Modeling Dyadic Attunement: Physiological Concordance in Newly Married Couples and Alliance Similarity in Patient-Therapist Dyads

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University of Massachusetts - Amherst

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Modeling Dyadic Attunement: Physiological Concordance in Newly Married Couples and Alliance Similarity in Patient-Therapist Dyads

A Dissertation Presented

by

HOLLY LAWS

Submitted to the Graduate School of the University of Massachusetts Amherst in partial fulfillment of the requirements for the degree of

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Clinical Psychology
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Approved as to style and content by:

__________________________________________
Aline Sayer, Chair

__________________________________________
Michael Constantino, Member

__________________________________________
Sally Powers, Member

__________________________________________
Paula Pietromonaco, Member

__________________________________________
Sanjiv Gupta, Member

__________________________________________
Niall Bolger, Member

__________________________________________
Melinda A. Novak, Department Chair
Department of Psychology
ABSTRACT

MODELING DYADIC ATTUNEMENT: PHYSIOLOGICAL CONCORDANCE IN NEWLY MARRIED COUPLES AND ALLIANCE SIMILARITY IN PATIENT- THERAPIST DYADS

FEBRUARY 2014

HOLLY LAWS, B.A., WESLEYAN UNIVERSITY
M.A., HUNTER COLLEGE OF THE CITY UNIVERSITY OF NEW YORK
Ph.D., UNIVERSITY OF MASSACHUSETTS AMHERST

Directed by: Professor Aline Sayer

Mutual influence within relationships is theorized as central to human development and functioning across the lifespan. Multiple theories posit a process of progressive bidirectional influence that results in greater similarity between dyad members over time, termed attunement. Yet attunement processes, from dyadic synchrony in healthy child development to partner influence within romantic relationships, are difficult to measure and model. One difficulty is that capturing information from both members of a relationship pair, or dyad, requires statistical modeling that appropriately accounts for the interdependence between them. The present study addressed this issue by putting forward a framework for modeling attunement processes between relationship members over time, and applied this framework to two distinct studies. The studies both tested whether attunement occurred in two large-scale dyadic samples, the first in a sample of newly-married couples, the second in a sample of psychotherapy dyads. Attunement was modeled both as an outcome (in Study 1) and a
predictor (in Study 2), providing interested researchers with an analytic framework for using measures of dyadic attunement as either an independent or a dependent variable.

Findings from Study 1 showed significant attunement in the stress hormone cortisol over the early years of marriage in newlywed couples. This finding is suggestive of bidirectional spousal influence over a longer term than previously tested, as other studies have only inferred attunement processes by finding covariation in spousal cortisol over a matter of days. This study also disaggregated cortisol fluctuations into discrete parts, allowing for tests of spousal attunement not only in cortisol level, but also in physiological response to a stressor. Findings from Study 2 also found significant attunement processes within patient-therapist dyads. Specifically, patient-therapist alliance attunement over time was predictive of better outcome for patients receiving psychotherapy for chronic depression.

A major contribution of these studies was that they modeled theorized relationship processes at the level of the dyad, rather than emphasizing individual outcomes. This dyadic-level modeling of bidirectional influence in turn related to outcomes relevant to psychological health, which may have been obscured or undetectable with other modeling techniques.
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CHAPTER I
INTRODUCTION TO THE TWO STUDIES

People in close relationships influence one another in ways that matter for their psychological functioning and well-being. Through repeated contact over time, they have multiple opportunities to affect one another. This influence is bidirectional, such that each member within a two-person relationship (or dyad) affects the other over time. People are more influenced by those they know and care about than by strangers. They are more likely to be influenced by a family member, close friend, or trusted therapist than by someone they do not know. Few psychologists would argue with these statements. Yet in the discipline of psychology, studies of mutual influence have tended to emphasize outcomes of individuals, rather than the development of relationship processes.

Yet finding ways to capture relationship processes is crucial, since they are the cornerstone of multiple theories of healthy development and functioning. Bidirectional, or mutual, influence is implicated in outcomes across the lifespan, from mother-infant affective synchrony (Feldman, 2007) to findings of physical health concordance in long-married couples (Meyler, Stimpson, & Peek, 2007). Each theory uses its own terminology to describe mutual influence between two members of a relationship dyad. Recent examples of prominent similarity constructs include attunement (Ruttle et al., 2011), synchrony (Laws & Dennis, 2007; Papp, Pendry, & Adam, 2009), concordance (Feichtl et al., 2010), and coregulation (Saxbe & Repetti, 2010).
A. Defining Attunement

