EFFECTS OF MISSING DATA ON STATISTICAL POWER TO DETECT CHANGE IN FAMILY-BASED PREVENTIVE INTERVENTION RESEARCH

by

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(Under the Direction of Adam Davey)

ABSTRACT

Loss of statistical power in family-based preventive intervention research often results from missing data. This dissertation examines interactions between type and amount of missing data, sample size and statistical power to detect group differences in longitudinal change.

Study 1, examines the statistical power of a 5-wave two-group (Treatment and Control) growth curve model with a small (.2) effect size as a function of missing data (5% to 95% missing), type of missingness (Missing Completely at Random, MCAR, or Missing at Random, MAR), and sample sizes (N=100 to N=1000). Results indicate that for moderate samples and amounts of missing data loss in power is proportionally higher. Interactions between sample size, statistical power and missing data are further considered.

Study 2 considers the effects of measurement and design decisions to improve and moderate the effects of missing data on statistical power in the two-group growth curve model as a function of varying amounts missing data and sample sizes. When reliability of indicators is high, the effect of missing data was found to diminish. Furthermore, inclusion of an auxiliary variable suggested that a covariate is particularly beneficial in increasing statistical power at smaller sample sizes and with models with high percentage of missing data. Lastly, drop-out
patterns were evaluated as a function of type of missing data (MCAR vs. MAR) with 50% missing data at different sample sizes. Results indicate that the pattern in which participants dropped out of a study affected power even for identical amounts of missing data.

Methods to increase the statistical power in the presence of varying amount of missing data and sample size are recommended, with suggestions for future research.

INDEX WORDS: Prevention, Family intervention, Missing data, Statistical power, Structural Equation Modeling, Reliability, Covariates, Pattern missingness, Growth curve model.
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DEDICATION

This dissertation is dedicated to my husband, Sital and my parents, Mani and Ramnik. Their enduring patience, support and encouragement are the very reason for what I am and where I am today.
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CHAPTER 1
INTRODUCTION

Family-centered intervention programs have proven to be effective in reducing problem behaviors, enhancing competencies and improving intrafamilial relationships (e.g. Redmond, Spoth, Shin, & Lepper, 1999; Spoth & Redmond, 2003; Spoth, Redmond, Trudeau, & Shin, 2002; Taylor & Biglan, 1998). However, a majority of these research programs lack the statistical power to clearly estimate whether the intervention program has produced desired effects over and above what would have occurred without an intervention (Hansen, 1992; Maxwell, 2004, Rossi, 1990). Lack of statistical power is a serious threat to future funding of such intervention programs. Several analytical, design and measurement issues in family-based preventive intervention research are responsible for the insufficient statistical power in this area of research; however among these issues the problem of missing data is the least understood among these.

Research in the present decade has relied on methods such as growth curve models to measure the efficacy of intervention programs. Growth curve models are far superior to ANCOVA models in terms of statistical power to detect treatment effects (Curran & Muthén, 1999; Fan, 2003). Since growth curve models allow for the estimation of individual differences in change over time, researchers can examine the differential responses to treatment to identify factors associated with stronger or weaker responsiveness to treatment. Researchers have also noted that growth curve models can easily be modified to allow for computation of specific estimates of power under a variety of assumptions and conditions such as the number of subjects...
in the control group, the number of subjects in an experimental group, and the number of waves of data (Curran & Muthén, 1999; Muthén & Curran, 1997). To date, however, the growth curve model has not been assessed in terms of its efficiency in the presence of different amounts of missing data or types of missing data (missing at random versus missing completely at random).

Several researchers have noted the importance of using reliable measures in order to provide sufficient rigor to the research design by reducing the measurement error and explaining higher variance, thus increasing power. However, not enough research has been done to understand how the reliability of instruments affects statistical power as a function of missing data and sample size.

Methodologists studying missing data mechanisms have often encouraged researchers to include covariates or predictors associated with missingness in their models to recover the parameter estimates due to missing data. Little research, however, has been done to examine whether the inclusion of a covariate at different sample sizes could increase statistical power in the presence of missing data.

Intervention researchers have also realized that in order to lower the rates of attrition and to collect quality data from subjects, a unique form of design known as planned missingness at different waves could be a cost-effective design. Recently, this design has received much attention; however, questions concerning which particular pattern of missing data causes the least drop in statistical power have not been fully addressed with regard to the broad spectrum of sample sizes, type and amount of missing data.

This dissertation examines the statistical power of a two-group growth curve model in the presence of different amounts of missing data using diverse sample sizes. Although one might expect that any amount of missing data would simply reduce the power, recent research suggests
that not all missing data are created equal, i.e. when data are missing completely at random and when data are missing at random. The loss of statistical power, therefore, is additionally examined as a function of the type of missing data. Additionally, the role of salient factors such as reliability of the measures used, the inclusion of covariates and the pattern of missingness in the different waves in moderating the effects of missing data on statistical power are examined. Finally, limitations and future research prospects are discussed.
CHAPTER 2
LITERATURE REVIEW

In recent years countries all over the world, including the United States, have been grappling with issues such as child abuse, juvenile violence and delinquency, school drop-out, substance abuse, teenage pregnancy, spouse abuse, elder abuse, divorce, single-parenthood and family caregiving (Ashery, Robertson, & Kumpfer, 1998; Forum on Child and Family Statistics, 2003; Grant, 2000; Johnston, O'Malley, & Bachman, 2004; Resnick et al., 1997; U.S. Department of Health and Human Services, 1998; Wilshire & Winterstein, 1998). As society’s concern over these persistent and perplexing problems has intensified, so have its efforts to resolve them. Several local, regional and national agencies have launched programs aimed at preventing, delaying or moderating the onset of these problems.

At the same time, scarce resources and budget deficits have forced policy makers to make choices concerning whom to fund depending on the demonstrated effectiveness of the intervention program. Evaluation research thus aims to determine the worth or merit of the program under investigation (Scriven, 1967). This is done by estimating whether the intervention program produced desired effects over and above what would have occurred without an intervention or, in some cases, with an innocuous intervention. It also does so by evaluating whether there is any change in the behavior or attitude of the person over time, above and beyond the developmental changes that occur in individuals and families over time. Recently, evaluation researchers have also been asking how, why and for whom do the preventive
intervention programs work? Much evaluation research however, lacks the statistical power to
detect effects of the intervention programs (Maxwell, 2004).

The review of literature in this section gives an overview of how methodological and
empirical traditions have developed in an effort to better address issues related to measuring
intervention effectiveness, particularly the power to detect significant changes in the context of
the family based preventive intervention programs. This chapter concentrates primarily on
issues of design and analysis. While a number of measurement issues such as factorial
invariance in longitudinal designs and autocorrelated residuals, are also relevant, these issues are
not reviewed here because they have not yet been fully developed within the family-based
preventive intervention research.

In order to address these questions, a brief introduction to power and its relation to
statistical inference will be given. Next, the state of affairs of family-based preventive
intervention programs will be examined. Thereafter, issues paramount to the low statistical
power in family based preventive interventions will be discussed. This will include the
developmental history of the measurement of change, followed by a discussion of the
contemporary research tools and techniques developed in response to the shortcomings of earlier
techniques of measuring change. Finally, methodological issues, in particular design issues,
analytical issues, and best practices developed in an effort to better address intervention
effectiveness and improve the ability to detect significant interindividual differences in
intraindividual change in the light of family-based preventive interventions, will be presented.
Power and Statistical Inference

About 40 years ago, Cohen (1969) built on the work of Neyman and Pearson (1928) to illustrate two types of possible errors in statistical inferences, depending on their unknown true state of affairs. When no effect is present in the population, but one is detected in an analysis, a Type I error ($\alpha$) has occurred. Generally, researchers consider the Type I error as a serious mistake and therefore want to minimize the probability of its occurrence. A probability of making an error 5 percent of the time is the criterion typically used by social scientists. In other words, an effect as large as the one detected in the study would occur only once every 20 times (i.e., $\alpha = 0.05$) by chance alone. This type of error and its role in statistical inference is familiar to every social scientist. In fact, even though Cohen (1994) himself has criticized the use of the $p < 0.05$ criterion on several grounds, typical research journals continue to require research articles to have a low Type I error as a prerequisite for publication (Cohen, 1994; Maxwell, 2004).

Cohen (1969, 1992) examined a second type of error, which occurs when there is an effect in the population but it is not detected in particular analysis. This Type II error ($\beta$) is the basis of statistical power calculations ($1 - \beta$) and is equally and sometimes more important to the process of valid statistical inference (Allison, Allison, Faith, Paultre, & PiSunyer, 1997; Kraemer, 1991; Maxwell, 2004). At a given level of statistical significance (e.g. $\alpha = 0.05$), statistical power, is the probability of detecting an effect of a given size (e.g. $d$; Cohen, 1988).

In the last decade there has been an increasing awareness of the role of statistical power in evaluation research (Cohen, 1992; Gillett, 1994, 1996, 2002; Hall & Heather, 1991; Jo, 2002; Kelley, Maxwell, & Rausch, 2003; Kraemer, 1991). Several books, journal articles and
statistical programs for power analysis have surfaced (see MacKinnon & Lockwood, 2003; Maxwell, 2004 for a review). At the same time, there are several other issues that have received growing attention. Specifically, the determination of power in Structural Equation Modeling (Jöreskog & Sörbom, 2000; Kaplan, 1995; Muthén & Curran, 1997; Satorra & Saris, 1985), alternatives to testing power for detecting whether an effect is small versus zero (Murphy & Myors, 2004; MacCallum, Browne, & Sugawara, 1996), and power calculations that adjust for the effects of clustered data (Brown, Wang, Muthén, & Dagne, 2000; Donner, 1985; Murray, 1998).

However, a majority of social and behavioral science research including intervention studies, continue to have a low probability of detecting a significant effect, if present (Bezeau & Graves, 2001; Hansen, 1992; Kosciulek & Szymanski, 1993; Maxwell, 2004; Mone, Mueller, & Mauland, 1996). This is largely because of several issues regarding design and analysis that have not been given enough consideration. For instance, how the nature and extent of missing data in intervention studies affect statistical power to detect change has been virtually ignored in the field of intervention research. Although one might expect that any missing data would simply reduce power, recent research suggests that not all missing data were created equal (i.e., some types may be more deleterious than others) (Davey, Shanahan, & Schafer, 2001; Schafer & Graham, 2002) and that planned missing data may actually be a cost-effective alternative to collecting complete data on all individuals (Graham & Donaldson, 1993; Graham, Hofer, Donaldson, MacKinnon, & Schafer, 1997; Graham, Hofer, & MacKinnon, 1996) and could also increase statistical power (Graham & Donaldson, 1993). In this chapter, several other design and analytical issues that have an impact on statistical power will be discussed in the light of family-based preventive intervention programs. Additionally, ongoing efforts and best practices in
order to improve designs and analysis of family-based preventive intervention programs in order to increase their statistical power to detect significant effects will also be discussed. The next section gives a brief overview of family-based intervention programs.

Family-Based Preventive Intervention Programs

Prevention researchers study individuals in their sociodemographic contexts. Family-based preventive intervention researchers often rely on a systems perspective (Bateson, 1971; Bertalanffy, 1975) to understand the influences of multiple contexts (e.g., individual variables, family, neighborhood, school, community) on human behavior Bronfenbrenner (1979). This perspective elucidates how the individual both influences and is influenced by these contexts over the course of development. One of the strongest proximal influences on individuals is the family context. A number of studies have substantiated the importance of risk and protective factors originating in the family as influences on problem behaviors in children and in adults alike (Masten et al., 1999; Garmezy, 1993; Gaugler, Zarit, & Pearlin, 1999; Redmond, Spoth, & Trudeau, 2002). Rigorous intervention research has also shown that family-centered interventions can be efficacious in reducing problem behaviors, enhance competencies, and improve intrafamilial relationships (e.g. Redmond et al., 1999; Spoth et al., 2002; Taylor & Biglan, 1998; Spoth & Redmond, 2003). Such findings highlight the potential benefits of interventions targeting relevant family processes such as family cohesion, parenting styles, parental monitoring and involvement.

Some of the well-known family-centered preventive intervention programs aiming to serve children, their families as well as older adults and their caregivers are Strong African-American Families Program (SAAF, Brody et al., 2004; Project FAMILY, Spoth, Redmond,
Shin, & Azevedo, in press; IOWA Strengthening Families Program (ISFP), Molgaard, Kumpfer, & Fleming, 1996; Frailty and Injuries: Cooperative Studies of Intervention Techniques (FICSIT), Ory et al., 2002). There are many more family-centered preventive intervention programs. Although the above list is not exhaustive, it gives a glimpse of the different areas and age groups which family intervention program target (for a detailed list of other preventive intervention programs and research groups see Society for Prevention Research, 2004; Spoth & Redmond, 2003).

While an emerging body of literature provides evidence of the benefits of family-focused preventive interventions for reducing general problematic behavior in the population (Brody et al., 2004; Spoth, Redmond, & Shin, 2001; Botvin, Baker, Dusenbry, Tortu, & Botvin, 1990; Roth, Haley, Owen, Clay, & Goode, 2001; Spoth et al., 2001), few of these programs have been evaluated rigorously. Also due to the design of these studies, many have inadequate power to detect significant change over time (Rossi, 1990; Hansen, 1992; Maxwell, 2004). This is primarily due to several methodological issues in the area of family preventive intervention research. Some examples of these issues include poor methods for studying change, design issues such as weak research designs, inadequate sample size, weak outcome measures and an insufficient number of waves of data, and analytic issues such as shape of trajectory of growth, and the non-independent nature of data and missing data. These issues will be discussed in more detail throughout this chapter.

Analysis of Change

This section of the chapter will briefly describe the historical development of methodologies that have been designed to study change and growth. Detailed below is a
summary of issues in the methodological field regarding whether change can actually be measured and ultimately how to improve its measurement. Next, contemporary research tools and techniques to measure change will be examined. To conclude this section, advantages of the latent growth curve models for preventive intervention research will be explored.

**History of Measuring Change**

Assessing intervention effects is inherently an investigation of change, in particular, the changes in the targeted behavior of interest brought about by the intervention program. Researchers have also realized that studying change essentially involves the implementation of longitudinal research designs.

Researchers in developmental psychology have distinguished between qualitative and quantitative changes for many years (Wohlwill, 1973; Baltes, 1987; Baltes, Reese, & Nesselroade, 1988). Qualitative changes typically examine differences in the factor structure of instruments (Jöreskog, 1979b). These factors, which change qualitatively, are referred to as dynamic constructs (Collins, Cliff, & Dent, 1988). On the other hand, quantitative changes employ instruments that provide precise scaling of individual variables throughout the range of attitudes and behavior to be measured over the entire span of the study. The methods and issues discussed in this chapter focus on these quantitative changes only.

No issue in the statistical literature has undergone so much scrutiny as how to measure change and whether or not researchers should even attempt to measure change (Cronbach & Furby, 1970; Linn & Slinde, 1977; Lord, 1963; Rogosa, 1988; Rogosa & Willett, 1983; Zimmerman & Williams, 1982a, 1982b). Willett (1988) argues that problems associated with this impasse stem primarily from the conceptualization of change instead of a flaw in the
statistical method of calculating different scores. Instead of looking at change as a continuous development over time, change when conceived of as “quantized acquisition of skills, attitudes and beliefs” (Willett, 1988, p. 347) is where the problem lies. In other words, the issue concerns whether we conceptualize change as a continuous process, for example, daily development changes in children or as stage-like changes, for example, entering into the developmental stage of adolescence.

An example of this confusion in methods employed in family preventive intervention research is the arbitrary use of two time points to measure change. One of the oldest methods to study change in these two-wave designs was to analyze the raw change (gain or difference) scores. Change scores based on two time points is computed as the difference between Time 1 and Time 2 score. Although simplistic, the change score is criticized chiefly because of the inverse relationship between the reliability of the difference score and the correlation between Time 1 and Time 2 measures (Bereiter, 1963; Cronbach & Furby, 1970; Furby, 1973; Lord, 1963; Rogosa, Brandt, & Zimowski, 1982). Critics note that the reliability of observed scores indicates the amount of error variance relative to the amount of true variance in the scores, wherein, reliability is the ratio of true variance to observed variance. Since the observed variance is made up of true and error variance, when there is no increase in true variance, greater error variance is synonymous with greater unreliability in the measure.

A related issue in the use of difference scores to represent change is the relationship between the resulting change score and Time 1 score (Benjamin, 1973; Bereiter, 1963; Bohrnstedt, 1969; Cronbach & Furby, 1970; Furby, 1973). One will notice that the correlation between the change score and Time 1 score tends to be negatively biased (Lacey & Lacey, 1962). As a result, critics have argued that methods that use difference scores as a measure of
change gives an unfair advantage to an individual with low initial values (Linn & Slinde, 1977). It is actually quite the reverse; the difference score is actually an unbiased estimate of true individual change. The problem is that it is an unbiased measure of true individual change; therefore, fallibility makes the difference score inadequate for studying correlates of true change (Rogosa et al., 1982; Rogosa & Willett, 1983; Rogosa & Willett, 1985a; Willett, 1988). Furthermore, if participants in the study change at different rates, then the time chosen to represent initial status will affect the magnitude and also possibly the direction of the correlation (Rogosa & Willett, 1983).

Additionally, (Rogosa & Willett, 1985b) noted that when the parameter of interest is positive but small or even zero, the correlation between observed status and observed change is expected to be negative. Thus the interpretation of the observed correlation between the difference score and initial status may be invalid when the correlation between true initial status and true change is small or zero. In summary, the raw difference score is not an optimal statistic for studying “correlates of change” (Willett, 1988) when the correlate is initial status or some other variable. These two-wave designs are also considered to be less powerful in detecting change (Maxwell & Delaney, 2004; Murphy & Myors, 2004).

Concerns about the problems with difference scores resulted in several other variations in order to “fix” raw difference scores to represent change. At the same time, researchers were preoccupied in analyzing change over time as characteristic of groups without considering change at the individual level. Several alternative methods were proposed that could be applied to longitudinal data for the purpose of studying mean group differences in change over time. These methods included residualized change scores (Cronbach & Furby, 1970; Webster & Bereiter, 1963), Analysis of Variances (ANOVA), and its variants such as multivariate analysis...
of variances (MANOVA) and analysis of covariances (ANCOVA) (Kenny, 1975; O'Brien & Kaiser, 1985). Although widely used, these methods have been criticized frequently on both statistical and theoretical grounds (Rogosa & Willett, 1985a; Stoolmiller, Duncan, Bank, & Patterson, 1993; Willett, 1988).

With regard to the residualized change scores, change is measured as the residual or error between the observed Time 2 score and the expected Time 2 score as predicted by the Time 1 score. Similarly, when subjects were measured at two time points only, the analysis of variance (ANOVA) and covariances (ANCOVA) continue to be concerned with the mean level change and requires that all individuals must be studied at the same fixed time points, which may not be optimal in all research studies (Willett, 1988). When there are more than two waves of data, repeated-measures MANOVA (R-MANOVA) also referred to as trend analysis change is construed as a linear trend over the multiple time points and group differences are examined with respect to the magnitude of the trend relative to the variance within each group. However, individual differences in rates of change over time are considered as error by this approach. Additionally, these methods allow one to incorporate discrete and categorical but not continuous predictors of growth in addition to expecting equidistant spacing of time points for all subjects. Finally, in a recent study, Curran & Muthén (1999) found that ANCOVA models were less efficient (required nearly 30% more subjects) than growth curve models in detecting small effect sizes (at power = .80).

On one side, a constant battle of direct and indirect attacks continued (e.g. Cronbach & Furby, 1970; Harris, 1963) primarily on two main issues (a) the reliability or the unreliability of the difference score and its inverse relationship to the correlation between Time 2 and Time 1 and (b) the correlation between the difference score and Time 1 scores and its implication for
using the difference score to study correlates of change. On the other side, researchers disregarded the arguments and warnings and either continued to use change scores or started working on other alternatives. None of these alternatives seemed promising to study change in longitudinal data (Rogosa, 1988; Rogosa & Willett, 1983; Rogosa & Willett, 1985a; Willett, 1988).

The next section of the chapter summarizes the contemporary research tools and techniques developed to measure change and growth with longitudinal data. Primarily, two popular methods, autoregressive models and growth curve models, are discussed. Relevant variations of these models to the area of family-based preventive intervention research are briefly described in a tabular form.

*Contemporary Research Tools and Techniques to Measure Change*

In the 1970s, with the surge of Structural Equation Modeling software, one of the most popular methods used to model change in longitudinal panel designs was the *autoregressive model* also known as the *fixed effects Markov simplex model* (Alwin, 1988; Jöreskog, 1979a; McArdle & Aber, 1990; Rogosa, 1979; Schaie & Hertzog, 1985). Its earliest development can be traced to the work of Guttman (1954). However, his work was further developed by Anderson (1960), Humphreys (1960), Heise (1969) and Jöreskog (1979b). In this approach, latent variables for each occasion are modeled at each time of measurement according to the flow of influence from earlier to later points in time. Figure 1 is an example of a first-order autoregressive model. Here, a single latent variable at Time 2 is modeled as being determined by the same latent variable as Time 1 and so on. Thus, the latter measures have increasingly lower correlations with earlier time points.
Change in this approach is described as an additive function influenced by the immediately proximal time point in addition to a random disturbance. This is why it is called “autoregressive” meaning that each time point measure is regressed onto the same measure as the point prior to it. Thus, variables assessed at time points earlier in the chain have no direct relation on the current value. This method is far superior to ordinary correlation or regression methods mainly due to its ability to model specific error structures and regression relationships between the disturbance terms. Thus, estimates of stability and mutual influence between variables are corrected for random measurement error, and sources of systematic error can be specified and modeled.

Several limitations, however, have been noted regarding this technique (Hertzog & Nesselroade, 2003). First, many critics have stated that the autoregressive models indirectly as opposed to explicitly model the manifestation of change (Rogosa, 1979, 1987). Also, some researchers (Rogosa, 1980) believe that this modeling technique does not identify a cause-effect relationship. Hertzog & Nesselroade (2003) have argued that autoregressive models assume
lower correlations of latent variables with time which could be problematic. These researchers have also argued that in studies where the first point of measurement is not the point of inception, this technique might be problematic. Finally, the form or nature of change over time (e.g., linear, quadratic, cubic) is not considered in this approach.

