C SE1D
SE1E SE2A SE2B SE2C SE2D SE2E
WCT_dct ALC2A ALC2B ALC2C
ALC3A ALC3B ALC3C WHITE HISI
PAAU PAED PAED2 PAAU2 MOMAST
DADAST;

MISSING are all (-999);

ANALYSIS:
BOOTSTRAP IS 1000;
MODEL:
!LAMBDA

Wct_Dct;
[Wct_Dct];

FE1 BY FE1A*(5)
   FE1B (6)
   FE1C (7)
   FE1D (8)
   FE1E @1;

FE2 BY FE2A*(5)
   FE2B (6)
   FE2C (7)
   FE2D (8)
   FE2E @1;

FE2A*; FE2B*; FE2C*; FE2D*; FE2E*;
FE1A*; FE1B*; FE1C*; FE1D*; FE1E*;

FE2A WITH FE1A*; FE2E WITH FE1E*; FE1B WITH FE2B*;
FE2C WITH FE1C*; FE2D WITH FE1D*;

!BETA
FE2 ON FE1 @1;
FDIFF BY FE2 @1;
FDIFF ON FE1*;
!PSI
FE1*; FE2@0; FDIFF*;

DAD1 BY DAD1A*(5)
   DAD1B (6)
   DAD1C @1;

DAD2 BY DAD2A*(5)
   DAD2B (6)
   DAD2C @1;
DAD2A*; DAD2B*; DAD2C*;
DAD1A*; DAD1B*; DAD1C*;

DAD2A WITH DAD1A*; DAD1B WITH DAD2B*;
DAD2C WITH DAD1C*;

!BETA
DAD2 ON DAD1 @1;
DDIFF BY DAD2 @1;
DDIFF ON DAD1*;
!PSI
DAD1*; DAD2@0; DDIFF*;

SE1 BY SE1A*(5)
  SE1B (6)
  SE1C (7)
  SE1D (8)
  SE1E @1;

SE2 BY SE2A*(5)
  SE2B (6)
  SE2C (7)
  SE2D (8)
  SE2E @1;

SE2A*; SE2B*; SE2C*; SE2D*; SE2E*;
SE1A*; SE1B*; SE1C*; SE1D*; SE1E*;

SE2A WITH SE1A*; SE2E WITH SE1E*; SE1B WITH SE2B*;
SE2C WITH SE1C*; SE2D WITH SE1D*;

!BETA
SE2 ON SE1 @1;
SDIFF BY SE2 @1;
SDIFF ON SE1*;
!PSI
SE1*; SE2@0; SDIFF*;
MOM1 BY MOM1A*(5)
MOM1B (6)
MOM1C (7)
MOM1D (8)
MOM1E @1;

MOM2 BY MOM2A*(5)
MOM2B (6)
MOM2C (7)
MOM2D (8)
MOM2E @1;

MOM2A*; MOM2B*; MOM2C*; MOM2D*; MOM2E*;
MOM1A*; MOM1B*; MOM1C*; MOM1D*; MOM1E*;

MOM2A WITH MOM1A*; MOM2E WITH MOM1E*; MOM1B WITH MOM2B*;
MOM2C WITH MOM1C*; MOM2D WITH MOM1D*;

!BETA
MOM2 ON MOM1 @1;
MDIFF BY MOM2 @1;
MDIFF ON MOM1*;

!PSI
MOM1*; MOM2@0; MDIFF*;

ALC2 BY ALC2A@1
ALC2B(8)
ALC2C (9);
ALC3 BY ALC3A@1
ALC3B(8)
ALC3C (9);

ALC3A*; ALC3C*; ALC3B*;
ALC2A*; ALC2C*; ALC2B*;

ALC3A WITH ALC2A*; ALC3C WITH ALC2C*;
ALC3B WITH ALC2B*;
ALC3A WITH ALC2A*; ALC3C WITH ALC2C*;
ALC3B WITH ALC2B*;

!BETA
ALC3 ON ALC2 @1;
ADIFF BY ALC3 @1;
ADIFF ON ALC2*;
!PSI
ALC2*; ALC3@0; ADIFF*;

!BETA
SDIFF ON Wct_dct;
DDIFF ON Wct_dct;
FDIFF ON Wct_dct;
MDIFF ON Wct_dct;
ADIFF ON SDIFF DDIFF FDIFF MDIFF Wct_Dct;

ADIFF ON WHITE HISP PAAU PAED
PAED2 PAAU2 MOMAST DADAST;

MODEL INDIRECT:

ADIFF IND SDIFF Wct_dct;
ADIFF IND DDIFF Wct_dct;
ADIFF IND FDIFF Wct_dct;
ADIFF IND MDIFF Wct_dct;

output: sampstat stdyx tech4 CINTERVAL (BCBOOTSTRAP) patterns;
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*Latent Difference Score Mediation Analysis in Developmental Research: A Monte Carlo Study and Application*

2013 M.A. Psychology
University of Hartford, West Hartford, CT

*The Impact of Body Dissatisfaction, Stress, and Eating Behaviors on Weight Cycling Among College Women*

2012 B.A. Psychology
University of Hartford, West Hartford, CT

*Project Weightless: A literature review and intervention concerning body image, eating disorders and mandatory Body Mass Index (BMI) weigh-ins nationwide*

RESEARCH AREAS

Primary: Longitudinal Mediation, Eating Disorder Prevention, Health Risk Behavior
Secondary: Gender and Racial Differences in Health Risk, Mixture Modeling

ACADEMIC POSITIONS

August 2014 – Present Pre-doctoral Fellow
Prevention Science Lab
Utah State University
Advisor: Ginger Lockhart, Ph.D.
December 2015 – January 2018  
**Co-Leader**  
Neurodiversity Group  
Utah State University

July 2014 – December 2014  
**Research Assistant**  
Avalon Hills Eating Disorder Clinic  
*Advisors: Ginger Lockhart, PhD., and Michael P. Twohig*

July 2011 – May 2013  
**Project Coordinator**  
Project Weightless | Women for Change  
University of Hartford  
*Advisor: Mala Matacin, Ph.D.*

July 2012 – May 2013  
**Graduate Administrative Assistant**  
Hillyer College  
University of Hartford

August 2012 – January 2013  
**Research Intern**  
The Center for Research, Inc.  
Meriden, CT

December 2009 – August 2013  
**Research Assistant**  
Department of Psychology  
University of Hartford  
*Advisor: Mala Matacin, Ph.D.*

December 2009 – August 2012  
**Leadership Facilitator**  
The Women’s Education and Leadership Fund  
University of Hartford

**GRANTS—Submitted but Not Funded**

**Principal Investigator** (Faculty Mentors on Submission: G. Lockhart, & M. Twohig)  
“Ecodevelopmental processes of disordered eating and substance use among girls and women”  
National Institutes of Health: National Institute on Drug Abuse (F31)  
Total Requested Costs: $86,000  
Status: Not funded

**Student Investigator** (Multiple PIs: G. Lockhart, & M. Twohig)  
“Ecodevelopmental processes of disordered eating and alcohol abuse among girls and women”  
National Institutes of Health: National Institute on Alcohol Abuse and Alcoholism (R03)  
Total Requested Costs: $100,000  
Status: Not funded
PUBLISHED WORKS IN REFEREED JOURNALS


WORKS UNDER REVIEW OR IN REVISION


WORKS IN PROGRESS


SELECTED PRESENTATIONS


**INVITED PRESENTATIONS**


Simone, M., Rivera, K., Swanson, P. (2012). *How to be Active in your Community: Alumnae Panel*. Women’s Leadership Summer Bridge Program. West Hartford, CT

**TEACHING EXPERIENCE**

*Undergraduate Scientific Thinking and Methods*
Upper division undergraduate course of methods in social science research.

*Introduction to Psychology as a Career and Profession*
Introductory course of the trajectory of a psychology major.

*Laboratory Section of Psychological Statistics*
Introductory course to descriptive and inferential statistics for the behavioral sciences.
ACADEMIC SERVICE

Graduate Student Representative (2015 – 2016): Utah State University
Chapter Vice President (2011 – 2012): Psi Chi; University of Hartford

HONORS

Recipient, Walter R. Borg Scholarship and Research Productivity Award, Utah State University; 2017-2018
Recipient, Graduate Enhancement Award, Utah State University Student Association; 2016-2017
Recipient, A. Richard Brayer Award for Graduating Masters Student with the Highest GPA, University of Hartford; 2012-2013
Recipient, Graduate Research Award, University of Hartford, 2012-2013
Recipient, Mary Louise Leonhardt Award for Greatest Community Contribution, University of Hartford, 2011-2012
Recipient, Undergraduate Research Award, University of Hartford, 2011-2012
Recipient, Program of the Year Award, University of Hartford, 2011-2012
Recipient, Dr. Melvin Goldstein English Scholars, University of Hartford, 2010-2011