Often, theories posit a process of progressive bidirectional influence that results in greater similarity between dyad members over time. In cross-sectional studies, bidirectional influence has most often been inferred by measuring outcomes of both dyad members at a single timepoint and relating them to one another to create a dyadic index. Such cross-sectional relationships between partners’ outcomes will be referred to throughout this dissertation as similarity. The most common ways of measuring similarity are correlational and discrepancy-based approaches (Dyrenforth, Kashy, Donnellan, & Lucas, 2010; Sayer & Klute, 2005). Multiple measures over time are needed, however, to model the process by which dyad members become more similar over time. Attunement is defined as an iterative process of bidirectional influence over time, which results in relationship members becoming more similar to one another over the course of a relationship. It is most often captured by measuring similarity between dyad members at multiple timepoints, and modeling change in these repeated measures over time. To summarize, modeling the dyadic process of becoming more similar over time requires 1) measurement of dyadic similarity at multiple timepoints, and 2) a test of whether this similarity significantly increases over time, indicating attunement.

B. The Importance of Attunement

Attunement is often studied in positive relationship processes. Studies of dyadic synchrony between mothers and their children, for example (Askan, Kochanska, & Ortmann, 2006; Harrist & Waugh, 2002), show that attunement of affect, eye gaze, and communication behaviors among mothers and their children are essential to children’s healthy development. Similarly, attunement of perspectives on psychotherapy between
patients and therapists leads to better outcomes for patients (Hersoug, Høglend, Monsen, & Havik, 2001; Horvath & Bedi, 2002; Swift & Callahan, 2009). Attunement within personal relationships, however, is not always predictive of positive health outcomes. For example, when one couple member is depressed or under great strain, there is evidence of “contagion” of these negative qualities to the other partner (Thompson & Bolger, 1999; Joiner & Katz, 1999).

It is therefore not uniformly the case that attunement is positive for members of a dyad. Rather, attunement is a relationship property within a dyad that can help to predict the degree to which dyad members influence one another, for better or for worse. In the case of therapeutic relationships, attunement between a patient and a therapist on their perceptions of the goals of psychotherapy and of their bond in the relationship may be predictive of a better psychotherapy outcome for the patient. In the case of married couples in which one member has a health vulnerability, on the other hand, in more attuned couples the other partner is at increased risk of developing health problems. Understanding processes of mutual influence over time is thus of vital importance to health researchers and practitioners, as interventions can be aimed at changing such dyad-level processes when they are maladaptive, and in augmenting such processes when they are positive.

C. Methodological Issues in Modeling Dyadic Attunement

The difficulty in modeling attunement is that dyadic indices often need to be created from individual-level measures. Although some studies use observational coding to create a single measure at the level of the dyad (e.g. Legerstee, Markova, & Fisher, 2007; Askan, Kochanska, & Ortmann, 2006), for many constructs dyadic-level
measurement is not possible. For example, in trying to understand mother-infant physiological attunement on the stress hormone cortisol, cortisol measurements from each member of the dyad must be taken separately, and related to one another through statistical methods (Sethre-Hofstad, Stansbury, & Rice, 2002; Neu, Laudenslager, & Robinson, 2009). Statistically created indices of dyadic attunement are also common in research on adult dyadic relationships. Such indices relate scores from each dyad member’s report to create an index that captures both partners’ perspectives.

A problem with many techniques used to create dyadic indices is that they do not account for the interdependence inherent in dyadic data. Methodologists have called for the use of new strategies such as multilevel modeling and structural equation modeling as a solution to more accurately modeling dyadic processes (Bryk & Raudenbush, 1992; Kenny, Kashy, & Cook, 2006). These techniques model data within dyads accounting for the interdependence inherent in dyadic data, while more conventional statistical techniques assume that the scores contributed by the two members of the dyad are independent. Family methodologists have developed advanced statistical techniques that can appropriately model dyadic processes and allow for testing models that more closely resemble the reality of everyday life (Dyrenforth et al., 2010).

One common technique that appropriately accounts for dependency and models partner influence is the Actor-Partner Interdependence Model (APIM; Cook & Kenny, 2005). This longitudinal version of this model is applied by using one partner’s data at a previous timepoint to predict the other partner’s outcome at a subsequent timepoint, while controlling for the original partner’s own influence on him- or herself. Figure 1.1 presents a schematic diagram depicting this conceptual idea of partner influence. Note
that this model provides tests of partner influences, but is framed in terms of individual
couple members’ outcomes.

While the APIM model has advanced the field by popularizing an approach to
analyzing couple-level data that correctly accounts for interdependency, some researchers
argue that it emphasizes interpretation at the level of the individual, rather than at the
level of the dyad (Shrout & Seidman, 2012). Since attunement is the dyadic process of
convergence, it is not adequately captured by models emphasizing outcomes for
individual dyad members. Instead, it requires modeling similarity at different timepoints,
and testing whether this similarity changes over time (see Figure 1.2).