An alternative approach introduced by McArdle & Nesselroade (1994) started gaining momentum and has become one of the most popular methods for studying change in longitudinal designs. This approach has been referred to in myriad ways, for instance latent change models, latent curve analysis, latent growth models, random coefficient models, etc. In this chapter, this approach shall be referred to as growth curve models (GCM). The growth curve models have been widely used in educational research (Anstey, Hofer, & Luszcz, 2003; Campbell, Pungello, Ramey, Miller-Johnson, & Burchinal, 2001; Kaplan, 2002; Rescorla & Rosenthal, 2004; Zhu & Erbaugh, 1997), biometrics (Albert & Shih, 2003; Cheng & Kuk, 2002; Mikulich, Zerbe, Jones, & Crowley, 1999; Scharfstein & Irizarry, 2003; Weerahandi & Berger, 1999), psychometrics (McArdle & Epstein, 1987; Rogosa & Willett, 1985b; Zimmerman & Williams, 1982b), as well as evaluation research (Aber, Brown, & Jones, 2003; Ashery et al., 1998; Hansen, 1992; Hser, Shen, Chou, Messer, & Anglin, 2001; R. Spoth & Redmond, 2003; Spoth et al., in press). A more detailed overview of this technique can be found in Browne & Du Toit (1989), McArdle, (1986, 1988, 1989, 1991), McArdle & Epstein (1987), Meredith & Tisak (1984, 1990), Muthén, (1994, 1991), Muthén & Curran (1997) and Willett & Sayer (1994). A short explanation of this model is given in the following paragraph.

When using growth-curve models, change is conceptualized in a different way from the autoregressive approach. These models do specify the occasion-specific factors, but they do not use an autoregressive approach to estimate stability coefficients for each occasion. Additionally,
difference scores are not calculated and are not directly factored. In its place, higher order latent variables as well as initial level and change, are specified. Figure 2 represents a basic growth curve model for a single observed variable, measured at three different time points.

Figure 2. A linear growth curve model centered on initial status

The Level latent variable represents the initial point of measurement and the Shape latent variable captures individual differences in rates of change over time. A repeated measure of the observed variable is modeled to estimate a single underlying growth trajectory for each person across all the time points. This allows one to compute an average intercept and an average slope (also known as fixed effects or *interindividual differences*) as well as the variability around these averages (also known as random effects or *intraindividual changes*). This makes the growth-curve models much more powerful in detecting interindividual differences and intraindividual change than other techniques (Curran & Muthén, 1999).
The main advantage of this approach is that change is characterized as a factor or a latent variable, thus allowing one to estimate the variance of the latent change factor as a parameter. Means of the observed variables are modeled as being determined by the latent variables, Level and Change. At each occasion, the variable has components of the initial level, growth level, for example, change from initial level and measurement error. The functional form of change, also referred to as the basis function, is defined by values of loadings for the observed variables on the Shape factor. The different types of basis functions will be discussed later in this chapter.

In Figure 2, the scaling of the basis function shows a linear growth or decline. This is evident by the equal interval of loadings on the Shape factor over the different measurements of time. An important assumption in this approach is that individuals can vary in the amount and rate of developmental change, but not in the functional form of development. The means of the initial status and growth rate factors reflect the group parameter values of the intercept and slope of the developmental trajectory. The variances of the initial status and growth rate factors represent the individual variability of each subject around the group parameters. Finally, the variances in the growth factors can be modeled as a function of other explanatory variables (covariates, predictors) to understand better observed individual variability in rates of change over time. Despite the aforementioned advantages of this methodological approach, the power to detect predictors of growth parameters has received only limited attention in the methodological and empirical literature (e.g. Curran & Muthén, 1999; Muthén & Curran, 1997; Raudenbush & Bryk, 2002).

Several variations and combinations of the latent variable models have been introduced in the field. Some of the most important ones related to this chapter are multilevel models for modeling nested or hierarchical data (Hierarchical Linear Models, (Raudenbush & Bryk, 2002),
combined autoregressive latent curve model (Curran & Bollen, 2001)), latent growth mixture modeling (Hedeker & Gibbons, 1997; Muthén et al., 2000; Muthén & Shedden, 1999) as well as Nagin’s group-based trajectory models (Nagin, 1999; Nagin & Tremblay, 2001). A brief description of these variant models is given in Table 1. There are several advantages of using growth curve models for studying interindividual differences in intraindividual change especially with research related to family-based preventive intervention research which is described below.

Advantages of Growth Curve Models for Preventive Intervention Research

Kellam and his colleagues (1991) remarked that not all interventions work for all people. Growth curve models (GCM) allow researchers to identify those individuals who benefit the most from the interventions and those who do not. Since growth curve models allow for the estimation of individual differences in change over time, researchers can examine the differential response to treatment to identify factors associated with stronger or weaker responsiveness to treatment.

Table 1. Variations and combinations of latent variable models relevant for studying interindividual differences in intraindividual change.

<table>
<thead>
<tr>
<th>Model</th>
<th>Utility</th>
<th>Empirical Examples</th>
</tr>
</thead>
<tbody>
<tr>
<td>Multilevel models</td>
<td>Used for modeling nested or hierarchical data.</td>
<td>Norton, Bieler, Ennett, &amp; Zarkin (1996)</td>
</tr>
<tr>
<td>(Hierarchical Linear Modeling, (Raudenbush &amp; Bryk, 2002)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Method</td>
<td>Description</td>
<td>References</td>
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<tr>
<td>--------------------------------------------</td>
<td>-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------</td>
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</tr>
<tr>
<td>Combined Autoregressive Latent curve models (Curran &amp; Bollen, 2001)</td>
<td>Used for studying conditions in which both continuous underlying trajectories as well as time-specific influences across constructs are of interest.</td>
<td>Stoolmiller (1994)</td>
</tr>
<tr>
<td>Latent growth mixture modeling (Muthén et al., 2000)</td>
<td>This model assumes that the growth in the population is heterogeneous and discrete and each latent class has different patterns of change.</td>
<td>Li, Duncan &amp; Hops (2001); Colder, Campbell, Ruel, Richardson, Flay (2002)</td>
</tr>
<tr>
<td>Group-based Trajectory model (Nagin, 1999; Nagin &amp; Tremblay, 2001)</td>
<td>This approach identifies distinctive clusters of individual trajectories. This model allows researchers to identify the shape of the trajectory for each distinct cluster and the proportion of the population that constitutes each cluster.</td>
<td>Nagin &amp; Tremblay (1999)</td>
</tr>
</tbody>
</table>

Two recent papers by Muthén and Curran (1999; 1997) comparing growth curve models and ANCOVA models concluded that growth curve models had greater power to detect a treatment effect compared to fixed effect models such as ANCOVA. They also noted that the GCM can be easily modified to allow for computation of specific estimates of power under a variety of assumptions and conditions such as number of subjects in control group, number of subjects in experimental group, and number of waves of data.

Growth curve models can also be used with data on multiple members of the family. For instance, Kurdek (2003) applied the growth curve analysis to dyadic data to examine the
relationship quality changes in married couples over the course of the marital career as well as explored if the same patterns of growth held for husbands and wives. These models have also proved to be suitable for dealing with missing and incomplete data (McArdle & Hamagami, 1991) which in turn increases the power of the study.

In addition to the above advantages, growth curve models can also include mediators to understand the specific mechanisms of change. These models do not rely solely on linear growth functions. Several different functions of growth such as quadratic and cubic functions of growth can be modeled. These models can then be analyzed using SEM based software that is user-friendly and easy to obtain. Finally, growth curve models can be evaluated through several fit indices that SEM based software generally report.

In summary, family-based preventive intervention research relies on methods that analyze change to measure the efficacy of intervention programs. Heated debates in the methodological field historically stagnated the field from analyzing and measuring change. Despite this debate, researchers continued to find alternative methods to analyze change. Some of the latest methods such as autoregressive models and growth curve models seem promising. Several variations have been suggested that have further advanced the field. However, several methodological issues still need to be addressed. In the next section of this chapter some of the major methodological issues and their relationship to statistical power and best practices for estimating interindividual differences in intraindividual change typically faced in the family-based preventive intervention context will be discussed.
Major Methodological issues and Their relationship to statistical power: best practices for estimating individual trajectories in family Preventive Intervention Research

Preventive intervention programs designed for families target the risk and protective factors associated with the family context. Several researchers have successfully demonstrated the importance of the family as an intervention context by showing that enhancing families’ protective factors can have a positive influence on other members of the family. Yet relatively few family-based preventive intervention studies have been subjected to rigorous efficacy tests and fewer have been replicated (but see Strengthening Families Program adapted for universal audience, Redmond et al., 1999). This protracted development of the field is chiefly due to some major methodological issues that affect the statistical power to detect interindividual differences in intraindividual changes resulting from family-based preventive interventions.

Some of these methodological issues affecting power while estimating individual trajectories in family-centered preventive intervention research will be discussed in this chapter. These issues have been addressed as design issues and analytical issues for clarity and ease of understanding. However, throughout the rest of this chapter, methodological and substantive developments that have informed each other and advanced this area of research will also be emphasized.

Design Issues

Several issues about research design are responsible for the lower powered preventive intervention studies. These issues range from research design, sampling, choice of measures,
determining the features of the repeated measures and the inclusion of predictors and covariates of change.

*Research design*

The more rigorous the research design the more confident a researcher can be about the validity of the intervention’s effectiveness (Rossi, Lipsey, & Freeman, 2004). The fundamental purpose of experimental designs and their quasi-experimental approximations is to maximize the chances of making causal inferences (Shadish, Cook, & Campbell, 2002). The importance of establishing such a cause-effect relationship in the field of intervention research is to determine with a degree of certainty whether the intervention program has worked or not. Several limitations in experimental and quasi-experimental research designs have been found. Additionally, how these limitations affect power to detect significant change are presented. Some of these issues along with improvements and developments relevant to this chapter are summarized below.

*Random assignment*, which is the trademark of true experimental designs, is often not practical in social intervention studies. Despite their ability to deliver the most valid and internally consistent conclusions about the effects of interventions, these designs can very rarely be used in social and behavioral sciences because of ethical, legal and practical considerations. Even if randomization is possible, these experimental designs consume a lot of time, energy, money, expertise and co-operation of the participants of the study. Even when successful randomization is possible (e.g. Gueron, 1997; Braucht & Reichardt, 1993), there is the practical difficulty of maintaining subjects participation until the end of the intervention program and data collection.
In some cases, there is a dearth of participants to be assigned to the control groups (Murray, Moskowitz, & Dent, 1996). Thus, although experimental designs have high internal validity, they show low external validity especially in behavioral and social sciences (Campbell & Stanley, 1966; Cook & Campbell, 1979). Due to these limitations, intervention researchers often use quasi-experimental designs with non-equivalent control groups either by intent or by default due to attrition (Chalk & King, 1998; Speer & Newman, 1996), thus reducing statistical power. To deal with the apparent bias in the nonequivalent groups, researchers have relied upon structural equation models using covariances and means to control for the initial differences between the intervention and control group to better approximate results from random assignment (Bentler, 1991). Yet, it is clear that experimental designs are far more robust in detecting change than quasi-experimental designs (Maxwell, 1994; Maxwell, Cole, Arvey, & Salas, 1991).

Because of the differences in the ability to detect power, many researchers (Lipsey & Wilson, 1993; Venter, Maxwell, & Bolig, 2002) have found that randomized and nonrandomized designs in the same substantive intervention area show quite divergent results. At the same time, other researchers who conducted meta-analyses (Heinsman & Shadish, 1996; Shadish & Ragsdale, 1996) found that although there were discrepancies in the results from the two designs, when the studies were equated for such features as amount of treatment to the control group, pretest effect size, selection and attrition, these discrepancies diminished. Thus, it is reassuring to know that when effective statistical control is exercised, nonrandomized designs can produce comparable results to randomized designs (Aiken, West, Schwalm, Carroll, & Hsiung, 1998; Reynolds & Temple, 1995).
Other researchers, who have scrutinized randomized but nonequivalent treatment and control groups, have found that unbalanced designs might in fact be cost-effective and have more statistical power than a balanced design (Allison, Allison, Faith, Paultre, & Pi-Sunyer, 1997; Cochran, 1963; Hsu, 1994; Liu, 2002; Nam, 1973; Yang, Sackett, & Arvery, 1996).

Intervention researchers have also realized that to maintain lower rates of attrition and collect quality data from the control and intervention groups, less burden needs to be placed on the participants regarding testing and completing long and time-consuming test instruments. A unique form of design known as planned missingness designs are of considerable importance for studying change (McArdle, 1994; McArdle & Hamagami, 1991). In this design not all measures need to be given to all participants at all occasions of measurement. Instead, measures are selectively given to individuals in a planned way so as to be able to estimate all parameters of interest. McArdle and Hamagami (1991, 2001) successfully applied this design using growth curve modeling procedures and methods borrowed from the research on missing data analysis to model change to produce a cost-effective design as well as increase the statistical power of the study to detect change (Duncan, Duncan, & Hops, 1996; Graham et al., 1997; Graham et al., 1996).

Another issue arises from the assumption that all intervention programs at different sites are equivalent without considering that there could be large variations in the implementation of the intervention program. For instance, some intervention programs might be inconsistently delivered, some participants may interact or react to the intervention differently and sometimes the intervention itself might have different effects on the participants. When assessing the program effects, ordinary trend analysis could not consider these variations due to their restricted ability of examining mean level effects of the program and thus are less powerful in detecting
interindividual difference and intraindividual change. However increasingly now, intervention researchers are including in their designs explanatory variables (predictors, mediators, moderators, covariates) in order to understand the variability in the outcome. At the same time, innovative statistical analyses such as the growth curve models are being used that have the capability of examining not only interindividual differences but also intraindividual changes caused by the intervention program. These models, as noted before, are more powerful compared to other statistical techniques in detecting change (Curran & Muthen, 1999).

**Sampling**

Many researchers do not have an overt rationale for the sample size that is included in their research designs. Although most know that the bigger the sample, the higher the power to detect change, many researchers in the past have followed the rule of the thumb: “Use as many subjects as you can possibly get and can afford to get” (Olejnik, 1984). In order to determine the sample size for family-centered preventive intervention research, factors such as sample size determination based on the significance level, statistical power, analysis used and effect size need to be considered. Issues regarding sample size in unbalanced designs and sample size determination for studies dealing with families are also noted in this section.

*Determining sample size: Relationship to significance level, statistical power, analysis and effect size.*

sample size for an experimental or a quasi-experimental research study, four factors must be taken into consideration. These are: (1) criterion used for statistical significance; (2) level of statistical power; (3) statistical analysis method; and (4) the effect size.

*Criterion chosen for statistical significance.* The level of significance chosen by the researcher is the probability that a Type I error will be made (which means that a difference was detected between the intervention and control groups when there really was none or that the intervention affected change in the treatment group when it actually did not). Generally, researchers consider the Type I error as a serious mistake and therefore want to minimize the probability of its occurrence. If other factors such as power, analyses strategy used, and effect size is constant, sample size would be inversely proportional to the significance levels. Thus a large sample size will be required if the probability of a Type I error is to be minimized. Most studies in the preventive intervention research have strictly adhered to a 5% criterion for significance, which is questionable. In situations, when an increased probability of a Type I error is acceptable, then a smaller sample size may be sufficient.

*Level of Statistical Power.* The second factor that affects the number of participants in an intervention study is statistical power. The probability of not finding an intervention effect or relationship between variables when there really is one is referred to as statistical power. Without sufficient power, conducting an intervention study would be an inefficient use of time and resources. There are no rigid guidelines for defining adequate statistical power. Most social scientists, however, consider statistical power ranging from .80 and above as acceptable (Cohen, 1988, 1994; Maxwell & Delaney, 2004; Murphy & Myors, 2004). When the other three factors, namely significance level, statistical analysis and effect size are constant, an increase in the level
of statistical power will mean that a larger sample size is needed. Similarly, a smaller sample size would result in lower statistical power.

Data Analysis Procedure. Sample size is also dependent on the statistical analysis procedure that a researcher chooses to use. Simple analysis with fewer hypotheses to test requires a smaller sample size. Similarly, studies that collect data both pretreatment and post-treatment require fewer subjects compared to studies that only look at post-treatment. Also, studies that collect more information on explanatory variables from the subjects require a smaller sample size (Olejnik, 1984) to achieve the same statistical power.

Effect Size. A fourth consideration that affects the decision about sample size depends on the degree to which the null hypothesis is false. When all the three factors described above are constant, the sample size is inversely proportional to the falseness of the null hypothesis. For instance, in preventive intervention studies when researchers are comparing two population means, for example experimental and control groups, the null hypothesis means the two population means are equal. If there were a very large difference in the two group means, fewer subjects would be needed to prove that. If there were a very small difference in the group, however, a larger sample would be needed to reject the null hypothesis. Thus, when planning a study and deciding on a sample size, researchers must specify the minimal relationship or difference in the two population means that would be important to detect. Cohen (1988) suggests mean differences (i.e. changes in the longitudinal context) of .2, .5 and .8 standard deviations as being small, medium and large effects which continue to be widely accepted in the field.

Several formulas and software programs are available to calculate power, effect size and sample size (Cohen, 1988, 1992; MacKinnon & Lockwood, 2003; Murphy & Myors, 2004).
Intervention researchers often have to balance optimum sample size and the cost for recruiting and retaining study participants. Cost-effectiveness often wins the battle, and most interventions have much smaller sample sizes than is optimal. Preventive intervention researchers should strive to base their decisions regarding power and sample size determination on the four factors mentioned earlier instead of chance.

*In the absence of balanced designs*

Often even after recruiting equal participants in the control and experimental groups, due to non-compliance, attrition and other reasons for missing data, unbalanced designs with unequal sample sizes in the control and experimental groups are the reality of intervention research (Rossi et al., 2004). Researchers continue to strive harder and harder to achieve a balanced design. This preference for a balanced design is largely due to the well-known fact that a balanced design produces maximum statistical power for a total sample size because group membership is uncorrelated with the other predictors (Maxwell & Delaney, 2004). However, several studies have shown that in fact unbalanced designs might be more cost-effective than balanced designs. An unbalanced design may increase the total sample size by allocating more subjects in the control group than in the treatment group and attain a higher power than a balanced design in a two-group design (Allison et al., 1997; Cochran, 1963; Hsu, 1994; Nam, 1973; Yang et al., 1996).

A recent paper by Liu (2002) examined multilevel models and derived the best possible sample sizes for the treatment and control groups between balanced and unbalanced designs when the cost per sampling unit was lowest and statistical power reasonably high. Finally, Curran & Muthén (1999) modified the growth curve model to compute specific power estimates
in a variety of conditions such as the number of participants in the control group, number of participants in treatment group and total number of waves.

**Family and Sampling**

It is imperative to consider the uniqueness of families when designing the study and determining the sample size for a family-based intervention program. The dynamic nature of the family structure and the rapidity with which membership to a family can change involves unique challenges for family-based preventive intervention researchers. For instance, when divorce occurs in a family over the course of the study, some family members would no longer be available. Further complicating the issue, when remarriage occurs within this time frame a previous family member may be replaced by a new member. The researcher then has to decide during the design of the study, how he or she would deal with this situation. Some researchers (Collins & Shanahan, 1998) have suggested that the sample should be limited to family who fit within certain characteristics (such as intact families, two-parent families, etc.). This would ensure a baseline observation where all families measured are homogeneous, thus reducing unexplained variance and potentially increasing statistical power and internal validity (Hansen & Collins, 1994).

However, this kind of sampling would also have disadvantages such as reduced ability to generalize results and consequent results in the external validity (Hansen & Collins, 1994). Researchers have also suggested that resources should be devoted to finding and collecting as much data as possible from those who drop out of the study. This is especially helpful with recent advances in correcting missing data and increasing the statistical power of the study (Graham et al., 1997; Graham et al., 1996; Schafer & Graham, 2002). These missing data
techniques will be discussed later in the chapter. However, it is important to note that the effects of the type and the extent of missing data on power are largely unknown (Davey, Savla, & Luo, 2004).

**Choice of Measures**

Another factor influencing statistical power is the choice of measures. Several researchers (Cook & Campbell, 1979; Maxwell, 1994; Krathwohl, 1985) have noted the importance of using reliable, valid and sensitive measures in order to arrive at scientifically meaningful conclusions as well as give enough rigor to the research design to detect change. However a relative lack of validated outcome measures to measure family-based preventive intervention outcomes continues to cause difficulties in estimating models (Chalk & King, 1998). There are three measurement properties that evaluation researchers should look for in the measures that one uses for evaluation of the intervention programs. These are reliability, validity, and sensitivity of a measure.

**Reliability.** The reliability of a measure is the extent to which the measure generates the same results even when used repeatedly to measure the same construct (Pedhazur & Schmelkin, 1991). Measurement error is the result of the variation in those results. Measurement error in the independent variables leads to a bias in estimates of the regression coefficients. Since the measures used are the only way of assessing specific dimensions, unreliability of a measure results in the dilution of real changes or differences (Type II error) thus reducing statistical power (Pedhazur & Schmelkin, 1991). In intervention research using longitudinal data (more than two waves) high test-retest reliability is an important consideration since you want the instruments to measure the same construct even if the measurements are not closely spaced.
However, some researchers have also found that collecting data on the same instrument in repeated waves results in increased reliability and power, regardless of the reliability of the measure (Maxwell, 1994; Nicewander & Price, 1978, 1983; Sutcliffe, 1980).

**Validity.** Validity of a measure signifies the extent to which the instrument measures what it is supposed to measure (Pedhazur & Schmelkin, 1991). It is difficult to measure whether a particular instrument is valid for the particular program goals or characteristics of the sample. By using multiple measures of the outcome variable, researchers can safeguard against the possibility that any one of those measures does not tap into the actual outcome of interest. Similarly, a recent study done by Little, Lindenberg, & Nesselroade (1999) shows that a high level of homogeneity in the manifest variables could limit the validity of the outcome. These authors suggest that optimal level of indicator correlations should be moderate rather than high in order to have sufficient power to detect change.

**Sensitivity.** The primary role of outcome measures is to detect changes or differences in outcomes that represent true program effects. This is another salient quality to look for in outcome measures especially when one wants to detect individual differences on a construct of interest as well as ensure sensitivity to change over time (Lambert & Hill, 1998; Vermeersch, Lambert & Burlingame, 2000). Although evaluation researchers recognize that outcome measures must be sensitive to change, there has been surprisingly little work done to identify sensitive measures or even how one can enhance the sensitivity of measures (but see Lipsey, 1990; Vermeersch, Lambert & Burlingame, 2000; Stewart & Archbold, 1992; 1993). This is important because of its potential to increase statistical power for a given sample size.

What variables one chooses and how well they represent the latent variables is critical to the design of an intervention study that measures change. Good measures result in lower
measurement error and higher variance that can be explained (Pedhazur & Schmelkin, 1991) thus improving the statistical power to detect change.

**Nature of outcome variables**

There has been much methodological development in the analysis of longitudinal data for analyzing change. The nature of the outcome variables drive the choice of statistical modeling techniques one could use. A variety of techniques are available today to test longitudinal models depending on the nature of outcome variables used for the study. For continuous variables, techniques such as autoregressive structural equation modeling, growth curve modeling and pooled time series are some of the options (see Collins & Sayer, 2001). For repeated categorical data, Generalized Estimating Equation models (GEE, Brown & Liao, 1999; Diggle et al., 1994) have been applied to intervention research (Chou, Liu & Chu, 2002). Also, Collins and her colleagues developed a procedure called latent transition analysis that generates stage-sequential models of individual categorical data (Collins & Wugalter, 1992) and have used these in intervention research (e.g. Hyatt & Collins, 2000). How the nature of outcome variables could affect statistical power, however, is still unknown.

**Number of waves of data collected**

Earlier in this chapter several disadvantages of using two-wave designs for studying change were discussed. It is important to note that in addition to those drawbacks, to identify the trajectory of change especially by using growth curve models, it is necessary to have more than two time points of data. Some researchers have also suggested that when studying change, researchers should consider it more important to increase the number of waves of data instead of
the number of subjects in the study (Boker & Nesselroade, 2002; McArdle & Woodcock, 1997; Nesselroade, 1991). According to Collins & Shanahan (1998) the key is to measure more frequently when the growth or change is most rapid. Similarly, a more slowly moving or an obviously linear growth process could be measured with fewer observations spaced further apart. At the same time one should be cautious of the potential for introducing measurement artifacts when data are collected too frequently (Wohlwill, 1973; Baltes, Reese & Nesselroade, 1988). This would clearly reduce power to detect true change (Maxwell, 1994; Maxwell et al., 1991; Maxwell & Delaney, 2004). To summarize, the number of waves and the spacing of observations play an important role in determining the power of the study to detect interindividual differences and intraindividual change.

Statistical analysis for modeling growth and change has different requirements and assumptions about the spacing of observations in a study. Methods such as repeated measures analysis of variance require that observations be evenly spaced and conducted at the same time for all individuals. Latent growth curve modeling (Willett & Sayer, 1994) and latent transition analysis (Collins & Wugalter, 1992), require that observations take place at the same time for all individuals, but they need not be evenly spaced. On the other hand, approaches based on hierarchical linear modeling (Raudenbush & Bryk, 2002) allow for variation in both timing and spacing of observations.

When studying change, one of the most important decisions a researcher has to make, is whether to have a longitudinal or a cross-sectional design. Schaie (1965) pointed out in a series of articles that cross-sectional studies were confounded by cohort and age. A researcher could examine the interindividual differences but cannot examine intraindividual differences. On the
other hand, longitudinal studies are advantageous since it allows one to examine intraindividual change over time. However, in longitudinal designs, age and time are confounded.

In an attempt to separate age, time and cohort effects, Schaie and Baltes (1975) later proposed several alternative designs. One of the most widely used and well-known designs among these is the cohort-sequential design also known as accelerated longitudinal design (also see Nesselroade & Baltes, 1979). This design involves a series of longitudinal designs with a different cohort of individuals. With such a design it is possible to determine whether the results obtained are replicated in other cohorts or not. If the effect is replicated across the cohorts on whom the data is collected at a specific time rather that at specific ages, it shows that the effect is due to historical or sociological factors affecting all cohorts at the same time. Given the constraints of time and limited financial expenses for a true longitudinal design, cohort-sequential designs are a good choice. These designs have also been found to be robust in detecting statistical power.

However concerns regarding their validity and their ability to approximate the true longitudinal growth curve have been raised (McArdle, 1991; Raudenbush & Chan, 1992, 1993; Tonry, Ohlin, & Farrington, 1991). Several researchers (Anderson, 1993; Duncan, Duncan, Strycker, Li & Alpert, 1999) have demonstrated the ability of the accelerated longitudinal design to recover the true longitudinal curve and lend support and validity to its use by using latent growth curve modeling techniques. However, there are still several questions that are unanswered; for instance, what is the optimal number of time points needed per subject, what is the optimal number of points of overlap across adjacent cohorts, and what is the optimal number of subjects required per cohort (Raudenbush & Chan, 1992; Mehta & West, 2000) in order to achieve the greatest statistical power.
Analytical Issues

Even though researchers try hard to employ the best designs for intervention studies, some of the analytical issues have an enormous impact on the power to detect change. In this section some of these issues will be discussed in the light of intervention research. These issues range from the shape of trajectory of change evident in the data, ignoring the interdependence of data, and missing data due to attrition and non-compliance.

Shape of trajectory

Due to the richness of longitudinal data with more than two time points, researchers are now able to understand the nature of the growth function. With the use of growth curve modeling techniques, the “true” growth trajectory of each individual can be represented by an algebraic function of time. Many possible mathematical functions are available to represent true individual change. There are some that depend linearly on time and those that do not. Willet and Sayer (1994) advises researchers to inspect each person’s empirical growth curve by plotting his or her observed status against time to determine the function of growth over time. The most commonly used growth curve function is linear followed by quadratic functions. Because non-linear polynomial models of growth add additional parameters, they can generally be expected to reduce statistical power, all else being equal; however, none have empirically tested how the shape of trajectory affects the detection of statistical power.
In the field of preventive intervention, most data structures are hierarchical in nature. Individuals who are sampled in a cluster will tend to be more alike in their responses than individuals who are sampled independently. For instance, siblings come from families, families come from neighborhoods, and children are part of classrooms within schools. There is relatively lower dependence among individuals from the same-school or classroom, but a much higher dependence among family members. This dependence among observations in the data is known to inflate Type I error rates if it is included in the statistical analyses (Barcikowski, 1981) as well as produce biased standard errors affecting statistical power (Norton et al., 1996). The amount of inflation in a Type I error rate is a function of the degree of dependence (known as the intraclass or intracluster correlation (ICC)) between the individuals in the clusters and the size of clusters. The larger the intraclass correlation, the larger is the distortion in parameter estimation that results from the cluster-level effect (Heck, 2001; Norton et al., 1996).

Several researchers have pointed out the importance of understanding and correctly analyzing multilevel or hierarchical data (Raudenbush & Bryk, 2002; Willett & Sayer, 1994). Some researchers have disregarded these warnings and have disaggregated the higher order variables to the individual level, therefore violating the assumption of independence of observations since the individuals in the data set might have the same value for particular variables. For instance, children from the same classroom would have the same value on the variables of class (Raudenbush & Bryk, 2002). Another alternative approach that is commonly used is to aggregate individual-level variables to a higher level and to analyze the higher order variables. An example would be aggregating student characteristics over classes and doing a class analysis weighted by class size (Raudenbush & Bryk, 2002). However, this would result in
a loss of data to detect within-group variation in change (Raudenbush & Bryk, 2002). Additionally, the analysis at this level can increase the Type II error rate because with limited sample size, there is statistical power to detect only large differences. In traditional linear analysis which assumes independence of observations, the variations in clusters get covered into the error term causing correlations between the errors.

Fortunately, advances in quantitative methods have permitted prevention researchers to test such nested data structures using hierarchical linear models (Bryk, Raudenbush & Congdon, 1996; Raudenbush & Bryk, 2002). These models, which are also known as mixed effects, multilevel, and cluster-specific models, have been described in the statistical, psychological, educational, biological, and epidemiological literature (see Raudenbush & Bryk, 2002).

Hierarchical linear models account for the dependence among observations in the model and estimates consistent standard errors in the presence of within-cluster correlation. Furthermore, growth curve models for repeated measures can also be estimated. Researchers can test several different levels of analysis. At one level they can tap the context of the family. At a second level, researchers could also include information from multiple family members. At a third within-persons level, the repeated measures could be used to determine the function of growth.

However, multilevel SEM analyses with small numbers of clusters result in underidentified models. Heck (2001) provided researchers with a formula that calculates the intra-class correlation to determine whether SEM can be used to analyze multilevel data. When the intraclass correlation is less than 0.5, indicating lower significance between the variability of clusters, Heck suggested that a nonmultilevel SEM provides correct estimates of the parameters and standard errors.
Few researchers have used multilevel modeling approaches with hierarchical data. Hedekar, Gibbons, & Flay (1994) used a random-effects regression model to analyze data on smoking prevention in students clustered in classrooms and schools. Norton et al. (1996) on the other hand used generalized estimating equations (GEE) a hierarchical linear modeling technique to assess the effectiveness of a school-based substance abuse prevention program called Project DARE (Drug Abuse Resistance Education). In their analysis they used GEE with continuous as well as binary response data. Redmond and his colleagues (2004) used multilevel analyses in which community-level SES effects on child susceptibility were simultaneously modeled with the family-level data describing the relationships among family sociodemographic factors and parents perceptions.

Several other researchers (e.g. Davila, Karney & Bradbury, 1999; Karney & Bradbury, 1997; Kurdek, 2003; Huston, et al., 2001) have used growth curve models from a hierarchical linear modeling approach to assess change in marital quality of couples over time. Finally, Murray and Blitstein (2003) showed in their paper that the use of two methods, namely modeling time in the analysis and making regression adjustments for covariates, could reduce the impact of intraclass correlation in group-randomized designs and increase statistical power.

**Missing data**

In any research, cross-sectional or longitudinal, missing data is inherently problematic. In family-based preventive intervention programs, data could be missing either due to subject attrition or due to non-compliance. In either case, missing data is one of the major reasons for having low power to detect change. Several methodological advancements have been made to accommodate for these missing data problems, these will be discussed below.
Missing data due to subject attrition

Preventive intervention researchers have found three missingness mechanisms (Little, 1995; Little & Rubin, 1989; Rubin, 1976) in family-based preventive intervention program. Attrition in preventive intervention programs could be due to completely random conditions (MCAR) such as transportation problems to the location of the intervention program, or it could be because of other random conditions related to the individual’s pre-dropout responses or other covariates (MAR) like poor reading skills, lower education etc. More serious, however, is the dropout due to non-random reasons (MNAR) for instance higher family risk or substance abuse recorded for the participant.

When the attrition is due to random reasons (MCAR), where every participant has an equal chance of dropping out, confidence intervals could be biased in the positive direction and lowered statistical power would be achieved due to a reduced sample size. When the attrition is due to MAR, however, analysis of complete cases will result in biased parameter estimates as well as inefficient confidence intervals, along with loss in statistical power.

A variety of statistical techniques for dealing with missing or incomplete data for MCAR and MAR data are readily available to researchers. Out of the various techniques available, full-information maximum likelihood (FIML, Allison, 1987; Arbuckle, 1997; Muthén et al., 1987) and multiple imputation (MI, Little & Rubin, 1995; Schafer, 1997) are considered to be most effective for the analysis of missing data (cf. Enders & Bandalos, 2001; Schafer & Graham, 2002).

Attrition due to non-random reasons (MNAR) can affect both internal as well as external validity of the results. Internal validity of the study is affected particularly when the attrition is
different for treatment and control groups (Collins & Sayer, 2000). Additionally, there is no statistical “fix” that will remedy the problem of MNAR data (Schafer & Graham, 2002). The only procedural measure one could use is to collect data on predictors of missingness and to include them in the model (Schafer & Graham, 2002). For instance, Spoth, Goldberg and Redmond (1999) used discrete-time survival analysis to examine whether family risk factors predicted attrition in a prevention program.

Attrition in preventive intervention research is more complicated when families are the focus of the study. Several family-based intervention studies (Spoth & Molgaard, 1993; Spoth & Redmond, 1993; Weinberger, Tublin, Ford & Feldman, 1990) have noted that it is not uncommon to have up to two thirds of families decline participation or eventually drop out. Sometimes, one member of the family and at times the whole family could also drop out of the study. The researcher then has to make decisions about whether to include data from the remaining family members or not. Most preventive intervention researchers (e.g. Redmond et al., 1999; Roth et al., 2001; Steele, Forehand, & Armistead, 1997; Windle & Windle, 2001) have removed such families from their analyses. However, this could result in biased and faulty results see (Davey et al., 2001).

Another problem faced by family-focused preventive intervention researchers is the failure to represent high-risk families adequately in their studies because these families refuse to participate and have higher chances of dropping out (Center for Substance Abuse, 1995; Kazdin, 1993; Center for Substance Abuse Prevention, 1995) affecting the ability to generalize study results to the general population.

Development and use of sophisticated software programs are clear evidence of the progress made in the social sciences to deal with missing data or incomplete data due to attrition
and dropout. Yet, several daunting questions are yet to be answered. What is the impact of missing data on the estimates of rates of change in preventive intervention programs? Additionally what is the degree of bias under different rates of missing data and different types of missing data analysis?

**Missing data due to non-compliance.**

Another related issue in preventive intervention is non-compliance or non-adherence to treatments. This is another threat to obtaining power to detect intervention effectiveness in prevention research. Non-compliance in intervention programs occurs when participants do not follow the randomized assignment (Angrist, Imbens, & Rubin, 1996). The most common forms of non-compliance are: (1) Never-takers – Individuals who are assigned to the experimental group and fail to show up and receive treatment, (2) Always-takers – Individuals who are assigned to the control group but who nevertheless find ways to receive the treatment meant for the experimental group, (3) Defiers – Individuals who do the opposite of what they are asked to do.

Since non-compliance by never-takers is among the most common reason for non-compliance, most research is done on this group. Individuals in this group are randomly assigned to the treatment group and agree to participate; however, they fail to show up at the intervention. This is one more cause of missing data in randomized intervention programs (Jo, 2002).

Recent developments in statistical methods have provided researchers with ways to estimate the intervention effects with noncompliance. Depending on the purpose of the intervention and the research question raised, one can decide whether to estimate the overall
intervention effect for the entire sample by using Intent-to-treat analysis (ITT), or to estimate the intervention effect for only those who comply with the treatment using Complier Average Causal Effect (CACE).

Few studies have looked at how non-compliance affects statistical power. Jo (2002) compared the use of these ITT and CACE analysis to determine statistical power depending on compliance, balanced versus unbalanced design, distribution of the outcome variables and different covariates in the model. He found that in general the validity of CACE estimates and its power is challenged. This study only looked at compliers versus non-compliers; however, in the practical world, there can also be partial compliers who choose to attend or receive different amounts of the intervention program exposure.

Another related issue is that intervention programs often suffer from both noncompliance as well as attrition. Yau and Little (2001) simultaneously modeled noncompliance and nonresponse, using ML and EM, assuming no correlation between noncompliance and nonresponse, i.e. assuming MAR. Similarly, Frangakis and Rubin (1999) simultaneously modeled noncompliance and nonresponse using Bayesian estimation methods allowing for possible correlation between noncompliance and nonresponse (MNAR). Both these studies found that statistical power is affected by the type of missingness and the correlation between noncompliance and nonresponse.

Systematic study has not been done regarding the issue of missing data due to attrition or non-compliance on the effect of the power to detect the estimates of change using different analytical procedures (but see Touloumi, Babiker, Kenward, Pocock, & Darbyshire, 2003).
**Evaluating Effects**

At some point in time, some determination still needs to be made regarding the effectiveness of the intervention program, and if so, its magnitude and its meaning within the context of the intervention program goals. At the present time, researchers continue to use statistical significance as the criterion for claiming an effect, despite several arguments regarding this misleading standard (Cohen, 1994; Lipsey, 1999; Prosavac, 1998; Reichardt & Gollob, 1997; Schmidt, 1996). Various alternatives in place of the statistical significance criterion have been proposed, but none has yet been widely adopted. These include reliance on confidence intervals rather than point significance testing (Reichardt & Gollob, 1997), identifying minimal detectable effect (Bloom, 1995) and reporting effect sizes by using the standardized mean difference effect size or odds ratio as a measure (Prosavac, 1998). Recently, Wilkinson and the Task Force on Statistical Inference (1999) recommended that significance tests be accompanied by effect size measures and, ideally, confidence intervals to better inform readers.

Lastly, Gerbing & Anderson (1993), using Monte Carlo techniques, made some suggestions regarding goodness-of-fit indices that researchers could use to evaluate Structural Equation Models such as growth curve models. These authors suggested that apart from reliance on the traditional chi-square and associated $p$ value, one could continue to look at the values of standardized residuals to locate the misspecification and provide an informal index of fit. They also recommend the use of the Hausman Test (Arminger & Schoenberg, 1989) for assessing whether or not the disturbances in the models are independent of the explanatory variables in the model. With regards to the overall assessment of fit, the authors recommend using the noncentrality index (McDonald, 1989). Additionally, RNI (Bentler, 1990; McDonald & Marsh, 1990) and Bollen’s DELTA2 (Bollen, 1989) have been the other best choices for incremental fit.
The assessment of power is more complicated in the domain of structural equation modeling. Unlike some other procedures, a typical structural equation model has several parameters such as the means, intercepts, regression coefficients, variances, covariances and so on. Within a single model, thus, each parameter can be estimated with a different degree of precision. Furthermore, parameters of a model are typically not independent, nor is the statistical power to estimate them. Moreover, researchers are often interested in determining the power to compare two nested models, or the power of an omnibus test of model fit, or the power to reject a trivial model versus a null model.

Several different approaches have been presented in the literature in order to evaluate statistical power with structural equation models. The earliest and most straightforward approach was presented by Satorra & Saris (1985). One begins by estimating a model with the constrained parameters (say that one or more path coefficients or means are equal to zero or some trivial value) using the population covariance matrix given the parameter’s true value (say equivalent to a moderate effect size). The chi-square obtained from fitting this model provides an estimate of the noncentrality parameter (NCP) for those effects. Statistical power is then obtained directly as

$$\text{Power} = 1 - \Pr \left( \chi^2 \leq \text{Crit}, df', NCP \right)$$

Although this approach requires explicit formulation of the alternative hypothesis for each parameter of interest, after 20 years it still remains one of the most useful and widely applied methods for assessing statistical power in structural equation models. Saris & Satorra (1993) subsequently presented an alternative approach that makes use of isopower contours, representing sets of alternative parameter values at which power is constant. While conceptually elegant, it has received relatively little direct application in the literature.
Summary and Conclusion

Family-based preventive interventions have been proven effective in moderating, delaying and often preventing the perplexing and persistent problems in our society today. Issues regarding research designs and analytical strategies used are the chief reason why many of these intervention programs fail to detect significant interindividual differences and intraindividual change in the context of the family. Several of these design and analytical issues have been given much attention; however, there is much work that still needs to be done. Paramount is the need to understand how missing information in family data affects the statistical power to detect change.
CHAPTER 3

STATEMENT OF THE PROBLEM

Based on the previous literature review it is quite evident that design, sampling and analytical issues can affect the statistical power of an evaluation study. Consideration of these issues with regard to missing data suggest the following research questions as an important starting point for this inquiry.

Issue 1: What is the association between type and extent of missing data and statistical power?

It is well known that missing data present a nearly ubiquitous problem in intervention studies. Although it has not been systematically addressed in previous research, it is also expected that missing data will result in some loss of statistical power. As family-based preventive intervention research has evolved, it has grown to rely to a much greater extent on methods that analyze change in order to measure the efficacy of intervention programs. In recent years, application of growth curve models to assess this change has increased in popularity. Growth curve models have been found to be more powerful in detecting treatment effects than ANCOVA models in multi-wave studies (Cole, Maxwell, Arvey, & Salas, 1993; Maxwell, 1994; Maxwell et al., 1991). With complete data, some researchers (Muthén & Curran, 1997; Curran & Muthen, 1999, Raudenbush & Liu, 2000, Raudenbush & Xiao-Feng, 2001) have computed power estimates of growth-curve models under conditions of differing sample sizes and number of waves of data. To date, however, none has looked at how the type of missing data and the extent of missing data affects the power of growth curve model to distinguish longitudinal
changes in treatment and control conditions. Even when missing data has been the focus of bias in parameter estimates (Enders & Bandalos, 2001) or statistical power analysis (Dolan, van der Sluis & Grasman, 2004; Graham, Taylor, & Cumsille, 2001), researchers have considered the effects of small amounts of missing data (2 to 25% of cases having some missing data in the former paper and up to 55% of observed values missing in the latter papers). None has looked at the statistical power across the whole range (for example from very little to extreme amounts of missing data) of the percent of missing data and type of missing data (both MCAR and MAR).

The aim of this first study, then, is to understand how the relationship between statistical power and missing data is influenced by the type of missing data (None vs. MAR or MCAR), amount of missing data (from 5% missing to 95% missing) and across a wide range of sample sizes (N = 100 to N = 1000). There are two ways this information could be helpful to researchers. First of all, when researchers are planning their study and expect some amount of missing data in their study based on previous research or the nature of their sample, for example, it would be beneficial to know how the statistical power of their study would be affected at that particular sample size as a result of different amounts of missing data. Second and most importantly, researchers could actually plan to have specific amounts of missing data (assigned either randomly or in a known systematic fashion) in order to achieve a specific statistical power with the minimum sample size and thus make the maximum use of available resources. Study 1 will address this question.
Issue 2: What factors moderate the association between the type and extent of missing data and statistical power?

Loss of participants and the resulting loss of human and financial resources in longitudinal research are inevitable, but this does not mean that the losses cannot be minimized through appropriate study planning. It seems important to identify ways to economize resources through the application of missing data designs and to make missing data designs more powerful when missing data are anticipated. To date, relatively little is known about factors that have the potential to moderate the association of missing data on statistical power. However, several variables hold considerable promise in this regard.

Higher reliability translates into lower measurement error, and can thus be expected to result in a greater ability to detect significant effects. However, the effects of reliability of an instrument on statistical power in the presence of missing data have not been appraised (Maxwell et al., 1991). Similarly, recent research (Collins et al., 2001; Graham, 2003) has suggested that inclusion of auxiliary variables may in general lead to more accurate estimates with missing data. However, the circumstances where auxiliary variables construe more or less advantage, and their ability to partially compensate for the loss of statistical power due to missing data has not been systematically explored.

Lastly, if there is a way to limit and reduce the testing burden through the application of missing data designs, then these should be identified and exploited. Recent advances in planned missingness (e.g. Curran & Muthén, 1999; Graham, Taylor & Cumsille, 2001) suggest that some incomplete data designs may be more powerful than others. In the context of longitudinal intervention research, how data are missing on different waves is an important consideration, as is whether planned missingness is designed to be random or systematic. Exploration of these
issues in Study 2 will serve to clarify the role of these factors in order to plan research with incomplete data and make the most efficient use of resources.
CHAPTER 4

STUDY 1: ASSOCIATION BETWEEN MISSING DATA AND STATISTICAL POWER

As family-based intervention research has developed, there has been increasing consensus that growth curve models are an important way to analyze change. As an alternative to traditional methods of repeated measures analysis of variance or ANCOVA approaches, growth curve models allow for estimating true change over time, as well as for investigation of interindividual differences in intraindividual change. Under many circumstances, growth curve models are also more powerful statistical techniques than the traditional alternatives mentioned above (cf. Cole et al., 1993; Maxwell et al., 1991; also Curran & Muthén, 1999; Muthén & Curran, 1997).