D. The Two Studies

In this dissertation, I examine dyadic similarity and attunement over time in two
distinct studies. The studies both test whether attunement is present in two large-scale
dyadic samples, the first in a sample of newly-married couples, the second in a sample of
psychotherapy dyads. The attunement process is modeled both as an outcome (in Study
1) and a predictor (in Study 2), providing interested researchers with an analytic
framework for using measures of dyadic attunement as either an independent or a
dependent variable.

In Study 1, I examine attunement of the stress biomarker cortisol in newly
married couples during the early years of marriage. The primary aim of this study is to
test whether spouses become significantly more similar in their stress reactivity over
time. As cortisol attunement in adult couples has rarely been examined (Saxbe & Repetti,
2010), this study provides important information about understanding what kinds of
couples are more likely to influence one another’s physiological functioning. Dyadic
attunement is the outcome of interest in this study, and several couple characteristics are used as predictors of different degrees of similarity and attunement across couples.

Study 2 examines attunement of patient and therapist perceptions of the therapeutic alliance, the collaborative relationship seen as essential to successful psychotherapy (Horvath et al., 2011). This study addresses whether similarity between patients’ and therapists’ in their views of the alliance, and attunement in this similarity over time, is predictive of lower depressive symptomatology. This study represents a contribution to the alliance literature in taking both the therapist’s and patient’s perspectives of the alliance into account (Kivlighan, 2007). Study 2 further contributes to the literature by testing whether these constructs, alliance similarity and attunement, are predictive of better outcome for patients receiving psychotherapy for chronic depression. In this study, the attunement process of interest is used as a predictor, rather than an outcome.

The substantive questions raised in each study contribute to a better understanding of the role of attunement in two distinct types of dyadic relationships, but share a third methodological contribution. For both studies, I applied statistical models that take into account the inherent interdependency in dyadic data, and created dyadic measures of the construct of attunement. This dissertation therefore also offers a potential contribution to the emerging methodological literature on modeling dyadic attunement. Although this inquiry will be applied to cortisol attunement in married couples and alliance attunement in therapy dyads, the dyadic modeling of discrepancy in this study can be applied to studies of attunement processes in any dyadic relationship.
Figure 1.1. Model capturing partner influences over time. Based on the Actor-Partner Interdependence Model (APIM; Cook & Kenny, 2005)
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CHAPTER II

STUDY 1: CORTISOL ATTUNEMENT IN NEWLY MARRIED COUPLES

A. Introduction

It is estimated that 73% of adults in the United States have been married at some time in their lives (United States Census Bureau, 2010). Those in healthy marriages have better physical and mental health than those who never marry or who have conflictual marriages (Nielsen, 2005; Huston & Melz, 2004). At the same time, studies indicate that interpersonal stressors, such as partner psychopathology, can have a profound, negative effect on the other partner’s mental health (e.g. Benazon & Coyne, 2000). Within marriages then, partners are uniquely placed to affect one another’s physical and mental health: to buffer against negative health outcomes when the relationship is strong, or by augmenting health risk, when maladaptive processes are at play.

1. Health Concordance in Married Couples

Multiple studies have documented the phenomenon that long-married spouses are more similar to one another than to the general population in a variety of domains, from cardiovascular health to depressive symptomatology (Meyler, Stimpson, & Peek, 2007). This phenomenon is known as health concordance, and three prominent theories have been put forward to explain it. The assortative mating theory argues that people tend to choose romantic partners who are similar to themselves (Segrin, 2004). The shared context theory, sometimes called “common fate” model, contends that by living in the same environment for many years, partners are likely to end up with similar health outcomes due to shared external factors (Ledermann & Kenny, 2012). The third theory to explain spousal health attunement is mutual influence. This theory argues that couple
members influence one another over time, and that through this bidirectional interplay, they become more similar over the course of their relationship (Kenny, Kashy, & Cook, 2006).

The assortative mating theory assumes a static level of similarity throughout life; it does not provide an adequate explanation for partners becoming more similar over time. Theories of shared environment and mutual influence, by contrast, posit that similarity among couple members is a dynamic, rather than static, process (Gonzaga et al., 2007). *Convergence* describes the phenomenon of partners becoming increasingly more similar over time (Anderson, Keltner, & John, 2003; Gonzaga et al., 2007; Swift & Callahan, 2009). Both the shared context and mutual influence explanations provide theoretical rationales for the convergence phenomenon, a process of *attunement* in which spouses become increasingly similar over time. Researchers use different terms to describe these phenomena. In my study, *concordance* refers to spousal *similarity* at a single timepoint. *Attunement* (or *convergence*) refers to the process by which spouses become more similar over time.