Some researchers (Curran & Muthén, 1999; Muthén & Curran, 1997) have estimated statistical power of growth curve models with complete data for a variety of different sample sizes, number of measurement occasions, and effect sizes. However, how these models would operate under conditions of missing data is not known. Furthermore, even when researchers have examined the effects of missing data on parameter estimates (Enders & Bandalos, 2001), evaluation of model fit (Davey, Savla, & Luo, 2004), and standard errors (Graham, Taylor, & Cumsille, 2001), they have typically considered only relatively small proportions of missing data. Additionally, most researchers have concentrated on examining MCAR data and fewer have looked at MAR data. Lastly, most intervention research has sample sizes between 200 and 700 (e.g. PDFY = 209 families; Spoth, Redmond & Shin, 2001; SAAF = 400 families; Brody et al., 2004; ISFP=667 families; Molgaard, Kumpfer & Fleming, 1996).
Even though we know that as sample size increases, statistical power also increases, we do not know how missing data, sample size and statistical power would interact with each other. This study examines the statistical power of a growth curve model with varying amounts of sample sizes and missing data. Below are some research questions that were examined in the present study.

Research Questions

1. What is the association between extent of missing data and statistical power to detect group differences in longitudinal change in a growth curve model? In particular, does statistical power decrease as quickly as missing data increases?

2. Does the type (i.e., MCAR or MAR) of missing data differentially affect the statistical power to detect group differences in longitudinal change in a growth curve model?

3. How does the sample size affect the rate of decrease in statistical power as a function of missing data?

4. Are there interactions between sample size, percentage of missing data and statistical power?

Design & Procedure

*The Statistical Model*

This first study extends the 5-wave complete-data power analysis simulation used by Curran & Muthén (1997, 1999) to missing data designs, with one slight simplification. Curran and Muthén (1999) employed a two-group growth curve model for their analyses that included differential rates of longitudinal change (which is retained in this study) as well as an interaction effect between initial status and rate of change (which is excluded in this study). Equal numbers
gof individuals were included in the treatment and control conditions. Expressed as a structural

equation model, this suggests that the population covariance matrix (\( \Sigma \)) equals \( \Lambda \Psi \Lambda' + \Theta \epsilon \)

\[
\Lambda_y = \begin{bmatrix}
1 & 0 \\
1 & 1 \\
1 & 2 \\
1 & 3 \\
1 & 4
\end{bmatrix},
\Psi = \begin{bmatrix}
1 & 1118 \\
1118 & .2
\end{bmatrix},
\Theta_y = \begin{bmatrix}
1.00 & 0 & 0 & 0 & 0 \\
0 & 1.42 & 0 & 0 & 0 \\
0 & 0 & 2.27 & 0 & 0 \\
0 & 0 & 0 & 3.47 & 0 \\
0 & 0 & 0 & 0 & 5.09
\end{bmatrix},
\]

where \( \alpha = \begin{bmatrix} 1 \\ .798 \end{bmatrix} \)

for the control group and \( \alpha = \begin{bmatrix} 1 \\ .981 \end{bmatrix} \)

for the treatment group. By simple matrix

arithmetic, these yield the following population covariance matrices for the control and treatment
groups.

\[
\Sigma = \begin{bmatrix}
2.000 & 1.112 & 1.224 & 1.335 & 1.447 \\
1.112 & 2.847 & 1.735 & 2.047 & 2.359 \\
1.224 & 1.735 & 4.494 & 2.759 & 3.271 \\
1.335 & 2.047 & 2.759 & 6.942 & 4.183 \\
1.447 & 2.359 & 3.271 & 4.183 & 10.17
\end{bmatrix}
\]

The corresponding vectors of means for the Control and Treatment group are

\[
\tau_{yControl} = \begin{bmatrix}
1.000 \\
1.798 \\
2.596 \\
3.394 \\
4.192
\end{bmatrix}, \quad \tau_{yTreatment} = \begin{bmatrix}
1.000 \\
1.981 \\
2.962 \\
3.943 \\
4.924
\end{bmatrix}
\]

Group differences in rates of change are selected to correspond to an effect size of .2,
based on the pooled standard deviation across treatment and control groups. Residual variances
are selected such that they represent reliabilities of .5 within each occasion (i.e., half of the total
variability at each occasion is true score variability).

The model described above is presented graphically in Figure 3.
Figure 3. A Graphical Representation of the Growth Curve Model from Study 1.
Design

This first study considered three factors: (a) Total sample size (control group + treatment group) varying from $N = 100$ to $N = 1000$ in increments of 50; (b) Type of data wherein there is complete data, MCAR data, or MAR data (MAR data were selected as a function of the pre-test or Time 1 score); (c). Finally, the extent of missing data ranging from 5% to 95%, with Times 1, 2 and 3 always observed and Times 4 and 5 partially observed.

Analysis

All analyses were performed with LISREL 8.5 using the multiple group structural equation modeling approach illustrated by Allison (1987), Muthén, Kaplan, & Hollis (1987) and more recently by Graham, Taylor, and Cumsille (2001) in their examination of power in planned missingness designs. For MCAR data, the population covariance matrices mentioned earlier were used. For MAR data, 1,000,000 cases were generated in Stata (Version 7) under the above mentioned population structure and sorted by their Time 1 values. Complete data and missing data covariance matrices were then generated for each quartile (5 to 95%) for use as input in the LISREL models.

To evaluate statistical power, the method suggested by Satorra and Saris (1985) was used. This approach uses the population covariance matrix to estimate a model in which one or more parameters are constrained to an alternative value. In this case, the model held latent means equal across treatment and control conditions in order to provide an estimated non-centrality parameter value for the comparison of mean change scores. Power is then calculated directly from this parameter as $Power = 1 - NPDF \chi^2(\chi^2_{\text{Crit}}', df', NCP)$ where $NPDF\chi^2$ is the noncentral chi-square probability density function, df is the degrees of freedom, in this case 1,
and NCP is the estimated noncentrality parameter. Non-centrality parameters were obtained for each sample size and each condition of missing data, and converted to their corresponding values of statistical power.

The STATA syntax used to generate the covariance matrices and mean vectors appear in Appendix A. A sample LISREL syntax used to analyze the incomplete data using multiple group approach is presented in Appendix B. The minimum values of the fit function ($F_{\text{Min}} = \chi^2/N$) and chi-square statistics and statistical power under each condition are available in Appendix C.

Results

The nature of simulation research is that the conditions and number of replications are arbitrary and under the control of the researcher. Thus, it becomes possible to select any conditions that lead to statistically significant results. Because this study focuses on the population (i.e., known) values, results are provided graphically rather than statistically. These graphs are used to address each of the four research questions for this study.

1. What is the association between the extent of missing data and statistical power to detect group differences in longitudinal change in a growth curve model? In particular, does statistical power decrease as quickly as missing data increase?

Figures 4 and 5 illustrate statistical power as a function of sample size for MCAR and MAR data respectively. As expected, statistical power increases with sample size under all conditions of missing data, and statistical power decreases with greater levels of missing data. In general, the pattern appears fairly similar for both MCAR and MAR data. Figures 6 and 7 represent the same data as a function of the extent of missing data.
Figure 4. Statistical Power & Sample Size for MAR Data

Figure 5. Statistical Power & Sample Size for MCAR data
Figure 6. Statistical Power & Missing data for MAR data

Figure 7. Statistical Power & Missing data for MCAR data
What these figures further illustrate is that the effects of missing data do not appear to be constant across all sample sizes. Power starts low and stays low at smaller sample sizes and begins high and stays high at larger samples sizes. At intermediate sample sizes, however, the effects of missing data on statistical power are steeper. These observations are borne out in Figures 8 and 9, which represent power for MCAR and MAR data respectively as a function of their complete-data values. It is clear from these figures that the proportional power is highest at small and large sample sizes. However, in terms of the first research question, even when relative statistical power is lowest (i.e., in this case for a sample sizes of 300), the relative loss of power is always much less than the loss of data. For example, even when 95% of the cases have incomplete data for \( N = 300 \), the statistical power is still 47% of its complete data value. For \( N = 1000 \), the statistical power is still 76% of its complete data value even when 95% of the cases have incomplete data.

Figure 8. Proportional Power with MAR Data
2. Does the type (i.e., MCAR or MAR) of missing data differentially affect the statistical power to detect group differences in longitudinal change in a growth curve model?

In general for this model, MCAR and MAR data appear to have quite similar effects on statistical power. However, it is also clear that there are some small but potentially important differences as well, and so these are described below. Figure 10 compares the percentage difference in power obtained with MCAR and MAR data. As seen, the difference in power between MCAR and MAR data increases with sample size up to $N = 650$ when the difference between MCAR and MAR data is greatest (6.89%) and when 60% of cases have incomplete data. After this point the difference in power between the two missing data mechanism decrease gradually with increasing sample size, although still remaining highest for 60% of missing data.
models. Since the maximum difference between the MCAR and MAR data is only approximately 7%, the results of MAR and MCAR data are not presented separately.

Figure 10. Difference in Statistical Power between MCAR and MAR Model

3. How does the sample size affect the rate of decrease in statistical power as a function of missing data?

Continuing the discussion of how missing data affects statistical power above, the variance in power is largest at medium sample sizes (500-750) compared to lower or higher sample sizes (see Figure 4, 5, 6 and 7). Interestingly, at N=1000, even when one has up to 85% missing data, in other words complete data on only 150 cases; the models had a power of 0.80 and higher, whereas at sample size 150 with complete data, the model had a power of about 0.22. When
there was more than 85% missing data with \( N=1000 \), the statistical power of these models reached close to 0.80 (approximately 0.76) (see Figure 4, 5, 6 and 7). Similarly in Figure 8 and 9, for both MAR and MCAR data, statistical power is at its maximum at the largest sample size and decreases with greater missing data in the model. There is a difference in the drop in statistical power for MAR and MCAR data at medium sample size and medium missing data percents, wherein the drop in power is slightly more pronounced for MAR data than for MCAR data.

4. Are there interactions between sample sizes, percent of missing data and statistical power?

As the preceding presentation of findings clearly illustrates, there appear to be interactions between each factor of sample size, type of missing data, and extent of missing data. The effects of missing data are relatively greater at moderate sample sizes than at sample sizes which are either small (associated with consistently low statistical power) or large (associated with consistently high statistical power) (See Figure 8 and 9). MCAR and MAR data, while generally showing similar trends showed a complex association with both sample size and the extent of missing data. While differences are generally trivial for this example, MCAR shows slightly greater statistical power than MAR data (see Figure 10).

To summarize, four important findings emerge from these results. First, the difference in power between MCAR models and MAR missing data mechanisms is quite small. Second, models with large sample sizes and large amounts of missing data still had more statistical power to detect group differences in longitudinal change than even models with little missing data but smaller sample sizes. Third, the loss of power seems to be more pronounced at moderate sample
sizes than either small or large sample sizes. Finally, sample size, type of missing data, and extent of missing data all appear to interact in terms of their effects on statistical power.
CHAPTER 5

STUDY 2: VARIABLES MODERATING EFFECTS OF MISSING DATA ON STATISTICAL POWER

Over and above the basic research issue of how missing data affects statistical power are at least two categories of more applied issues. The first is how best to compensate for missing observations that are expected by researchers, i.e., the aspect of missing data that is beyond the researchers’ control. In family-based intervention programs, it is not uncommon to have more than two-thirds of families decline participation or eventually drop-out of the study (Spoth, Molgaard, 1993; Spoth & Redmond, 1993; Weinberger, Tublin, Ford & Feldman, 1990). Most researchers spend vast amounts of resources (human and financial) to recruit and retain participants in a study. When researchers expect some amount of missing data in their studies, which happens almost always, it would be most beneficial to know whether there are any concrete strategies that can be applied in order to diminish the effects of missing data on statistical power without having to invest huge amounts of resources in recruiting or retaining participants. Reliability of indicators and inclusion of covariates seem to be the most promising among other ideas.

A second applied component pertains to designed missingness studies, in which researchers deliberately impose a missing data structure prior to actual data collection. Previous research has suggested that the way in which such a study is planned can affect statistical power, but has not offered insights into how best to design such studies. This second study is designed
to inform each of these situations by addressing the following research questions. As with Study 1, results are presented visually rather than statistically.

Research Questions

Study 2 has been divided into three distinct parts, each addressing a distinct research question. The research questions are noted accordingly.

1. What is the role of reliability of indicators in moderating the effects of missing data on statistical power?

2. What is the role of an auxiliary variable (i.e., a covariate not associated with missingness) in moderating the effect of missing data on statistical power?

3. How does the specific pattern or patterns of missing data affect statistical power?

Study 2A: Reliability of Indicators

The controversy over increasing the power to detect a treatment effect by increasing the reliability of a dependent variable has existed for over two decades (see Cleary & Linn, 1969; Fleiss, 1976; Humphreys & Drasgow, 1989; Nicewander & Price, 1983; Overall, 1989; Overall & Woodard, 1975; Sutcliffe, 1980). Whether reliability can increase statistical power largely depends on how much it can decrease the error variance. Some researchers have examined whether one could use the reliability of instruments to increase estimates and power. For instance, Maxwell and his colleagues (1991) noted that ANCOVA models were more statistically powerful when one had higher reliability of indicators. On the other hand, in the presence of marginal reliability ANOVA models with larger gaps between the two measurement points were found to be more powerful and required fewer subjects. Similarly, Duncan, Duncan,
and Strycker (2002) also emphasized in their study the relationship between the reliability of a study’s measures and simultaneous increases in power obtained within the SEM framework. Although much research has looked at how reliability of instruments could increase statistical power by decreasing the error variance, researchers have not considered how the reliability of indicators was associated with statistical power in the presence of missing data. In other words, to what extent can the reliability of indicators compensate for the loss of statistical power due to missing data? In the first part of Study 2 this is examined in an exploratory fashion.

**Design and Procedure**

This study extends the same model used in Study 1 to consider three levels of reliability: .3, .5, and .7. The resulting diagonal elements of the Θε matrices used for this study are shown in the Table 2 below.

<table>
<thead>
<tr>
<th>Reliability</th>
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<td>3.321757</td>
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<td>11.88700</td>
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</tbody>
</table>

As in Study 1, multiple-group analyses using LISREL were estimated for models with no missing data, 30%, 50% and 70% missing data, again keeping Times 1, 2, and 3 completely observed, and Times 4 and 5 partially observed. Once again the FMin values from these analyses were used to calculate the chi-square values for sample sizes ranging from 100 to 1000 in increments of 50. Statistical power was calculated using the Satorra-Saris approach (1985) described earlier. Complete FMin values, chi-square statistics and statistical power under each condition are presented in Appendix D.
Results

1. What is the role of instrument reliability in moderating the effects of missing data on statistical power?

Figures 11, 12, and 13 plots the statistical power to detect group differences in longitudinal change as a function of the proportion of missing data for indicators with low (.3), moderate (.5), or high (.7) reliability, respectively. As can be seen, statistical power is greater when reliability of the indicators is higher. The effects of reliability as a moderator of the effects of missing data are quite striking. For a sample size of 100, for example, an indicator with a reliability of .5 and 70% missing data has roughly the same power as an indicator with a reliability of .3 and no missing data. Comparable effects can be observed for the difference in reliability of .7 compared with reliability of .5. Similar trends can be observed at other sample sizes, as well. Interestingly, one will also notice that the drop in power due to missing data is steeper for lower reliabilities at all sample sizes. In other words, when reliability is higher, the effects of missing data are diminished. This provides clear support for the idea that reliability moderates the effects of missing data on statistical power. As has been shown, relatively modest increases in reliability can lead to fairly substantial improvements in statistical power even in the face of very high levels of missing data. This suggests one important strategy that researchers can employ in situations where missing data are either expected or planned.

To make this point clearer, Figure 14 plots the statistical power as a function of sample size. This plot reiterates the idea that effects of higher reliability can be used to offset effects of a great deal of missing data. For instance, at all sample sizes, the models with 70% missing data and 0.5 as reliability of indicators was just as much powerful (approximately 1% lower) as models with 0% missing data with 0.3 reliability.
Figure 11. Statistical Power based on Sample size, Percent Missing (0, 30, 50 & 70%) & Reliability = 0.3

Figure 12. Statistical Power based on Sample size, Percent Missing (0, 30, 50 & 70%) & Reliability = 0.5
Figure 13. Statistical Power based on Percent of Missing Data (0, 30, 50 & 70%) & Reliability = 0.7

Figure 14. Statistical Power Based on Sample Size (100 to 1000), Percent of Missing Data (0%, 30%, 50% & 70%) and Reliability of Indicators (0.3, 0.5, 0.7).
To further understand the drop in statistical power, Figures 15, 16, and 17 compare the models with different proportions of missing data to their complete-data values. Compared to the no-missing data situation, there is a greater proportional reduction in power at smaller sample sizes than at larger sample sizes and with more missing data than less missing data.

Intriguingly, in the proportional graphs (Figure 15, 16 & 17) for models with 30, 50 & 70% missing data with high reliability (.70), medium reliability (.50) and low reliability (.30), demonstrate that at lower sample sizes the loss of power is greater, and more so for variables with lower reliability, however, these increase with higher sample sizes. The slight dip in power seen at medium sample sizes in Study 1 diminishes when the model has indicators with high reliability.

![Figure 15. Proportional power of Model A with 0.3 reliability and missing data (30%, 50% & 70%).](image)
Figure 16. Proportional power of Model B with 0.5 reliability and missing data (30%, 50% & 70%).

Figure 17. Proportional power of Model C with 0.7 reliability and missing data (30%, 50% & 70%).
Conclusions

It is quite evident that in terms of statistical power, models that have indicators with high reliabilities seem to fare much better than those with low reliabilities. Additionally, in the presence of missing data, models with higher reliability and large percentage of data missing seem to have just as much statistical power as a model with no missing data but uses measures with low reliabilities. It is thus recommended that researchers should take every step possible to increase reliability; especially when a moderate to large degree of missing data is expected in the selected sample.

Study 2B: Inclusion of an Auxiliary Variable

Design and Procedures

Inclusion of an auxiliary variable that is related to Time 1 scores, but is unrelated to the missing data mechanism is included in the two-group growth curve model from Study 1, following Graham’s (2003) observation that inclusion of such auxiliary variables can increase the efficiency of estimates with incomplete data. The auxiliary variable represented correlations ranging from .1 to .7 in increments of .2 for this study.

In this study, inclusion of an auxiliary variable is evaluated for different types of missing data (none, MAR vs. MCAR), percentage of missing data (5% to 95% of cases) and sample size (100 to 1000 in increments of 50). To create the data for MCAR data, the variance and covariances were deleted at Time 4 and Time 5 and a covariate which was correlated with Time 1 was added to the covariance matrix, as illustrated in Figure 18.
Figure 18. Graphical Representation of a Growth Curve Model with an Auxiliary variable correlated with Time 1.
To create the data for MAR data for the control and treatment group, datasets with 1,000,000 observations with 5 waves of data and a covariate correlated with Time 1 were simulated, with missingness weighted on Time 1, to create covariance matrices and mean vectors for different amounts of missing data. The STATA code used appears in Appendix E.

The resulting covariance matrices and mean vectors were used to estimate the model presented in Figure 18 using LISREL with multiple group full information maximum likelihood to estimate model parameters with missing data. The resulting $F_{\text{Min}}$ values were used to calculate Chi-squares for sample size ranging from 100 to 1000 total cases, followed by computation of statistical power using procedures identified by Satorra and Sarris (1985) (See Appendix F and Appendix G for $F_{\text{Min}}$, chi-square statistics and statistical power of each of these models under MCAR and MAR data respectively).

Results

To simplify the results of this study only two different levels of association of the auxiliary variables are discussed here with the lowest covariate being treated with the greatest detail. Model A is least correlated to Time 1 with a correlation of 0.1 and Model B is highly correlated with Time 1 data with a correlation of 0.7. Since the differences between MCAR and MAR data were very slight, the graphs from the MAR data are not discussed here.

Direct comparisons between models with and without the inclusion of auxiliary variables were found to be most informative. Figure 19 below illustrates this point by showing the percentage difference between Study 1 Model with MCAR data and Model A with a covariate with $r=0.10$ in the presence of MCAR data. It becomes quite clear from this graph that there is a large proportional increase in statistical power at lower sample sizes, and a smaller percent of
increase in power at medium and higher sample sizes. At the same time there also seems to be lower proportional increases in power when there are medium amounts of missing data compared to low amount of missing data and high amounts of missing data. Finally, at the largest sample size considered here ($N = 1000$), there seems to be no difference in the power between the two models when there is a small or mediocre amount of missing data. However, when there is a large amount of missing data, adding even a weak covariate could increase the statistical power by almost 16%.

Figure 19. Percentage Difference in Statistical Power between Model 1 (without auxiliary variable) and Model A (with auxiliary variable = .10).
Subsequent to the previous comparison where even a weak covariate could increase the statistical power of a model, another comparison is made between Model B with a strong covariate \( (r = 0.7) \) and Model A with a weak covariate \( (r = 0.1) \) to illustrate that by adding a stronger auxiliary variable one can further augment the statistical power of the model to detect change. Figure 20 plots the percentage difference between Model A \( (r = 0.1) \) and Model B \( (r = 0.7) \) by sample size. It is quite evident from these graphs that by adding a stronger auxiliary variable, there can be about 25\% to 30\% increases in power at smaller sample sizes. This increase in power is predominantly for models with a large percent of missing data, for instance, at a sample size of 250 with 95\% missing data, statistical power of a model with a stronger covariate is 32\% more than a model with a weaker covariate of 0.1.

![Figure 20. Percentage Difference between Model B (r = 0.7) and Model A (r = 0.1) by Sample size.](image-url)
The percentage difference between the two models however drop with increasingly large sample sizes, yet it still continues to have almost 8% (at sample size 1000) to 13% (at sample size 750) with higher percentages of missing data. This suggests that adding a stronger covariate would help increase the power especially at low to medium sample sizes and when there are large amounts of missing data.

Figure 21. Percentage Difference between Model B (r = 0.7) and Model A (r = 0.1) by Percent of Missing Data.

Figure 21, shown above, plots the percentage difference between Model B with a strong covariate (0.7) and Model A with a weaker covariate (0.1) by the percentage of missing data. Two things are quite evident in this graph. First, adding a stronger covariate in models with
smaller sample sizes is more beneficial since there is more than 20 to 25% increase in power regardless of the percentage of missing data. Secondly, it is once again, quite evident from this graph that adding a stronger covariate with models with moderate to larger sample sizes would be beneficial only when there is a larger percentage of missing data. For instance, there is little difference between the two models at a sample size of 1000 when the missing data is limited to 50% and lower. However, when there is 95% missing data, the power increases by 7.5%. The results from the comparison between a stronger and a weaker covariate illustrates that a stronger auxiliary variable could be recommended when the percentage of missing data is large and the sample size is small to medium.

Conclusions

Even in the presence of an extremely weak auxiliary variable (r = .10), the statistical power of models with missing data could be boosted. Adding a covariate seems to be especially beneficial when one has a small sample size since that is where the greatest proportional increase in power can be realized. At the same time, adding a covariate would be more beneficial in the presence of either large or small amounts of missing data instead of moderate amounts of missing data since the increase in power is lower in medium amounts missing data models. However, this should be qualified by the fact that the addition of an auxiliary variable is always seen to increase statistical power. Likewise, when one has a large sample size it would be more beneficial to use a covariate only when a large amount of missing data is expected, since that where the relative efficiency of a model that includes an auxiliary variable is higher. Lastly, a stronger auxiliary variable can boost the power particularly when there is large percentage of missing data and the sample size is small to medium.
Study 2C: Pattern Missingness in Waves

*Design and Procedures*

Because of the potential complexities introduced by considering 5-wave data fully, Study 2C represents a simplification of the models used in other parts of this dissertation. Specifically, Study 2C uses 3-wave data. For this part of the study, a $3 \times 3$ covariance matrix with Wave 1, Wave 3 and Wave 5 of the original study were used. The model in which 50% of the data were missing in the control and treatment group was used as an example to examine whether missing data in specific waves differentially affects statistical power. In this study, whether the pattern of missingness was affected by whether data were MCAR or MAR was of particular interest. Table X shows the covariance matrices used for this part of the study, following the notation used by Graham, Taylor, and Cumsille (2001).

Four different patterns of missingness were evaluated and compared in this study. Model A is a four-group (two patterns of missing data in treatment and control groups) model with 50% data missing on Wave 3 in both the control and the treatment groups. Model B is a four-group model with 50% of cases missing on Wave 5 in the control and the treatment group. Model C is a six-group model with 25% cases missing on Wave 3 data and another 25% cases missing on Wave 5 in the control and treatment group. Model 4 is an eight-group model with 25% data missing on Wave 3, 25% missing on Wave 5 and another 25% cases missing on Wave 3 as well as Wave 5. Table 3 illustrates which waves were observed or missing, and the number of observations in each pattern of missing/observed data.

For the sake of simplicity, from this point forward, Wave 1 is addressed as Time 1, Wave 3 is addressed as Time 2 and Wave 5 is addressed as Time 3.
Table 3. Distribution of the pattern of missingness in the four models. (Only Control group shown here. Same pattern exists for the Treatment group).

<table>
<thead>
<tr>
<th>Groups</th>
<th>Time 1</th>
<th>Time 2</th>
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<tr>
<td></td>
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<tr>
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<td>Observed</td>
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<tr>
<td></td>
<td>Group 2</td>
<td>Observed</td>
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<td>Missing</td>
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<td></td>
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</tr>
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<td>Model D</td>
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<td></td>
<td>Group 3</td>
<td>Observed</td>
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</tr>
<tr>
<td></td>
<td>Group 4</td>
<td>Observed</td>
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</tr>
</tbody>
</table>

Procedures

The above mentioned multi-group LISREL models with a total of 50% missing data (MCAR and MAR) were run to estimate the minimum value of the fit function. The resulting $F_{\text{Min}}$ was used to calculate Chi-squares for sample sizes ranging from 100 to 1000 in increments of 50. Using the Satorra-Sarris (1985) approach, power estimates were calculated and then
graphed. Complete values of $F_{\text{Min}}$, chi-square statistics and resulting statistical power under all conditions examined are presented in Appendix H.

Because there were large differences between MCAR and MAR data for designed missingness, results are presented separately for each type of missing data before comparisons are made. Figures 22 and 24 plot the statistical power of the four models by sample size under MCAR conditions and MAR conditions, respectively. Similarly figures 23 and 25 plot the percentage difference between the four models and complete data design by sample size under the MCAR conditions and MAR conditions, respectively.

**MCAR Data**

*Models A and B*

Model A had 50% missing data on Time 2 and Model B had 50% missing data on Time 3. These two models had nearly identical power at all sample sizes (see Figure 22). Compared to the complete data situation in Figure 23, there is a 10 -12% difference in statistical power at medium sample sizes such as 250 to 500. At smaller sample size (N=100) and large sample size (N=1000) the percentage difference between Model A/Model B and complete data Design is quite small (4%). This suggests that when a researcher has complete control over whether values will be missing or observed, there is quite a lot to be gained by taking advantage of designed missingness, consistent with what previous researchers have suggested (Graham et al., 2001).

*Model C*

For models with MCAR data, Model C, which has 50% of data missing divided equally between either Time 2 or Time 3, came closest to achieving the power estimates of a complete data design (see Figure 22).
Figure 22. Statistical Power of Models by Pattern Missingness for MCAR data

Figure 23. Percentage Difference between Pattern Missingness Models and Complete Data Models under MCAR data conditions
Compared to the complete data model in Figure 23, there is only a 4% drop in power compared to the complete data situation at the sample size of 100. However, this difference increases gradually up to the sample size of 400 (9.45%), after which the drop in power decreases to as low as 3% at sample sizes of 1000. The greatest difference in power is at medium sample sizes such as 250 to 500 which ranged from 8.25% and peaked at 9.46%. This suggests that including both patterns of missing data provides additional gains over including either one separately when individuals can be randomized to specific missing data conditions.

*Model D*

Model D had 50% missing data divided equally across W2 alone, W3 alone, and both W2 and W3. This pattern showed the greatest drop in power compared to the complete data models shown in Figure 23. At small sample size the decrease in power was about 5.54% and at moderate sample sizes it peaked at 14%. However, with larger sample sizes, the percentage drop in power was only about 6%. This finding makes sense given that, framed in terms of the number of data values that are missing (cf. Graham et al., 2001), Model D has more missing data values than models A through C.

*MAR Models*

*Model A*

Interestingly, when one had missing at random data (MAR), Model A with 50% missing data on only Time 2 showed the smallest drop in statistical power compared to the other three models (see Figure 24). As is evident in Figure 25, the drop in power was minimal at small and large sample sizes (about 1 to 2%) and greater at moderate sample sizes (approximately 4%). With sample sizes of at least 550, this model reaches the statistical power of 0.8.
Model B

Intriguingly, Model B, with 50% missing data only at Time 3 had the highest percentage drop in MAR data (See figure 25). At small sample size the drop in power was about 8%. At sample size 450, the drop in power reached its peak at 22%. This is especially informative given that, in the longitudinal context, data are most likely to be missing in a systematic fashion (i.e., MAR), and most likely to be missing at later waves. In other words, the pattern of missing data associated with the greatest reduction in statistical power is the one that appears most likely to occur in practice.

Figure 24. Statistical Power of Models by Pattern Missingness for MAR data
Figure 25. Percentage Difference between Pattern Missingness Models and Complete Data Models under MAR data conditions

*Model C*

Next in line is Model C which has 25% missing data on Time 2 and 25% missing data on Time 3. Model C continues to have similar amounts of power as in the MCAR data (see Figure 24). As can be seen in Figure 25, at low sample sizes, the drop in power was about 3.35% with a still lower difference at larger sample sizes (2.45%). At moderate sample sizes, the difference in power rises to about 8%.
Model D

Model D has missing data on either Time 2 or on Time 3 and some also had missing data on Time 2 and Time 3 (see Figure 24). This model also seems to hold the same amount of power regardless of the type of missing data. At lower sample sizes the percentage drop was roughly around 6%. The highest percentage drop was at 17% at the sample size of 400.

Conclusion

When data is missing completely at random the loss in power is significantly associated with the loss of data points. This was clearly seen in the case of Model D with MCAR data wherein the loss of data points is the highest. However, when both patterns of missing data are provided (such as in Model C) the gains in power is a lot more than including only one of the patterns (such as in Model A and Model B). For MAR data, however, the model with missing data on Time 3 was found to be most detrimental to statistical power. On the other hand, when data was missing at the intermediate time-points (Model A), the loss in power was trivial. These results suggest that the power obtained by models with designed missingness would differ depending on whether the data is MCAR or MAR. More work is required to untangle the effects of the type of missingness on different waves of a study.
CHAPTER 6
DISCUSSION

Summary of Key Findings

Study 1: Association between Missing Data and Statistical Power

When researchers want to measure change in participants over time as an effect of the intervention program, one limiting factor inhibiting a statistically powerful study is the dilemma of missing data. Limited work has been done on the topic of missing data and its association with the statistical power of growth curve models in the intervention context (but see Graham et al., 2001). Thus, the aim of an initial study was to examine the interaction of missing data (amount and type) with sample size and statistical power in a 5-wave growth curve model. The results showed anticipated increases in statistical power with increasing sample size under all conditions of missing data and decreases with greater levels of missing data. For instance, at lower sample sizes the power was found to be low and it remained low with missing data. Similarly, at higher sample sizes the power started high and remained high even with a great deal of missing data.

However, a more interesting finding was that the effects of incomplete data were found to vary as a function of sample size. At medium sample sizes the effects of missing data were found to be steeper and the loss of power greater than small and larger sample sizes. This is a significant finding for intervention researchers, since most evaluation studies (e.g. PDFY = 209 families; Spoth, Redmond & Shin, 2001; SAAF = 400 families; Brody et al., 2004; ISFP=667 families; Molgaard, Kumpfer & Fleming, 1996) have relied on having sample sizes between 200
and 600 (medium by the standards of the present study), when that is where the missing data affects statistical power the most.

At the same time, the present study found that models with larger sample sizes and higher percentage of missing data seem to have relatively more power than models with the same amount of missing data but with lower sample sizes or even complete data with the same number of valid observations. For instance, with a sample size of 1000 with 85% missing data (complete data on 150 cases only) has a power of 0.8 and higher. Whereas, when one had 0% missing data with a sample size of 150, the statistical power of that model was 0.22. Most researchers spend a lot of time and money to retain subjects in their study over the course of the study, but this finding suggests that it may be more economical to recruit more participants initially and then retain only 15% of the original sample in the latter waves (85% missing) and yet have more power to detect change.

Another interesting finding is that the differences between MCAR and MAR non-response were generally found to be quite trivial in this study. The difference between the two types of missing data peaked at moderate sample sizes (N=650) with 60% missing data, with MCAR data models being slightly more powerful than models with MAR data. This difference slowly decreases with increasing sample sizes and missing data. Although both MCAR and MAR data require that the variable with missing data be unrelated to whether or not a person has missing data on that variable, however, the missingness in MAR data is related to some of the variables in the data set (Time 1 data in the present study). Consequently, when one has MCAR data, the loss of power is simply due to the loss of sample size, on the other hand, with MAR data the reason for the loss of power is considerably more complex and requires further examination.
At the same time, as most researchers have studied MCAR data, we know less about the how and why MAR data perform. One study that requires mentioning here is the one by Davey et al. (2004), who simulated data using a complex grid of factors such sample size, missing data, misspecified models at the structural and measurement levels and type of missing data. They found that when models were slightly misspecified at the measurement level, the difference in the fit indices of models with MAR and MCAR data was very minimal; however, these differences increased with misspecifications at the structural level of the CFA model. This study once again serves as a reminder of the complexity of assessing statistical power in the context of structural equation models with missing data.

In summary, the interactions between the sample size, statistical power and missing data were quite evident from the various graphs. The results from this study suggest that it may be more economical to recruit more participants initially and then plan to have systematic missing data on the latter waves of study. The proportional power graphs clearly show that models with medium sample sizes were more prone to the deleterious effects of missing data than small or large sample sizes. Intervention researchers using a similar design should therefore be more cautious of using medium sample sizes especially when they are expecting a medium to large drop-out rate in the last few waves of their studies as is evident in the literature.

**Study 2: Variables Moderating Effects of Missing Data on Statistical Power**

Intervention researchers are challenged by two issues simultaneously. On one hand, it has become more and more important to conduct studies with adequate statistical power in order to publish and obtain grant funding. On the other hand, funding agencies have fewer resources
to give out and therefore researchers have to tolerate larger budget cuts. A budget cut automatically translates into smaller sample sizes which in turn translate into low statistical power to detect changes. To make things worse, there is always a threat of still lower power due to attrition and missing data.

In the face of these challenges, Study 2 provides three crucial suggestions to not only increase statistical power without having to increase sample size, but also utilize missing data as a tool to increase statistical power. An important feature of this study is that since these three methods are under the researcher’s control as a result they provide more flexibility in designing the study. The three moderators of missing data on statistical power that are examined in Study 2 are: (a) reliability of indicators; (b) inclusion of an auxiliary variable; and (c) pattern of incomplete data in waves.

Reliability of indicators

Several researchers have pointed out that power can be increased by using methods that decrease the error variance (Zimmerman & Williams, 1986). Some researchers have shown that when one uses highly reliable dependent variables a smaller sample size is required to achieve adequate power (Maxwell et al., 1991), however, the extent to which reliability of indicators can compensate for the loss of power due to missing data has not been systematically studied.

As expected, statistical power was found to be greater with models with high reliability of indicators. Interestingly, even with some amount of missing data, relatively modest increases in reliability were found to yield fairly substantial improvements in power.
In the presence of missing data, at lower reliabilities the drop in power was found to be steeper as a function of missing data for models at all sample sizes. In contrast, as reliability of models increased the effects of missing data diminished, particularly at higher sample sizes.

A unique finding of this study was that models with higher reliability (e.g. r = 0.7) but with large amount of missing data (70%) performed nearly as well as models with lower reliability (e.g. r = 0.5) and complete data. This suggests that researchers might be at an advantage using a more reliable instrument than worrying about the drop-out rate or retaining their participants in their study. Moreover, the proportional graphs that compared the power of a complete data models with models with varying percent of missing data and sample size found that the increase in the statistical power is greatest at lower to medium sample sizes instead of the larger sample sizes.

In summary, based on the above discussion, it is recommended that researchers who plan to have small to medium sample sizes and expect missing data should use indicators with higher reliability to increase the statistical power of the model to detect significant effects of the intervention program.

*Inclusion of an auxiliary variable*

In the past, researchers have found that including a covariate that was correlated with the dependent variables could also increase power without having to necessarily increase the sample size (Arvey, Maxwell & Salas, 1992; Maxwell, Cole, Arvey & Salas, 1991). They argue that to the degree that the covariate and the dependent variables are correlated, use of the covariates reduces within-group variances and consequently increase the effect size and the statistical power of the test.
Other researchers (Collins, Schafer, & Kam, 2001; also see Graham, Cumsille, & Elek-Fisk, 2003; Schafer & Graham, 2002) that have examined the role of covariates in estimating models with missing data have argued that including a covariate that was related to the missingness mechanism would be helpful only when there is a substantial amount of missing data (50% or more), and when the variable causing the missingness was substantially correlated with the variable that contained the missingness (r > 0.4).

Furthermore, Collins et al. (2001) also noted that when variables that are not the cause of missingness but are nonetheless highly correlated with the variable containing missingness are included in the model they reduce the bias and increase the efficiency in estimating models using procedures such as multiple imputations and EM algorithms for covariance matrices. Collins et al. (2001) refer to these variables as “auxiliary” variables and show how to use these variables with multiple imputations procedure. More recently, Graham (2003) demonstrated ways to include these variables using SEM/FIML procedures. Inclusion of auxiliary variables in longitudinal studies of change seem quite promising; however, to date, none have systematically examined the role of auxiliary variables as a moderator to increase the power of a growth curve model in the presence of varying amounts of missing data and sample sizes using SEM/FIML procedures.

It would be extremely beneficial and cost-effective for intervention researchers who struggle with the loss of power in their longitudinal studies due to attrition, to gather information on such an auxiliary variable which was correlated with pre-test data, and still have significant improvements in power.

This study examines the role of such an auxiliary variable in moderating the effects of missing data on statistical power. The most important feature of this study is that the role of an
auxiliary variable that ranges from very high correlation ($r = 0.7$) to very low correlations ($r = 0.1$) with Time 1 data are examined with different amounts of missing data (5% to 95%) and at different sample sizes ($N = 100$ to 1000) so as to explore the whole range of conditions that would be reflective of real data from the field.

Validating the conclusions of previous studies, the results in the present study shows that including an auxiliary variable in the two-group growth curve model increases statistical power at all sample sizes. Furthermore, even in the presence of a small covariate ($r = 0.1$ with Time 1) the results show that there is a large percent of increase in power at lower sample size rather than at medium or higher sample sizes. This result suggests that researchers who are limited by either resources or availability of participants and have smaller sample sizes, would benefit the most by adding even a modestly correlated auxiliary variable.

Collins et al. (2001) found that adding a covariate related to the missingness mechanism was beneficial only when there is more than 50% missing data. Contrary to their suggestion, the results in this study show that the proportionately greatest increases in statistical power are either when one has low missing data or very high amounts of missing data. In fact, at medium amounts of missing data, even though there is some gain in power, the percentage gain is much lower than the other two extremes. Researchers are thus advised to add a covariate in their designs if they are expecting low amounts of missing data or are designing large amounts of systematic missing data. Finally, the results show that at large sample sizes, little power is gained by adding a covariate, unless one is going to design large amounts of missing data which results in a 16% increase in statistical power.

Thus, when missing data is inevitable, as in the case of any longitudinal study, not only would the inclusion of a covariate increase the statistical power of the study, but it would also aid
in providing an unbiased estimation of the model during the analysis phase of the intervention program.

*Pattern Missingness*

John McArdle (1994)) suggested that whenever there is missing data there is a loss of statistical power; however, he argued that some patterns of incomplete data retain more power than others. He further argued that under some conditions it would be beneficial for the researchers to plan on having large amounts of incomplete data. Unfortunately, this idea has not been widely adopted within intervention research. Recently, however, Graham, Taylor and Cumsille (2001) started making some preliminary comparisons between different patterns of missingness using growth curve models with MCAR data. They found some of the patterns of missingness have higher standard errors than others. However, their simulation was limited to a sample size of 1000 cases with data missing completely at random (MCAR).

The present study extends these preliminary examinations to inspect how statistical power is associated with the pattern of missingness by whether the data are MCAR or MAR. Due to the complexity of a 5-wave model, the study was limited to the inspection of a 3-wave growth curve model with 50% missing data.

When the data was MCAR in the different patterns of missingness models, the models that included both the patterns of missingness (Model C – 50% missing data equally divided between either Wave 2 or Wave 3) came the closest to achieving the same power as the complete data model and was more powerful than Model A and Model B that had missing data on only one of the waves. This suggests that including both patterns of missing data provides additional gains over including either one separately especially when the missingness has been randomly
assigned by the investigator. Also, when data is MCAR, and every participant has an equal probability of dropping out, then the only problem is the loss of statistical power associated with a reduced sample size. This is clearly seen in Model D which has 50% missing data divided equally across Wave 2 alone, Wave 3 alone and both Wave 2 and Wave 3, thus having a larger number of data values missing compared to the other three models. Obviously, this model faired much poorer in power compared to the other four models and also the complete data model. There was a difference of about 14% between the complete data model and Model D at medium sample size.

When the missing data is random (MAR) but the missingness is related to some variable in the model, the findings were quite different. Model A which had 50% missing data on Wave 2 showed the smallest drop in power compared to the other three models. The drop in power was quite minimal (2% - 4%), with large drops in power at medium sample sizes. This could have been due to the linear function of growth in the model, and therefore the need of an intermediary wave is not as crucial in measuring change.

On the other hand, Model B which had 50% missing data at Wave 3 had the highest percentage of drop in power (8% - 22%). From a longitudinal standpoint, this pattern of missingness is a close reflection of how missing data appears in real studies, where the maximum drop-out or attrition occurs in the later waves. This illustrates the fact that systematic missing data in later waves of a study is detrimental to the statistical power, regardless of whether it is instigated by the investigator or by the participants.

Model C (25% MAR data on Wave 2 and another 25% of Wave 3) continued to show similar amounts of power as in the MCAR data. The difference in power ranged from 2% (at
smaller and larger sample sizes) to 8% (at medium sample sizes). Finally, Model D, also continued to show similar levels of power as in MCAR data ranging from 6% to 17%.

To summarize, the weakest model with MCAR data was the one with the maximum loss of data points (Model D). On the other hand, Model C provided at least some information from all the waves and therefore fared better than the other three models. When the data are MAR, the model with missing data on Wave 3 (Model B) resulted in a maximum drop in power thus underscoring the point that in longitudinal studies systematic missing data in later waves is most detrimental to statistical power. On the other hand, when data is missing at the intermediate wave in the 3-wave linear growth model the gain or loss in power is quite trivial. This finding is quite opposite to the study of Venter, Maxwell, and Bolig (2002) who found that a single intermediate time point adds power to a traditional pretest-posttest design and may reduce the effect of missing data. Further explorations of the effect of missing data patterns on statistical power are needed to clarify these findings.

Greater appreciation of the pattern of incomplete data for statistical power, as well as the potential to exploit systematically missing data in designed missingness are two potential contributions from this aspect of Study 2. As well, these results clearly indicate that there may be many relatively simple solutions (e.g. inclusion of auxiliary variables and superior instrument reliability) to problems of missing data in family-based preventive intervention research.

Limitations

Although several novel and promising ideas have been presented in this dissertation, there are some important limitations of this study as well. In the simulation studies of this dissertation the principal model was based on a linear growth curve model with a small effect
size (analogous to a \( d \) of 0.2, representing a small effect size). The emphasis on a single model is the major limit of the ability to generalize the results of this study to those where statistical power is most likely to be compromised.

Several assumptions were made with the basic growth curve model used in this study. First of all the trajectories of all individuals were assumed to be of the same function namely, linear. Additionally, since the data on the five waves were summarized by their means and covariances it was assumed that the repeated measures, the growth parameters and the residuals were assumed to be multinormally distributed as well (Raudenbush, 2001). However, in the real world, non-linear growth functions and non-normal distribution of measures especially in the context of intervention research is closer to reality (Bauer & Curran, 2003) and these were not examined in this dissertation.

The real world intervention studies use a variety of models and components to study growth trajectories such as using growth mixture models, latent transition models, second-order growth curve models, models with autocorrelated residuals to name a few. However, the present study stayed focused on a simple growth curve model with a linear growth function and uncorrelated residuals. A variety of the models mentioned above continue to remain unexplored even in the literature.

In addition, the benchmark effect size of 0.2 (small in Cohen’s world) as measured by the standardized mean difference between the control and treatment group was used in this study. Although this is as close to the effect size normally seen in family-based preventive intervention research, it would be interesting to look at how the association of missing data with statistical power changes at medium and higher effect sizes.
Another argument that could be made is about the examination of the parameters of the growth curve models. A growth curve model only has a few estimable parameters involving the slope, intercept, variance between these, covariances between the intercept and slope and the error variances. However, the present study focused its attention on only one of these parameters.

Furthermore, researchers who study unbalanced designs, a commonly found design in a real-world situation, have often argued that they are more powerful than balanced designs (e.g. (Allison, Allison, Faith, Paultre, & PiSunyer, 1997b; Liu, 2002). However, in the present study the simulations are based on balanced designs (equal number of subject in the control and treatment groups). Consequently the effects of differential drop-out rates between the intervention and control group were also not studied.

Lastly, most simulation studies that examine artificial data often try and examine their results in view of real world data, thus allowing for generalizability of their simulation results. This was not addressed in the present study; however, it opens doors for several future research prospects.

Future Research

Based on the limitation of the present study, several future researches scenarios could be considered with the main goal of making the simulation study as externally valid as possible to the real intervention situations.

Examination of the association of missing data and statistical power with regard to a variety of common world models should be undertaken to inform intervention researchers in
future studies. Similarly, real-world scenarios such as unbalanced designs, differential drop-out rates in control and experimental groups, non-normality of measures, examination of NMAR data (Not Missing At Random), multiple patterns of missing data (e.g. MCAR and MAR data missing at a Wave), comparisons of models with different effect sizes (small, medium and large), and examination of a variety of parameters should be undertaken in future research to examine its effects on power.

Another beneficial outcome for researchers would be to present to the extension agents involved with the intervention programs and funding agencies a more meaningful report on how much a preventive intervention study would cost in dollar amounts by using simple cost-analysis techniques instead of statistical jargon such as standard errors of parameter estimates and fit indices of models.

Lastly, when studying family-based preventive intervention research, several multi-level variables are significant and need to be included in the analysis. These variables could be at the family, school, community or neighborhood level. How missing data on these levels (Level 1 or Level 2) affects power is another area that continues to remain unexplored.

Finally, we know that real world data and intervention research is a complex affair. Translating simulation data results and generalizing it to real world intervention programs would provide maximum benefit to researchers, practitioners and participants in identifying whether the intervention program produces the promised results.
Conclusion

Invariably, when faced with a situation of missing data, researchers have often abandoned cases with missing data and prefer to have none or even ‘some’ rather than ‘more’ missing data. The simulation studies in this dissertation, however, show that large amounts of missing data in latter waves with higher sample sizes could in fact increase statistical power of a model to detect change. Consequently, researchers are encouraged to essentially plan systematic missing data in their designs and make use of the modern-day missing data analysis to reach more valid and statistically powerful conclusions. At the same time, if researchers, out of no choice of their own, are compelled to have small to medium sample sizes and expect to have ‘some’ or ‘more’ missing data in their studies, based on the results of the simulations studies they could make calculated design and measurement choices to increase the power of their models to detect change. As a final point, researchers are urged to consider missing data not as a problem, but rather as a potential solution for issues such as testing burden and selective attrition which are commonly faced by family-based intervention researchers.
REFERENCES


Little, T. D., Lindenberg, U., & Nesselroade, J. (1999). One selecting indicators for multivariate measurement and modeling with latent variables: When "good" indicators are bad and "bad" indicators are good. *Psychological Methods, 4*, 192-211.

Liu, X. (2002). *Statistical power and optimum sample allocation ratio for treatment and control having unequal costs per unit of randomization*. Unpublished manuscript, Columbia, SC.


APPENDIX A

STATA CODE FOR STUDY 1

STATA (Version 7) Code to Generate the Covariance Matrices and Mean Vectors for MAR Data.

For Control Group

#delimit; 
set mem 200m; 
capture log close; 

/*Save the output */
log using "c:\study1\Curran_control.log", replace; 

/*Define Covariance Matrix */
/*Curran & Muthen, 1999 */
/*Control Group Matrix*/

matrix S4 = ( 
2.000000, 1.1118034, 1.2236068, 1.3354102, 1.4472136\ 
1.1118034, 2.8472136, 1.7354102, 2.0472136, 2.3590170\ 
1.2236068, 1.7354102, 4.4944272, 2.7590170, 3.2708204\ 
1.3354102, 2.0472136, 2.7590170, 6.9416408, 4.1826238\ 
1.4472136, 2.3590170, 3.2708204, 4.1826238, 10.188854); 
matrix M4 = ( 
1.000000, 1.797998, 2.595996, 3.393994, 4.191992); 

/*Set Replicable See*/
set seed 1234567; 

/*Generate a 1,000,000 Observation Dataset*/ 
/* 5 Waves of Data*/ 
corr2data t1-t5, n(1000000) cov(S4) means(M4); 

/*Establish a selection variable to make MAR data */
/*Weighted on Time1 (Pretest) score */
generate scrit01 = -1*t1; 

/*Sort Cases by values on selection variable */
/*Note that this is like the phenotypic (i.e., observed variable) sorting of Dolan */
sort scrit01; 

/*Now determine extent of MAR missing data*/
/*First initialize variables*/
generate sel00 = 0;
generate sel05 = 0;
generate sel10 = 0;
generate sel15 = 0;
generate sel20 = 0;
generate sel25 = 0;
generate sel30 = 0;
generate sel35 = 0;
generate sel40 = 0;
generate sel45 = 0;
generate sel50 = 0;
generate sel55 = 0;
generate sel60 = 0;
generate sel65 = 0;
generate sel70 = 0;
generate sel75 = 0;
generate sel80 = 0;
generate sel85 = 0;
generate sel90 = 0;
generate sel95 = 0;

/*Then make groups representing different proportions of missing data */
replace sel05=1 if _n <= 50000;
replace sel10=1 if _n <= 100000;
replace sel15=1 if _n <= 150000;
replace sel20=1 if _n <= 200000;
replace sel25=1 if _n <= 250000;
replace sel30=1 if _n <= 300000;
replace sel35=1 if _n <= 350000;
replace sel40=1 if _n <= 400000;
replace sel45=1 if _n <= 450000;
replace sel50=1 if _n <= 500000;
replace sel55=1 if _n <= 550000;
replace sel60=1 if _n <= 600000;
replace sel65=1 if _n <= 650000;
replace sel70=1 if _n <= 700000;
replace sel75=1 if _n <= 750000;
replace sel80=1 if _n <= 800000;
replace sel85=1 if _n <= 850000;
replace sel90=1 if _n <= 900000;
replace sel95=1 if _n <= 950000;

/*Now we make and store some matrices by group */
/* 5% Missing */
matrix accum c05 = t1-t5 if sel05==0, means(mc05) dev noconst;
matrix accum m05 = t1-t5 if sel05==1, means(mm05) dev noconst;
/* 10% Missing */
matrix accum c10 = t1-t5 if sel10==0, means(mc10) dev noconst;
matrix accum m10 = t1-t5 if sel10==1, means(mm10) dev noconst;
/* 15% Missing */
matrix accum c15 = t1-t5 if sel15==0, means(mc15) dev noconst;
matrix accum m15 = t1-t5 if sel15==1, means(mm15) dev noconst;
/* 20% Missing */
matrix accum c20 = t1-t5 if sel20==0, means(mc20) dev noconst;
matrix accum m20 = t1-t5 if sel20==1, means(mm20) dev noconst;
/* 25% Missing */
matrix accum c25 = t1-t5 if sel25==0, means(mc25) dev noconst;
matrix accum m25 = t1-t5 if sel25==1, means(mm25) dev noconst;
/* 30% Missing */
matrix accum c30 = t1-t5 if sel30==0, means(mc30) dev noconst;
matrix accum m30 = t1-t5 if sel30==1, means(mm30) dev noconst;
/* 35% Missing */
matrix accum c35 = t1-t5 if sel35==0, means(mc35) dev noconst;
matrix accum m35 = t1-t5 if sel35==1, means(mm35) dev noconst;
/* 40% Missing */
matrix accum c40 = t1-t5 if sel40==0, means(mc40) dev noconst;
matrix accum m40 = t1-t5 if sel40==1, means(mm40) dev noconst;
/* 45% Missing */
matrix accum c45 = t1-t5 if sel45==0, means(mc45) dev noconst;
matrix accum m45 = t1-t5 if sel45==1, means(mm45) dev noconst;
/* 50% Missing */
matrix accum c50 = t1-t5 if sel50==0, means(mc50) dev noconst;
matrix accum m50 = t1-t5 if sel50==1, means(mm50) dev noconst;
/* 55% Missing */
matrix accum c55 = t1-t5 if sel55==0, means(mc55) dev noconst;
matrix accum m55 = t1-t5 if sel55==1, means(mm55) dev noconst;
/* 60% Missing */
matrix accum c60 = t1-t5 if sel60==0, means(mc60) dev noconst;
matrix accum m60 = t1-t5 if sel60==1, means(mm60) dev noconst;
/* 65% Missing */
matrix accum c65 = t1-t5 if sel65==0, means(mc65) dev noconst;
matrix accum m65 = t1-t5 if sel65==1, means(mm65) dev noconst;
/* 70% Missing */
matrix accum c70 = t1-t5 if sel70==0, means(mc70) dev noconst;
matrix accum m70 = t1-t5 if sel70==1, means(mm70) dev noconst;
/* 75% Missing */
matrix accum c75 = t1-t5 if sel75==0, means(mc75) dev noconst;
matrix accum m75 = t1-t5 if sel75==1, means(mm75) dev noconst;
/* 80% Missing */
matrix accum c80 = t1-t5 if sel80==0, means(mc80) dev noconst;
matrix accum m80 = t1-t5 if sel80==1, means(mm80) dev noconst;
/* 85% Missing */
matrix accum c85 = t1-t5 if sel85==0, means(mc85) dev noconst;
matrix accum m85 = t1-t5 if sel85==1, means(mm85) dev noconst;
/* 90% Missing */
matrix accum c90 = t1-t5 if sel90==0, means(mc90) dev noconst;
matrix accum m90 = t1-t5 if sel90==1, means(mm90) dev noconst;
/* 95% Missing */
matrix accum c95 = t1-t5 if sel95==0, means(mc95) dev noconst;
matrix accum m95 = t1-t5 if sel95==1, means(mm95) dev noconst;

/*Make Covariance Matrix by Dividing by Sample Size*/
matrix c05=c05/950000;
matrix m05=m05/500000;
matrix c10=c10/900000;
matrix m10=m10/100000;
matrix c15=c15/850000;
matrix m15=m15/150000;
matrix c20=c20/800000;
matrix m20=m20/200000;
matrix c25=c25/750000;
matrix m25=m25/250000;
matrix c30=c30/700000;
matrix m30=m30/300000;
matrix c35=c35/650000;
matrix m35=m35/350000;
matrix c40=c40/600000;
matrix m40=m40/400000;
matrix c45=c45/550000;
matrix m45=m45/450000;
matrix c50=c50/500000;
matrix m50=m50/500000;
matrix c55=c55/450000;
matrix m55=m55/550000;
matrix c60=c60/400000;
matrix m60=m60/600000;
matrix c65=c65/350000;
matrix m65=m65/650000;
matrix c70=c70/300000;
matrix m70=m70/700000;
matrix c75=c75/250000;
matrix m75=m75/750000;
matrix c80=c80/200000;
matrix m80=m80/800000;
matrix c85=c85/150000;
matrix m85=m85/850000;
matrix c90=c90/100000;
matrix m90=m90/900000;
matrix c95=c95/50000;
matrix m95=m95/950000;

/*Print the Output*/
matrix list c05;
matrix list mc05;
matrix list m05;
matrix list mm05;
matrix list c10;
matrix list mc10;
matrix list m10;
matrix list mm10;
matrix list c15;
matrix list mc15;
matrix list m15;
matrix list mm15;
matrix list c20;
matrix list mc20;
matrix list m20;
matrix list mm20;
matrix list c25;
matrix list mc25;
matrix list m25;
matrix list mm25;
matrix list c30;
matrix list mc30;
matrix list m30;
matrix list mm30;
matrix list c35;
matrix list mc35;
matrix list m35;
matrix list mm35;
matrix list c40;
matrix list mc40;
matrix list m40;
matrix list mm40;
matrix list c45;
matrix list mc45;
matrix list m45;
matrix list mm45;
matrix list c50;
matrix list mc50;
matrix list m50;
matrix list mm50;
matrix list c55;
matrix list mc55;
matrix list m55;
matrix list mm55;
matrix list c60;
matrix list mc60;
matrix list m60;
matrix list mm60;
matrix list c65;
matrix list mc65;
matrix list m65;
matrix list mm65;
matrix list c70;
matrix list mc70;
matrix list m70;
matrix list mm70;
matrix list c75;
matrix list mc75;
matrix list m75;
matrix list mm75;
matrix list c80;
matrix list mc80;
matrix list m80;
matrix list mm80;
matrix list c85;
matrix list mc85;
matrix list m85;
matrix list mm85;
matrix list c90;
matrix list mc90;
matrix list m90;
matrix list mm90;
matrix list c95;
matrix list mc95;
matrix list m95;
matrix list mm95;
corr t1 t2 t3 t4 t5, m c;
log close;
clear;
For Treatment Group

#delimit;
set mem 200m;
capture log  close;

/*Save the output */
log using "c:\study1\Curran_treatment_0.2.log", replace;

/*Define Covariance Matrix */
/*Curran & Muthen, 1999 */
/*Treatment Group Matrix*/

matrix S4 = (2.0000000, 1.1118034, 1.2236068, 1.3354102, 1.4472136)
1.1118034, 2.8472136, 1.7354102, 2.0472136, 2.3590170
1.2236068, 1.7354102, 4.4944272, 2.7590170, 3.2708204
1.3354102, 2.0472136, 2.7590170, 6.9416408, 4.1826238
1.4472136, 2.3590170, 3.2708204, 4.1826238, 10.188854);
matrix M4 = (1.0000000, 1.9809430, 2.9618860, 3.9428290, 4.9237720);

/*Set Replicable See*/
set seed 1234567;

/*Generate a 1,000,000 Observation Dataset*/
/* 5 Waves of Data*/
corr2data t1-t5, n(1000000) cov(S4) means(M4);

/*Establish a selection variable to make MAR data */
/*Weighted on Time1 (Pretest) score only */
generate scrit01 = -1*t1;

/*Sort Cases by values on selection variable */
/*Note that this is like the phenotypic (i.e., observed variable) sorting of Dolan */
sort scrit01;

/*Now determine extent of MAR missing data*/
/*First initialize variables*/
generate sel00 = 0;
generate sel05 = 0;
generate sel10 = 0;
generate sel15 = 0;
generate sel20 = 0;
generate sel25 = 0;
generate sel30 = 0;
generate sel35 = 0;
generate sel40 = 0;
generate sel45 = 0;
generate sel50 = 0;
generate sel55 = 0;
generate sel60 = 0;
generate sel65 = 0;
generate sel70 = 0;
generate sel75 = 0;
generate sel80 = 0;
generate sel85 = 0;
generate sel90 = 0;
generate sel95 = 0;

/*Then make groups representing different proportions of missing data */
replace sel05=1 if _n <= 50000;
replace sel10=1 if _n <= 100000;
replace sel15=1 if _n <= 150000;
replace sel20=1 if _n <= 200000;
replace sel25=1 if _n <= 250000;
replace sel30=1 if _n <= 300000;
replace sel35=1 if _n <= 350000;
replace sel40=1 if _n <= 400000;
replace sel45=1 if _n <= 450000;
replace sel50=1 if _n <= 500000;
replace sel55=1 if _n <= 550000;
replace sel60=1 if _n <= 600000;
replace sel65=1 if _n <= 650000;
replace sel70=1 if _n <= 700000;
replace sel75=1 if _n <= 750000;
replace sel80=1 if _n <= 800000;
replace sel85=1 if _n <= 850000;
replace sel90=1 if _n <= 900000;
replace sel95=1 if _n <= 950000;

/*Now we make and store some matrices by group */
/* 5% Missing */
matrix accum c05 = t1-t5 if sel05==0, means(mc05) dev noconst;
matrix accum m05 = t1-t5 if sel05==1, means(mm05) dev noconst;

/* 10% Missing */
matrix accum c10 = t1-t5 if sel10==0, means(mc10) dev noconst;
matrix accum m10 = t1-t5 if sel10==1, means(mm10) dev noconst;

/* 15% Missing */
matrix accum c15 = t1-t5 if sel15==0, means(mc15) dev noconst;
matrix accum m15 = t1-t5 if sel15==1, means(mm15) dev noconst;
/* 20% Missing */
matrix accum c20 = t1-t5 if sel20==0, means(mc20) dev noconst;
matrix accum m20 = t1-t5 if sel20==1, means(mm20) dev noconst;

/* 25% Missing */
matrix accum c25 = t1-t5 if sel25==0, means(mc25) dev noconst;
matrix accum m25 = t1-t5 if sel25==1, means(mm25) dev noconst;

/* 30% Missing */
matrix accum c30 = t1-t5 if sel30==0, means(mc30) dev noconst;
matrix accum m30 = t1-t5 if sel30==1, means(mm30) dev noconst;

/* 35% Missing */
matrix accum c35 = t1-t5 if sel35==0, means(mc35) dev noconst;
matrix accum m35 = t1-t5 if sel35==1, means(mm35) dev noconst;

/* 40% Missing */
matrix accum c40 = t1-t5 if sel40==0, means(mc40) dev noconst;
matrix accum m40 = t1-t5 if sel40==1, means(mm40) dev noconst;

/* 45% Missing */
matrix accum c45 = t1-t5 if sel45==0, means(mc45) dev noconst;
matrix accum m45 = t1-t5 if sel45==1, means(mm45) dev noconst;

/* 50% Missing */
matrix accum c50 = t1-t5 if sel50==0, means(mc50) dev noconst;
matrix accum m50 = t1-t5 if sel50==1, means(mm50) dev noconst;

/* 55% Missing */
matrix accum c55 = t1-t5 if sel55==0, means(mc55) dev noconst;
matrix accum m55 = t1-t5 if sel55==1, means(mm55) dev noconst;

/* 60% Missing */
matrix accum c60 = t1-t5 if sel60==0, means(mc60) dev noconst;
matrix accum m60 = t1-t5 if sel60==1, means(mm60) dev noconst;

/* 65% Missing */
matrix accum c65 = t1-t5 if sel65==0, means(mc65) dev noconst;
matrix accum m65 = t1-t5 if sel65==1, means(mm65) dev noconst;

/* 70% Missing */
matrix accum c70 = t1-t5 if sel70==0, means(mc70) dev noconst;
matrix accum m70 = t1-t5 if sel70==1, means(mm70) dev noconst;
matrix accum c75 = t1-t5 if sel75==0, means(mc75) dev noconst;
matrix accum m75 = t1-t5 if sel75==1, means(mm75) dev noconst;

matrix accum c80 = t1-t5 if sel80==0, means(mc80) dev noconst;
matrix accum m80 = t1-t5 if sel80==1, means(mm80) dev noconst;

matrix accum c85 = t1-t5 if sel85==0, means(mc85) dev noconst;
matrix accum m85 = t1-t5 if sel85==1, means(mm85) dev noconst;

matrix accum c90 = t1-t5 if sel90==0, means(mc90) dev noconst;
matrix accum m90 = t1-t5 if sel90==1, means(mm90) dev noconst;

matrix accum c95 = t1-t5 if sel95==0, means(mc95) dev noconst;
matrix accum m95 = t1-t5 if sel95==1, means(mm95) dev noconst;

/*Make Covariance Matrix by Dividing by Sample Size */
matrix c05=c05/950000;
matrix m05=m05/500000;
matrix c10=c10/900000;
matrix m10=m10/100000;
matrix c15=c15/850000;
matrix m15=m15/150000;
matrix c20=c20/800000;
matrix m20=m20/200000;
matrix c25=c25/750000;
matrix m25=m25/250000;
matrix c30=c30/700000;
matrix m30=m30/300000;
matrix c35=c35/650000;
matrix m35=m35/350000;
matrix c40=c40/600000;
matrix m40=m40/400000;
matrix c45=c45/550000;
matrix m45=m45/450000;
matrix c50=c50/500000;
matrix m50=m50/500000;
matrix c55=c55/450000;
matrix m55=m55/550000;
matrix c60=c60/400000;
matrix m60=m60/600000;
matrix c65=c65/350000;
matrix m65=m65/650000;
matrix c70=c70/300000;
matrix m70=m70/700000;
matrix c75=c75/250000;
matrix m75=m75/750000;
matrix c80=c80/200000;
matrix m80=m80/800000;
matrix c85=c85/150000;
matrix m85=m85/850000;
matrix c90=c90/100000;
matrix m90=m90/900000;
matrix c95=c95/500000;
matrix m95=m95/950000;

/*Print the Output*/
matrix list c05;
matrix list mc05;
matrix list m05;
matrix list mm05;
matrix list c10;
matrix list mc10;
matrix list m10;
matrix list mm10;
matrix list c15;
matrix list mc15;
matrix list m15;
matrix list mm15;
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matrix list mm90;
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matrix list mc95;
matrix list m95;
matrix list mm95;
corr t1 t2 t3 t4 t5, m c;
log close;
clear;
APPENDIX B

LISREL CODE FOR STUDY 1

Example of LISREL 8.5 Code using Multiple Group Approach with Incomplete Data (MCAR)

!Study 1 Control Group - MCAR (Complete data on 95% cases)
!Number of groups = 4
da ni=5 no=95 ng=4
la
Time1 Time2 Time3 Time4 Time5
cm
2.0000000
1.1118034 2.8472136
1.2236068 1.7354102 4.4944272
1.3354102 2.0472136 2.7590170 6.9416408
1.4472136 2.3590170 3.2708204 4.1826238 10.188854
me
1.0000000 1.797998 2.595996 3.393994 4.191992
mo ny=5 ne=2 ly=fu,fi ps=sy,fr te=sy,fi ty=fi al=fr
le
Level Shape
va 1.0 ly(1,1) ly(2,1) ly(3,1) ly(4,1) ly(5,1)
va 0.0 ly(1,2)
va 1.0 ly(2,2)
va 2.0 ly(3,2)
va 3.0 ly(4,2)
va 4.0 ly(5,2)
fr te(1,1) te(2,2) te(3,3) te(4,4) te(5,5)
ou ad=off

!Study 1 Control Group - MCAR (Missing data on 5% cases)
da ni=5 no=5
la
Time1 Time2 Time3 Time4 Time5
cm
2.0000000
1.1118034 2.8472136
1.2236068 1.7354102 4.4944272
0 0 0 1
0 0 0 0 1
me
1.0000000 1.797998 2.595996 0 0
mo ny=5 ne=2 ly=fu,fi ps=sy,fr te=sy,fi ty=fi al=fr
le
Level Shape
va 1.0 ly(1,1) ly(2,1) ly(3,1)
va 0.0 ly(1,2)
va 1.0 ly(2,2)
va 2.0 ly(3,2)
fr te(1,1) te(2,2) te(3,3)
eq te(1,1,1) te(1,1)
eq te(1,2,2) te(2,2)
eq te(1,3,3) te(3,3)
va 1.0 te(4,4) te(5,5)
eq ps(1,1,1) ps(1,1)
eq ps(1,1,2) ps(1,2)
eq ps(1,2,2) ps(2,2)
eq al(1,1) al(1)
eq al(1,2) al(2)
ou ad=off

!Treatment Group - MCAR (Complete Data on 95% cases)
da ni=5 no=95
la
Time1 Time2 Time3 Time4 Time5
cm
2.0000000
1.1118034 2.8472136
1.2236068 1.7354102 4.4944272
1.3354102 2.0472136 2.7590170 6.9416408
1.4472136 2.3590170 3.2708204 4.1826238 10.188854
me
1.0000000 1.9810000 2.9620000 3.9430000 4.9240000
mo
ny=5 ne=2 ly=fu,fi ps=sy,fr te=sy,fi ty=fi al=fr
va 1.0 ly(1,1) ly(2,1) ly(3,1) ly(4,1) ly(5,1)
va 0.0 ly(1,2)
va 1.0 ly(2,2)
va 2.0 ly(3,2)
va 3.0 ly(4,2)
va 4.0 ly(5,2)
eq al(1,1) al(1)
eq al(1,2) al(2)
fr te(1,1) te(2,2) te(3,3) te(4,4) te(5,5)
ou

!Treatment Group - MCAR (Missing Data on 5% cases)
da ni=5 no=5
la
Time1 Time2 Time3 Time4 Time5
cm
2.000000
1.1118034 2.8472136
1.2236068 1.7354102 4.4944272
0 0 0 1
0 0 0 0 1
me
1.0000000 1.9810000 2.9620000 0 0
mo ny=5 ne=2 ly=fu,fi ps=sy,fr te=sy,fi ty=fi al=fr
va 1.0 ly(1,1) ly(2,1) ly(3,1)
va 0.0 ly(1,2)
va 1.0 ly(2,2)
va 2.0 ly(3,2)
fr te(1,1) te(2,2) te(3,3)
eq te(3,1,1) te(1,1)
eq te(3,2,2) te(2,2)
eq te(3,3,3) te(3,3)
va 1.0 te(4,4) te(5,5)
eq ps(3,1,1) ps(1,1)
eq ps(3,1,2) ps(1,2)
eq ps(3,2,2) ps(2,2)
eq al(3,1) al(1)
eq al(3,2) al(2)
ou
APPENDIX C

STUDY 1: $F_{\text{Min}}$, CHI-SQUARE AND POWER TABLES

Appendix C contains the following tables:

Table 1. $F_{\text{Min}}$ and Chi-squares for MAR Data by Percentage of Missing data and Sample Size
Table 2. Statistical Power for MAR Data by Percentage of Missing Data and Sample Size
Table 3. $F_{\text{Min}}$ and Chi-square for MCAR data by Percentage of Missing data and Sample Size
Table 4. Statistical Power for MCAR Data by Percentage of Missing Data and Sample Size
Table 5. Proportional Power for MAR Data by Percentage of Missing Data and Sample Size
Table 6. Proportional Power for MCAR Data by Percentage of Missing Data and Sample Size.
Table 7. Percentage Difference in Statistical Power between MCAR and MAR Data
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Table 2. Statistical Power for MAR Data by Percentage of Missing Data and Sample Size

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Table 3. $F_{\text{Min}}$ and Chi-square for MCAR data by Percentage of Missing data and Sample Size

| % Missing | $F_{\text{Min}}$ | 100  | 150  | 200  | 250  | 300  | 350  | 400  | 450  | 500  | 550  | 600  | 650  | 700  | 750  | 800  | 850  | 900  | 950  | 1000 |
|-----------|------------------|------|------|------|------|------|------|------|------|------|------|------|------|------|------|------|------|------|------|------|------|
| 0         | 3.89             | 1.93 | 2.91 | 3.89 | 4.87 | 5.85 | 6.84 | 7.82 | 8.80 | 9.78 | 10.77| 11.75| 12.73| 13.71| 14.70| 15.68| 16.66| 17.64| 18.62| 19.61|
| 5         | 3.8              | 1.88 | 2.84 | 3.80 | 4.76 | 5.72 | 6.68 | 7.64 | 8.60 | 9.56 | 10.52| 11.48| 12.44| 13.40| 14.36| 15.32| 16.27| 17.23| 18.19| 19.15|
| 15        | 3.6              | 1.78 | 2.69 | 3.60 | 4.51 | 5.42 | 6.33 | 7.24 | 8.15 | 9.05 | 9.96 | 10.87| 11.78| 12.69| 13.60| 14.51| 15.42| 16.33| 17.24| 18.15|
| 20        | 3.5              | 1.73 | 2.62 | 3.50 | 4.38 | 5.27 | 6.15 | 7.04 | 7.92 | 8.80 | 9.69 | 10.57| 11.45| 12.34| 13.22| 14.11| 14.99| 15.87| 16.76| 17.64|
| 25        | 3.4              | 1.68 | 2.54 | 3.40 | 4.26 | 5.12 | 6.08 | 7.03 | 7.98 | 8.85 | 9.71 | 10.57| 11.43| 12.30| 13.16| 14.03| 14.91| 15.79| 16.66| 17.54|
| 30        | 3.3              | 1.63 | 2.47 | 3.30 | 4.13 | 5.04 | 6.01 | 7.00 | 7.91 | 8.80 | 9.67 | 10.58| 11.44| 12.29| 13.14| 14.00| 14.87| 15.73| 16.59| 17.45|
| 40        | 3.1              | 1.53 | 2.32 | 3.10 | 3.88 | 4.75 | 5.64 | 6.55 | 7.46 | 8.38 | 9.29 | 10.19| 11.01| 11.82| 12.62| 13.41| 14.20| 15.00| 15.80| 16.59|
| 50        | 2.9              | 1.44 | 2.17 | 2.90 | 3.65 | 4.53 | 5.42 | 6.33 | 7.25 | 8.17 | 9.09 | 10.01| 10.89| 11.77| 12.64| 13.50| 14.35| 15.20| 16.05| 16.90|
| 55        | 2.79             | 1.38 | 2.09 | 2.79 | 3.49 | 4.29 | 5.19 | 6.10 | 7.02 | 7.96 | 8.89 | 9.83 | 10.75| 11.67| 12.59| 13.50| 14.39| 15.28| 16.15| 17.02|
| 60        | 2.69             | 1.33 | 2.01 | 2.69 | 3.37 | 4.17 | 5.02 | 5.92 | 6.84 | 7.76 | 8.69 | 9.63 | 10.55| 11.47| 12.39| 13.30| 14.20| 15.12| 16.04| 16.95|
| 65        | 2.59             | 1.28 | 1.94 | 2.59 | 3.29 | 4.05 | 4.86 | 5.78 | 6.69 | 7.61 | 8.54 | 9.48 | 10.41| 11.34| 12.27| 13.19| 14.09| 15.00| 15.91| 16.82|
| 70        | 2.49             | 1.23 | 1.86 | 2.49 | 3.12 | 3.83 | 4.66 | 5.57 | 6.48 | 7.41 | 8.34 | 9.28 | 10.21| 11.14| 12.06| 13.00| 13.91| 14.82| 15.73| 16.65|
| 75        | 2.39             | 1.18 | 1.79 | 2.39 | 2.99 | 3.60 | 4.42 | 5.25 | 6.18 | 7.11 | 8.06 | 9.00 | 9.94 | 10.88| 11.81| 12.73| 13.66| 14.59| 15.52| 16.45|
| 80        | 2.29             | 1.13 | 1.71 | 2.29 | 2.87 | 3.54 | 4.32 | 5.18 | 6.13 | 7.07 | 8.02 | 8.97 | 9.92 | 10.87| 11.82| 12.77| 13.71| 14.65| 15.59| 16.53|
| 85        | 2.19             | 1.08 | 1.64 | 2.19 | 2.74 | 3.33 | 4.05 | 4.88 | 5.82 | 6.77 | 7.72 | 8.68 | 9.64 | 10.59| 11.56| 12.51| 13.46| 14.41| 15.36| 16.31|
| 90        | 2.09             | 1.03 | 1.56 | 2.09 | 2.62 | 3.15 | 3.87 | 4.70 | 5.63 | 6.57 | 7.52 | 8.48 | 9.45 | 10.40| 11.36| 12.32| 13.28| 14.23| 15.19| 16.15|
| 95        | 1.99             | 0.98 | 1.49 | 1.99 | 2.49 | 3.00 | 3.50 | 4.00 | 4.50 | 5.01 | 5.51 | 6.01 | 6.51 | 7.02 | 7.52 | 8.02 | 8.52 | 9.03 | 9.53 | 10.03|
Table 4. Statistical Power for MCAR Data by Percentage of Missing Data and Sample Size

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APPENDIX D

STUDY 2A: $F_{\text{Min}}$, CHI-SQUARE AND POWER TABLES AT VARYING RELIABILITIES

Appendix D contains the following tables:

Table 8: $F_{\text{Min}}$ and Chi-squares for Models with Varying Reliabilities, Sample Sizes and Percentage of Missing Data.

Table 9: Statistical Power of Models with Varying Reliabilities, Sample Sizes and Percentage of Missing Data.

Table 10: Proportional Power of Models with Varying Reliabilities, Sample Sizes and Percentage of Missing Data.
Table 8. $F_{\text{Min}}$ and Chi-squares for Models with Varying Reliabilities, Sample Sizes and Percentage of Missing Data.

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Table 8 (continued). $F_{\text{Min}}$ and Chi-squares for Models with Varying Reliabilities, Sample Sizes and Percentage of Missing Data

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<td>7.11</td>
</tr>
<tr>
<td></td>
<td>30%</td>
<td>0.0167</td>
<td>5.90</td>
</tr>
<tr>
<td></td>
<td>50%</td>
<td>0.0146</td>
<td>5.06</td>
</tr>
<tr>
<td></td>
<td>70%</td>
<td>0.0126</td>
<td>4.21</td>
</tr>
<tr>
<td>Reliability</td>
<td>Missing Data</td>
<td>100</td>
<td>150</td>
</tr>
<tr>
<td>-------------</td>
<td>--------------</td>
<td>-----</td>
<td>-----</td>
</tr>
<tr>
<td>0.3</td>
<td>0%</td>
<td>0.20</td>
<td>0.28</td>
</tr>
<tr>
<td></td>
<td>30%</td>
<td>0.18</td>
<td>0.24</td>
</tr>
<tr>
<td></td>
<td>50%</td>
<td>0.16</td>
<td>0.22</td>
</tr>
<tr>
<td></td>
<td>70%</td>
<td>0.14</td>
<td>0.19</td>
</tr>
<tr>
<td>0.5</td>
<td>0%</td>
<td>0.28</td>
<td>0.40</td>
</tr>
<tr>
<td></td>
<td>30%</td>
<td>0.25</td>
<td>0.35</td>
</tr>
<tr>
<td></td>
<td>50%</td>
<td>0.22</td>
<td>0.31</td>
</tr>
<tr>
<td></td>
<td>70%</td>
<td>0.20</td>
<td>0.28</td>
</tr>
<tr>
<td>0.7</td>
<td>0%</td>
<td>0.37</td>
<td>0.52</td>
</tr>
<tr>
<td></td>
<td>30%</td>
<td>0.33</td>
<td>0.47</td>
</tr>
<tr>
<td></td>
<td>50%</td>
<td>0.31</td>
<td>0.43</td>
</tr>
<tr>
<td></td>
<td>70%</td>
<td>0.28</td>
<td>0.39</td>
</tr>
</tbody>
</table>
Table 10. Proportional Power of Models with Varying Reliabilities, Sample Sizes and Percentage of Missing Data.

<table>
<thead>
<tr>
<th>Reliability</th>
<th>Missing Data</th>
<th>Sample Size</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.3</td>
<td>30%</td>
<td>0.90 0.90 0.91 0.92 0.94 0.95 0.96 0.97 0.97 0.98 0.98 0.99 0.99 1.00 1.00 1.00 1.00 1.00</td>
</tr>
<tr>
<td></td>
<td>50%</td>
<td>0.83 0.83 0.85 0.86 0.88 0.90 0.92 0.93 0.95 0.96 0.97 0.97 0.98 0.99 0.99 0.99 1.00 1.00</td>
</tr>
<tr>
<td></td>
<td>70%</td>
<td>0.75 0.76 0.78 0.80 0.82 0.85 0.87 0.89 0.91 0.93 0.94 0.95 0.96 0.97 0.98 0.98 0.99 0.99</td>
</tr>
<tr>
<td>0.5</td>
<td>30%</td>
<td>0.87 0.87 0.88 0.89 0.91 0.92 0.93 0.93 0.94 0.95 0.96 0.97 0.97 0.98 0.98 0.98 0.99 0.99</td>
</tr>
<tr>
<td></td>
<td>50%</td>
<td>0.79 0.78 0.79 0.80 0.81 0.83 0.85 0.86 0.88 0.89 0.91 0.92 0.93 0.94 0.95 0.96 0.96 0.97</td>
</tr>
<tr>
<td></td>
<td>70%</td>
<td>0.70 0.69 0.70 0.71 0.72 0.74 0.76 0.78 0.80 0.82 0.84 0.86 0.88 0.89 0.91 0.92 0.93 0.94</td>
</tr>
<tr>
<td>0.7</td>
<td>30%</td>
<td>0.87 0.86 0.86 0.86 0.86 0.87 0.87 0.88 0.89 0.89 0.90 0.91 0.92 0.92 0.93 0.94 0.94 0.95</td>
</tr>
<tr>
<td></td>
<td>50%</td>
<td>0.78 0.76 0.75 0.76 0.76 0.77 0.78 0.79 0.80 0.81 0.82 0.83 0.84 0.85 0.87 0.88 0.89 0.90</td>
</tr>
<tr>
<td></td>
<td>70%</td>
<td>0.69 0.66 0.65 0.65 0.66 0.66 0.67 0.68 0.69 0.71 0.72 0.73 0.75 0.76 0.78 0.79 0.81 0.82</td>
</tr>
</tbody>
</table>
APPENDIX E

STATA CODE FOR STUDY 2 WITH AN AUXILIARY VARIABLE

STATA (Version 7) Code to Generate the Covariance Matrices and Mean Vectors for MAR Data

with Auxiliary Variable of 0.1 Correlation with Pre-test Data

For Control Group:

#delimit;
set mem 200m;
capture log close;

/*Save the output */
log using "c:\study2\covariates\control_0.1 covariate.log", replace;

/*Define Covariance Matrix */
/*Curran & Muthen, 1999 */
/*Control Group Matrix*/
/*Auxiliary variable correlated at 0.1 with Time 1 Data*/
matrix S4 = (2.0000000, 1.1118034, 1.2236068, 1.3354102, 1.4472136, 0.1414200\1.1118034, 2.8472136, 1.7354102, 2.0472136, 2.3590170, 0.000000\1.2236068, 1.7354102, 4.4944272, 2.7590170, 3.2708204, 0.000000\1.3354102, 2.0472136, 2.7590170, 6.9416408, 4.1826238, 0.000000\1.4472136, 2.3590170, 3.2708204, 4.1826238, 10.188854, 0.000000\0.1414200, 0.000000, 0.000000, 0.000000, 00.000000, 1.000000);

matrix M4 = (1.00000, 1.797998, 2.595996, 3.393994, 4.191992, 0.000000);

/*Set Replicable See*/
set seed 1234567;

/*Generate a 1,000,000 Observation Dataset*/
/* 5 Waves of Data*/
corr2data t1-t5 cov, n(1000000) cov(S4) means(M4);

/*Establish a selection variable to make MAR data */
/*Weighted on Time1 (Pretest) score only */
generate scrit01 = -1*t1;

/*Sort Cases by values on selection variable */
/*Note that this is like the phenotypic (i.e., observed variable) sorting of Dolan */
sort scrit01;

/*Now determine extent of MAR missing data*/
/*First initialize variables*/
generate sel00 = 0;
generate sel05 = 0;
generate sel10 = 0;
generate sel15 = 0;
generate sel20 = 0;
generate sel25 = 0;
generate sel30 = 0;
generate sel35 = 0;
generate sel40 = 0;
generate sel45 = 0;
generate sel50 = 0;
generate sel55 = 0;
generate sel60 = 0;
generate sel65 = 0;
generate sel70 = 0;
generate sel75 = 0;
generate sel80 = 0;
generate sel85 = 0;
generate sel90 = 0;
generate sel95 = 0;

/*Then make groups representing different proportions of missing data */
replace sel05=1 if _n <= 50000;
replace sel10=1 if _n <= 100000;
replace sel15=1 if _n <= 150000;
replace sel20=1 if _n <= 200000;
replace sel25=1 if _n <= 250000;
replace sel30=1 if _n <= 300000;
replace sel35=1 if _n <= 350000;
replace sel40=1 if _n <= 400000;
replace sel45=1 if _n <= 450000;
replace sel50=1 if _n <= 500000;
replace sel55=1 if _n <= 550000;
replace sel60=1 if _n <= 600000;
replace sel65=1 if _n <= 650000;
replace sel70=1 if _n <= 700000;
replace sel75=1 if _n <= 750000;
replace sel80=1 if _n <= 800000;
replace sel85=1 if _n <= 850000;
replace sel90=1 if _n <= 900000;
replace sel95=1 if _n <= 950000;
/*Now we make and store some matrices by group */
/* 5% Missing */
matrix accum c05 = t1-t5 cov if sel05==0, means(mc05) dev noconst;
matrix accum m05 = t1-t5 cov if sel05==1, means(mm05) dev noconst;

/* 10% Missing */
matrix accum c10 = t1-t5 cov if sel10==0, means(mc10) dev noconst;
matrix accum m10 = t1-t5 cov if sel10==1, means(mm10) dev noconst;

/* 15% Missing */
matrix accum c15 = t1-t5 cov if sel15==0, means(mc15) dev noconst;
matrix accum m15 = t1-t5 cov if sel15==1, means(mm15) dev noconst;

/* 20% Missing */
matrix accum c20 = t1-t5 cov if sel20==0, means(mc20) dev noconst;
matrix accum m20 = t1-t5 cov if sel20==1, means(mm20) dev noconst;

/* 25% Missing */
matrix accum c25 = t1-t5 cov if sel25==0, means(mc25) dev noconst;
matrix accum m25 = t1-t5 cov if sel25==1, means(mm25) dev noconst;

/* 30% Missing */
matrix accum c30 = t1-t5 cov if sel30==0, means(mc30) dev noconst;
matrix accum m30 = t1-t5 cov if sel30==1, means(mm30) dev noconst;

/* 35% Missing */
matrix accum c35 = t1-t5 cov if sel35==0, means(mc35) dev noconst;
matrix accum m35 = t1-t5 cov if sel35==1, means(mm35) dev noconst;

/* 40% Missing */
matrix accum c40 = t1-t5 cov if sel40==0, means(mc40) dev noconst;
matrix accum m40 = t1-t5 cov if sel40==1, means(mm40) dev noconst;

/* 45% Missing */
matrix accum c45 = t1-t5 cov if sel45==0, means(mc45) dev noconst;
matrix accum m45 = t1-t5 cov if sel45==1, means(mm45) dev noconst;

/* 50% Missing */
matrix accum c50 = t1-t5 cov if sel50==0, means(mc50) dev noconst;
matrix accum m50 = t1-t5 cov if sel50==1, means(mm50) dev noconst;

/* 55% Missing */
matrix accum c55 = t1-t5 cov if sel55==0, means(mc55) dev noconst;
matrix accum m55 = t1-t5 cov if sel55==1, means(mm55) dev noconst;

/* 60% Missing */
matrix accum c60 = t1-t5 cov if sel60==0, means(mc60) dev noconst;
matrix accum m60 = t1-t5 cov if sel60==1, means(mm60) dev noconst;

/* 65% Missing */
matrix accum c65 = t1-t5 cov if sel65==0, means(mc65) dev noconst;
matrix accum m65 = t1-t5 cov if sel65==1, means(mm65) dev noconst;

/* 70% Missing */
matrix accum c70 = t1-t5 cov if sel70==0, means(mc70) dev noconst;
matrix accum m70 = t1-t5 cov if sel70==1, means(mm70) dev noconst;

/* 75% Missing */
matrix accum c75 = t1-t5 cov if sel75==0, means(mc75) dev noconst;
matrix accum m75 = t1-t5 cov if sel75==1, means(mm75) dev noconst;

/* 80% Missing */
matrix accum c80 = t1-t5 cov if sel80==0, means(mc80) dev noconst;
matrix accum m80 = t1-t5 cov if sel80==1, means(mm80) dev noconst;

/* 85% Missing */
matrix accum c85 = t1-t5 cov if sel85==0, means(mc85) dev noconst;
matrix accum m85 = t1-t5 cov if sel85==1, means(mm85) dev noconst;

/* 90% Missing */
matrix accum c90 = t1-t5 cov if sel90==0, means(mc90) dev noconst;
matrix accum m90 = t1-t5 cov if sel90==1, means(mm90) dev noconst;

/* 95% Missing */
matrix accum c95 = t1-t5 cov if sel95==0, means(mc95) dev noconst;
matrix accum m95 = t1-t5 cov if sel95==1, means(mm95) dev noconst;

/*Make Covariance Matrix by Dividing by Sample Size */
matrix c05=c05/950000;
matrix m05=m05/50000;
matrix c10=c10/900000;
matrix m10=m10/100000;
matrix c15=c15/850000;
matrix m15=m15/150000;
matrix c20=c20/800000;
matrix m20=m20/200000;
matrix c25=c25/750000;
matrix m25=m25/250000;
matrix c30=c30/700000;
matrix m30=m30/300000;
matrix c35=c35/650000;
matrix m35=m35/350000;
matrix c40=c40/600000;
matrix m40=m40/400000;
matrix c45=c45/550000;
matrix m45=m45/450000;
matrix c50=c50/500000;
matrix m50=m50/500000;
matrix c55=c55/450000;
matrix m55=m55/550000;
matrix c60=c60/400000;
matrix m60=m60/600000;
matrix c65=c65/350000;
matrix m65=m65/650000;
matrix c70=c70/300000;
matrix m70=m70/700000;
matrix c75=c75/250000;
matrix m75=m75/750000;
matrix c80=c80/200000;
matrix m80=m80/800000;
matrix c85=c85/150000;
matrix m85=m85/850000;
matrix c90=c90/100000;
matrix m90=m90/900000;
matrix c95=c95/50000;
matrix m95=m95/950000;

/*Print the Output*/
matrix list c05;
matrix list mc05;
matrix list m05;
matrix list mm05;
matrix list c10;
matrix list mc10;
matrix list m10;
matrix list mm10;
matrix list c15;
matrix list mc15;
matrix list m15;
matrix list mm15;
matrix list c20;
matrix list mc20;
matrix list m20;
matrix list mm20;
matrix list c25;
matrix list mc25;
matrix list m25;
matrix list mm25;
matrix list c30;
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matrix list m70;
matrix list mm70;
matrix list c75;
matrix list mc75;
matrix list m75;
matrix list mm75;
matrix list c80;
matrix list mc80;
matrix list m80;
matrix list mm80;
matrix list c85;
matrix list mc85;
matrix list m85;
matrix list mm85;
matrix list c90;
matrix list mc90;
matrix list m90;
matrix list mm90;
matrix list c95;
matrix list mc95;
matrix list m95;
matrix list mm95;

corr t1 t2 t3 t4 t5 cov, m c;

log close;
clear;
For Treatment Group:

#delimit;
set mem 200m;
capture log  close;

/*Save the output */
log using "c:\study\covariates\Treatment_0.1 Covariate.log", replace;

/*Define Covariance Matrix */
/*Curran & Muthen, 1999 */
/*Treatment Group Matrix*/

matrix S4 =
2.0000000, 1.1118034, 1.3354102, 1.4472136, 0.1414200\ 
1.1118034, 2.8472136, 1.7354102, 2.0472136, 0.0000000\ 
1.2236068, 1.7354102, 4.4944272, 2.7590170, 3.2708204, 0.0000000\ 
1.3354102, 2.0472136, 2.7590170, 6.9416408, 4.1826238, 0.0000000\ 
1.4472136, 2.3590170, 3.2708204, 4.1826238, 10.188854, 0.0000000\ 
0.1414200, 0.0000000, 0.0000000, 0.0000000, 0.0000000, 1.0000000);
matrix M4 =
1.0000000, 1.9809430, 2.9618860, 3.9428290, 4.9237720, 0.0000000);

/*Set Replicable See*/
set seed 1234567;

/*Generate a 1,000,000 Observation Dataset*/
/* 5 Waves of Data & Covariance*/
corr2data t1-t5 cov, n(1000000) cov(S4) means(M4);

/*Establish a selection variable to make MAR data */
/*Weighted on Time1 (Pretest) score only */
generate scrit01 = -1*t1;

/*Sort Cases by values on selection variable */
/*Note that this is like the phenotypic (i.e., observed variable) sorting of Dolan */
sort scrit01;

/*Now determine extent of MAR missing data*/
/*First initialize variables*/
generate sel00 = 0;
generate sel05 = 0;
generate sel10 = 0;
generate sel15 = 0;
generate sel20 = 0;
generate sel25 = 0;
generate sel30 = 0;
generate sel35 = 0;
generate sel40 = 0;
generate sel45 = 0;
generate sel50 = 0;
generate sel55 = 0;
generate sel60 = 0;
generate sel65 = 0;
generate sel70 = 0;
generate sel75 = 0;
generate sel80 = 0;
generate sel85 = 0;
generate sel90 = 0;
generate sel95 = 0;

/*Then make groups representing different proportions of missing data */
replace sel05=1 if _n <= 50000;
replace sel10=1 if _n <= 100000;
replace sel15=1 if _n <= 150000;
replace sel20=1 if _n <= 200000;
replace sel25=1 if _n <= 250000;
replace sel30=1 if _n <= 300000;
replace sel35=1 if _n <= 350000;
replace sel40=1 if _n <= 400000;
replace sel45=1 if _n <= 450000;
replace sel50=1 if _n <= 500000;
replace sel55=1 if _n <= 550000;
replace sel60=1 if _n <= 600000;
replace sel65=1 if _n <= 650000;
replace sel70=1 if _n <= 700000;
replace sel75=1 if _n <= 750000;
replace sel80=1 if _n <= 800000;
replace sel85=1 if _n <= 850000;
replace sel90=1 if _n <= 900000;
replace sel95=1 if _n <= 950000;

/*Now we make and store some matrices by group */
/* 5% Missing */
matrix accum c05 = t1-t5 cov if sel05==0, means(mc05) dev noconst;
matrix accum m05 = t1-t5 cov if sel05==1, means(mm05) dev noconst;

/* 10% Missing */
matrix accum c10 = t1-t5 cov if sel10==0, means(mc10) dev noconst;
matrix accum m10 = t1-t5 cov if sel10==1, means(mm10) dev noconst;

/* 15% Missing */
matrix accum c15 = t1-t5 cov if sel15==0, means(mc15) dev noconst;
matrix accum m15 = t1-t5 cov if sel15==1, means(mm15) dev noconst;

/* 20% Missing */
matrix accum c20 = t1-t5 cov if sel20==0, means(mc20) dev noconst;
matrix accum m20 = t1-t5 cov if sel20==1, means(mm20) dev noconst;

/* 25% Missing */
matrix accum c25 = t1-t5 cov if sel25==0, means(mc25) dev noconst;
matrix accum m25 = t1-t5 cov if sel25==1, means(mm25) dev noconst;

/* 30% Missing */
matrix accum c30 = t1-t5 cov if sel30==0, means(mc30) dev noconst;
matrix accum m30 = t1-t5 cov if sel30==1, means(mm30) dev noconst;

/* 35% Missing */
matrix accum c35 = t1-t5 cov if sel35==0, means(mc35) dev noconst;
matrix accum m35 = t1-t5 cov if sel35==1, means(mm35) dev noconst;

/* 40% Missing */
matrix accum c40 = t1-t5 cov if sel40==0, means(mc40) dev noconst;
matrix accum m40 = t1-t5 cov if sel40==1, means(mm40) dev noconst;

/* 45% Missing */
matrix accum c45 = t1-t5 cov if sel45==0, means(mc45) dev noconst;
matrix accum m45 = t1-t5 cov if sel45==1, means(mm45) dev noconst;

/* 50% Missing */
matrix accum c50 = t1-t5 cov if sel50==0, means(mc50) dev noconst;
matrix accum m50 = t1-t5 cov if sel50==1, means(mm50) dev noconst;

/* 55% Missing */
matrix accum c55 = t1-t5 cov if sel55==0, means(mc55) dev noconst;
matrix accum m55 = t1-t5 cov if sel55==1, means(mm55) dev noconst;

/* 60% Missing */
matrix accum c60 = t1-t5 cov if sel60==0, means(mc60) dev noconst;
matrix accum m60 = t1-t5 cov if sel60==1, means(mm60) dev noconst;

/* 65% Missing */
matrix accum c65 = t1-t5 cov if sel65==0, means(mc65) dev noconst;
matrix accum m65 = t1-t5 cov if sel65==1, means(mm65) dev noconst;

/* 70% Missing */
matrix accum c70 = t1-t5 cov if sel70==0, means(mc70) dev noconst;
matrix accum m70 = t1-t5 cov if sel70==1, means(mm70) dev noconst;
/* 75% Missing */
matrix accum c75 = t1-t5 cov if sel75==0, means(mc75) dev noconst;
matrix accum m75 = t1-t5 cov if sel75==1, means(mm75) dev noconst;

/* 80% Missing */
matrix accum c80 = t1-t5 cov if sel80==0, means(mc80) dev noconst;
matrix accum m80 = t1-t5 cov if sel80==1, means(mm80) dev noconst;

/* 85% Missing */
matrix accum c85 = t1-t5 cov if sel85==0, means(mc85) dev noconst;
matrix accum m85 = t1-t5 cov if sel85==1, means(mm85) dev noconst;

/* 90% Missing */
matrix accum c90 = t1-t5 cov if sel90==0, means(mc90) dev noconst;
matrix accum m90 = t1-t5 cov if sel90==1, means(mm90) dev noconst;

/* 95% Missing */
matrix accum c95 = t1-t5 cov if sel95==0, means(mc95) dev noconst;
matrix accum m95 = t1-t5 cov if sel95==1, means(mm95) dev noconst;

/* Make Covariance Matrix by Dividing by Sample Size */
matrix c05=c05/950000;
matrix m05=m05/500000;
matrix c10=c10/900000;
matrix m10=m10/100000;
matrix c15=c15/850000;
matrix m15=m15/150000;
matrix c20=c20/800000;
matrix m20=m20/200000;
matrix c25=c25/750000;
matrix m25=m25/250000;
matrix c30=c30/700000;
matrix m30=m30/300000;
matrix c35=c35/650000;
matrix m35=m35/350000;
matrix c40=c40/600000;
matrix m40=m40/400000;
matrix c45=c45/550000;
matrix m45=m45/450000;
matrix c50=c50/500000;
matrix m50=m50/500000;
matrix c55=c55/450000;
matrix m55=m55/550000;
matrix c60=c60/400000;
matrix m60=m60/600000;
matrix c65=c65/350000;
matrix m65=m65/650000;
matrix c70=c70/300000;
matrix m70=m70/700000;
matrix c75=c75/250000;
matrix m75=m75/750000;
matrix c80=c80/200000;
matrix m80=m80/800000;
matrix c85=c85/150000;
matrix m85=m85/850000;
matrix c90=c90/100000;
matrix m90=m90/900000;
matrix c95=c95/50000;
matrix m95=m95/950000;

/*Print the Output*/
matrix list c05;
matrix list mc05;
matrix list m05;
matrix list mm05;
matrix list c10;
matrix list mc10;
matrix list m10;
matrix list mm10;
matrix list c15;
matrix list mc15;
matrix list m15;
matrix list mm15;
matrix list c20;
matrix list mc20;
matrix list m20;
matrix list mm20;
matrix list c25;
matrix list mc25;
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matrix list mm60;
matrix list c65;
matrix list mc65;
matrix list m65;
matrix list mm65;
matrix list c70;
matrix list mc70;
matrix list m70;
matrix list mm70;
matrix list c75;
matrix list mc75;
matrix list m75;
matrix list mm75;
matrix list c80;
matrix list mc80;
matrix list m80;
matrix list mm80;
matrix list c85;
matrix list mc85;
matrix list m85;
matrix list mm85;
matrix list c90;
matrix list mc90;
matrix list m90;
matrix list mm90;
matrix list c95;
matrix list mc95;
matrix list m95;
matrix list mm95;
corr t1 t2 t3 t4 t5 cov, m c;
log close;
clear;
APPENDIX F

STUDY 2B: \( F_{\text{MIN}} \), CHI-SQUARE AND POWER TABLES FOR MODELS WITH AN AUXILIARY VARIABLE UNDER MCAR DATA CONDITIONS

Appendix F contains the following tables:

Table 11. \( F_{\text{MIN}} \) and Chi-square statistic for Models with Covariate = 0.1 under MCAR Data conditions.

Table 12. Statistical Power for Models with Covariate = 0.1 under MCAR Data conditions.

Table 13. \( F_{\text{MIN}} \) and Chi-square statistic for Models with Covariate = 0.3 under MCAR Data conditions.

Table 14. Statistical Power for Models with Covariate = 0.3 under MCAR Data conditions.

Table 15. \( F_{\text{MIN}} \) and Chi-square statistic for Models with Covariate = 0.5 under MCAR Data conditions.

Table 16. Statistical Power for Models with Covariate = 0.5 under MCAR Data conditions.

Table 17. \( F_{\text{MIN}} \) and Chi-square statistic for Models with Covariate = 0.7 under MCAR Data conditions.

Table 18. Statistical Power for Models with Covariate = 0.7 under MCAR Data conditions.
Table 11. F_{min} and Chi-square Statistic for Models with Covariate = 0.1 under MCAR data condition.

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Table 13. $F_{\text{Min}}$ and Chi-square Statistic for Models with Covariate = 0.3 under MCAR data condition.

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</table>

The table shows the $F_{\text{Min}}$ and Chi-square Statistic values for different sample sizes under the MCAR data condition. The values are listed for each percentage of missing data. The table highlights the statistical comparison between models with varying covariate values under the condition of Missing Completely at Random (MCAR).
Table 14. Statistical Power for Models with Covariate = 0.3 under MCAR data condition.

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<tr>
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</tr>
<tr>
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</tr>
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</tr>
<tr>
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</tr>
<tr>
<td>60</td>
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</tr>
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<tr>
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<tr>
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</tr>
</tbody>
</table>
Table 15. $F_{\text{Min}}$ and Chi-square Statistic for Models with Covariate = 0.5 under MCAR data condition.

<table>
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<th>$F_{\text{Min}}$</th>
<th>Sample Size</th>
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Table 16. Statistical Power for Models with Covariate = 0.5 under MCAR data condition.

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Table 17. $F_{\text{Min}}$ and Chi-square Statistic for Models with Covariate = 0.7 under MCAR data condition.

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Table 18. Statistical Power for Models with Covariate = 0.7 under MCAR data condition.

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APPENDIX G

STUDY 2B: $F_{\text{MIN}}$, CHI-SQUARE AND POWER TABLES FOR MODELS WITH AN AUXILIARY VARIABLE UNDER MAR DATA CONDITIONS

Appendix G contains the following tables:

Table 19. $F_{\text{MIN}}$ and Chi-square statistic for Models with Covariate = 0.1 under MAR Data conditions.

Table 20. Statistical Power for Models with Covariate = 0.1 under MAR Data conditions.

Table 21. $F_{\text{MIN}}$ and Chi-square statistic for Models with Covariate = 0.3 under MAR Data conditions.

Table 22. Statistical Power for Models with Covariate = 0.3 under MAR Data conditions.

Table 23. $F_{\text{MIN}}$ and Chi-square statistic for Models with Covariate = 0.5 under MAR Data conditions.

Table 24. Statistical Power for Models with Covariate = 0.5 under MAR Data conditions.

Table 25. $F_{\text{MIN}}$ and Chi-square statistic for Models with Covariate = 0.7 under MAR Data conditions.

Table 26. Statistical Power for Models with Covariate = 0.7 under MAR Data conditions.
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Table 19. F_{Min} and Chi-square statistic for Models with Covariate = 0.1 under MAR Data conditions.
Table 20. Statistical Power for Models with Covariate = 0.1 under MAR Data conditions.

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Table 21. $F_{\text{Min}}$ and Chi-square statistic for Models with Covariate = 0.3 under MAR Data conditions.

| % Missing | $F_{\text{Min}}$ | 100  | 150  | 200  | 250  | 300  | 350  | 400  | 450  | 500  | 550  | 600  | 650  | 700  | 750  | 800  | 850  | 900  | 950  | 1000 |
|-----------|-----------------|------|------|------|------|------|------|------|------|------|------|------|------|------|------|------|------|------|------|------|------|
| 0         | 0.02            | 1.99 | 3.00 | 4.01 | 5.03 | 6.04 | 7.05 | 8.07 | 9.08 | 10.09| 11.11| 12.12| 13.13| 14.15| 15.16| 16.17| 17.19| 18.20| 19.21| 20.23|
| 5         | 0.02            | 1.94 | 2.94 | 3.93 | 4.92 | 5.91 | 6.90 | 7.90 | 8.89 | 9.88 | 10.87| 11.86| 12.86| 13.85| 14.84| 15.83| 16.82| 17.82| 18.81| 19.80|
| 10        | 0.02            | 1.87 | 2.83 | 3.79 | 4.74 | 5.70 | 6.65 | 7.61 | 8.57 | 9.52 | 10.48| 11.43| 12.39| 13.35| 14.30| 15.26| 16.21| 17.17| 18.13| 19.08|
| 15        | 0.02            | 1.80 | 2.72 | 3.64 | 4.55 | 5.47 | 6.39 | 7.31 | 8.23 | 9.14 | 10.06| 10.98| 11.90| 12.82| 13.73| 14.65| 15.57| 16.49| 17.41| 18.32|
| 20        | 0.02            | 1.72 | 2.60 | 3.48 | 4.36 | 5.24 | 6.12 | 7.00 | 7.88 | 8.75 | 9.63 | 10.51| 11.39| 12.27| 13.15| 14.03| 14.91| 15.79| 16.67| 17.54|
| 25        | 0.02            | 1.65 | 2.49 | 3.33 | 4.17 | 5.01 | 5.85 | 6.69 | 7.53 | 8.37 | 9.21 | 10.05| 10.89| 11.73| 12.57| 13.41| 14.25| 15.10| 15.94| 16.78|
| 30        | 0.02            | 1.57 | 2.38 | 3.18 | 3.98 | 4.78 | 5.59 | 6.39 | 7.19 | 7.99 | 8.80 | 9.60 | 10.40| 11.20| 12.01| 12.81| 13.61| 14.41| 15.22| 16.02|
| 35        | 0.02            | 1.50 | 2.27 | 3.03 | 3.80 | 4.57 | 5.33 | 6.10 | 6.86 | 7.63 | 8.40 | 9.16 | 9.93 | 10.69| 11.46| 12.23| 12.99| 13.76| 14.52| 15.29|
| 40        | 0.01            | 1.44 | 2.17 | 2.90 | 3.63 | 4.37 | 5.10 | 5.83 | 6.56 | 7.30 | 8.03 | 8.76 | 9.49 | 10.23| 10.96| 11.69| 12.42| 13.16| 13.89| 14.62|
| 45        | 0.01            | 1.37 | 2.07 | 2.78 | 3.48 | 4.18 | 4.88 | 5.58 | 6.28 | 6.98 | 7.68 | 8.38 | 9.08 | 9.79 | 10.49| 11.19| 11.89| 12.59| 13.29| 13.99|
| 50        | 0.01            | 1.32 | 1.99 | 2.66 | 3.34 | 4.01 | 4.68 | 5.35 | 6.03 | 6.70 | 7.37 | 8.04 | 8.72 | 9.39 | 10.06| 10.73| 11.41| 12.08| 12.75| 13.42|
| 55        | 0.01            | 1.27 | 1.92 | 2.56 | 3.21 | 3.86 | 4.50 | 5.15 | 5.80 | 6.44 | 7.09 | 7.74 | 8.39 | 9.03 | 9.68 | 10.33| 10.97| 11.62| 12.27| 12.91|
| 60        | 0.01            | 1.22 | 1.85 | 2.47 | 3.09 | 3.72 | 4.34 | 4.96 | 5.59 | 6.21 | 6.83 | 7.46 | 8.08 | 8.70 | 9.33 | 9.95 | 10.57| 11.20| 11.82| 12.45|
| 65        | 0.01            | 1.18 | 1.78 | 2.39 | 2.99 | 3.59 | 4.20 | 4.80 | 5.40 | 6.01 | 6.61 | 7.21 | 7.81 | 8.42 | 9.02 | 9.62 | 10.23| 10.83| 11.43| 12.04|
| 70        | 0.01            | 1.15 | 1.73 | 2.31 | 2.90 | 3.48 | 4.07 | 4.65 | 5.24 | 5.82 | 6.41 | 6.99 | 7.58 | 8.16 | 8.74 | 9.33 | 9.91 | 10.50| 11.08| 11.67|
| 75        | 0.01            | 1.11 | 1.68 | 2.25 | 2.81 | 3.38 | 3.95 | 4.52 | 5.08 | 5.65 | 6.22 | 6.79 | 7.35 | 7.92 | 8.49 | 9.06 | 9.62 | 10.19| 10.76| 11.33|
| 80        | 0.01            | 1.08 | 1.64 | 2.19 | 2.74 | 3.30 | 3.85 | 4.40 | 4.95 | 5.51 | 6.06 | 6.61 | 7.17 | 7.72 | 8.27 | 8.83 | 9.38 | 9.93 | 10.48| 11.04|
| 85        | 0.01            | 1.06 | 1.60 | 2.14 | 2.68 | 3.22 | 3.75 | 4.29 | 4.83 | 5.37 | 5.91 | 6.45 | 6.99 | 7.53 | 8.07 | 8.61 | 9.15 | 9.69 | 10.23| 10.77|
| 90        | 0.01            | 1.03 | 1.56 | 2.08 | 2.61 | 3.14 | 3.66 | 4.19 | 4.72 | 5.24 | 5.77 | 6.30 | 6.82 | 7.35 | 7.88 | 8.40 | 8.93 | 9.46 | 9.98 | 10.51|
| 95        | 0.01            | 1.01 | 1.52 | 2.04 | 2.55 | 3.07 | 3.58 | 4.10 | 4.61 | 5.13 | 5.64 | 6.16 | 6.67 | 7.19 | 7.70 | 8.22 | 8.73 | 9.25 | 9.76 | 10.28|
Table 22. Statistical Power for Models with Covariate = 0.3 under MAR Data conditions.

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Table 23. $F_{\text{Min}}$ and Chi-square statistic for Models with Covariate = 0.5 under MAR Data conditions.

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Table 24. Statistical Power for Models with Covariate = 0.5 under MAR Data conditions.

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Table 25. $F_{\text{Min}}$ and Chi-square statistic for Models with Covariate $= 0.7$ under MAR Data conditions.

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Table 26. Statistical Power for Models with Covariate = 0.7 under MAR Data conditions.

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</table>
APPENDIX H

STUDY 2C: $F_{\text{MIN}}$, CHI-SQUARE AND POWER TABLES FOR PATTERN MISSINGNESS MODELS

Appendix H contains the following tables:

Table 27. $F_{\text{MIN}}$ and Chi-square statistic for Complete Data and Model A, B, C & D under MCAR Data Conditions.

Table 28. Statistical Power for Complete Data and Model A, B, C & D under MCAR Data Conditions.

Table 29. Percentage Difference in Statistical Power of Complete Data and Model A, B, C & D under MCAR Data Conditions.

Table 30. $F_{\text{MIN}}$ and Chi-square statistic for Complete Data and Model A, B, C & D under MAR Data Conditions.

Table 31. Statistical Power for Complete Data and Model A, B, C & D under MAR Data Conditions.

Table 32. Percentage Difference in Statistical Power of Complete Data and Model A, B, C & D under MAR Data Conditions.
Table 27. $F_{\text{Min}}$ and Chi-square statistic for Complete data and Models A, B, C & D under MCAR Data Conditions.

<table>
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<tr>
<th>Pattern Missingness</th>
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<td>Model D</td>
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Note.
Model A – 50% cases missing at Time 2
Model B – 50% cases missing at Time 3
Model C – 25% cases missing at Time 2 and 25% cases missing at Time 3
Model D – 16.67% cases missing at Time 2, 16.67% cases missing at Time 3, and 16.67% cases missing at Time 2 and Time 3 both.
Table 28. Statistical Power for Complete Data and Model A, B, C & D under MCAR Data Conditions.

<table>
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Note.
Model A – 50% cases missing at Time 2
Model B – 50% cases missing at Time 3
Model C – 25% cases missing at Time 2 and 25% cases missing at Time 3
Model D – 16.67% cases missing at Time 2 and 16.67% cases missing at Time 3, and 16.67% cases missing at Time 2 and Time 3 both.
Table 29. Percentage Difference in Statistical Power of Complete Data and Model A, B, C & D under MCAR Data Conditions.

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Note.  
Model A – 50% cases missing at Time 2  
Model B – 50% cases missing at Time 3  
Model C – 25% cases missing at Time 2 and 25% cases missing at Time 3  
Model D – 16.67% cases missing at Time 2, 16.67% cases missing at Time 3, and 16.67% cases missing at Time 2 and Time 3 both.
Table 30. $F_{\text{Min}}$ and Chi-square statistic for Complete Data and Model A, B, C & D under MAR Data Conditions.

<table>
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<th>Pattern Missingness</th>
<th>$F_{\text{Min}}$</th>
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Note.
Model A – 50% cases missing at Time 2
Model B – 50% cases missing at Time 3
Model C – 25% cases missing at Time 2 and 25% cases missing at Time 3
Model D – 16.67% cases missing at Time 2, 16.67% cases missing at Time 3, and 16.67% cases missing at Time 2 and Time 3 both.
Table 31. Statistical Power for Complete Data and Model A, B, C & D under MAR Data Conditions.

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Note.
Model A – 50% cases missing at Time 2
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Table 32. Percentage Difference in Statistical Power of Complete Data and Model A, B, C & D under MAR Data Conditions.

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Note.
Model A – 50% cases missing at Time 2
Model B – 50% cases missing at Time 3
Model C – 25% cases missing at Time 2 and 25% cases missing at Time 3
Model D – 16.67% cases missing at Time 2, 16.67% cases missing at Time 3, and 16.67% cases missing at Time 2 and Time 3 both.