Attunement appears to occur in both health-promoting and maladaptive processes within romantic relationships. Anderson et al. (2003) found evidence that partners who became more similar in their emotional responding had healthier relationship outcomes. Gonzaga et al. (2007) found that personality convergence was predictive of greater emotional convergence, which in turn predicted higher relationship satisfaction. Researchers have also found evidence of maladaptive attunement processes. When one couple member is depressed or under great strain, research has shown an increase in partner stress level as well (Thompson & Bolger, 1999; Joiner & Katz, 1999).
While evidence of spousal similarity has been found across multiple literatures, it has been difficult for researchers to untangle the mechanism underlying this similarity. In many research studies couples have been dating or married for some time when they first enroll. Thus, any observed similarities could be due to assortative mating, convergence (either through mutual influence or shared environment), or a combination of the two. One recent prospective longitudinal study of public health in Norway indicates that both assortative mating (selection effects) and convergence processes take place. Ask, Idstad, Engdahl, and Tambs, (2013) were able to identify a subsample of individuals who selected their partners during the years of data collection. This meant that they had prospective data on partners’ characteristics before they met, and were able to follow couples \( N = 1551 \) to look for evidence of convergence in these characteristics after they married or moved in together.

Their study found evidence for both mating selection and convergence processes on a variety of outcomes, from life satisfaction to mental health symptomatology. Members of future couples were indeed more similar to one another than to the general population, averaging a correlation of about .26 in outcomes such as life satisfaction and global mental health measured prior to marriage. Once couples were married, this correlation increased to .42, indicating convergence processes in multiple relationship domains, above and beyond selective mating effects. In addition, there was evidence that convergence processes were non-linear, strongest in the earlier years of the relationship.

Findings from this study suggest that attunement processes may be particularly strong in the early years of relationships. Newlywed marriages are of interest because it is thought that processes of mutual influence may be more apparent in certain
developmental stages of relationships, such as the beginning of married life, than at other points in the marriage (Rehman, Gollan, & Mortimer, 2008). Such processes are considered steepest early in relationships and may lessen over time (Tambs & Mourn, 1992). In addition, patterns observed in early marriage have been shown to be important predictors of relationship functioning and dissolution over a decade later (Huston, Niehaus, & Smith, 2001; Huston, Caughlin, Houts, Smith, & George, 2001).

2. Relevance of Examining Cortisol Similarity and Attunement

The present study will examine similarity and attunement in the stress biomarker cortisol, as it is implicated in multiple mental and physical health outcomes. The hypothalamic-pituitary-adrenal (HPA) axis is the body’s primary system for reacting to and regulating stress. In response to a variety of stressors, the pituitary sends a signal to the adrenal glands to release cortisol into the bloodstream. It takes about 15 minutes for this series of reactions to occur. Because there is a lag from time of secretion from the adrenal gland, cortisol measured in saliva is actually reflective of a physiological reaction 15 to 20 minutes prior to collection (Stansbury & Gunnar, 1994).

HPA dysregulation has been related to anxiety and depression disorders, decreased immune functioning, increased hypertension, and increased cardiovascular risk (Glaser & Kiecolt-Glaser, 1994; McEwen, 1998). Schernhammer et al. (2003) found evidence that HPA reactivity is a risk factor for breast and colon cancers. Cortisol is also implicated in aging-related processes. In a study of normal and pathological aging, Lupien et al. (2005) found that increased cortisol secretion was significantly associated with impairment in cognitive function. Wolf et al. (2005) found significant associations between subjective memory complaints and higher cortisol levels in a sample of health
middle-aged and older participants. Higher cortisol has also been implicated in the development of Alzheimer’s disease, and is considered a risk factor for developing age-related disorders (Otte et al., 2005). Better understanding how spouses affect one another’s HPA function thus has implications for both mental and physical health.

Dickerson and Kemeny (2004) have suggested that greater variability in cortisol, as cortisol levels, may be particularly implicated in increased health risk. Following Powers, Pietromonaco, Gunlicks, & Sayer (2006), this study separates fluctuations in cortisol in response to an acute stressor into two distinct processes: reactivity to and recovery from a particular stressor. Cortisol reactivity is the degree to which an individual’s cortisol level increases in response to an acute stressor. Recovery is defined as the rate at which cortisol levels decrease after the stressor and return to baseline.

Although converging evidence points to cortisol dysregulation as a marker of health risk across many diseases and mental health disorders, fewer studies connect cortisol to the relationship contexts within which most people operate. Since the majority of adults in this country are married, it is essential for health researchers to understand how what is known about cortisol and health at the individual level translates to the context of long-term romantic partnership.

Several studies of romantic relationships indicate that cortisol levels are affected by aspects of the relationship such as quality, conflict, and attachment (e.g. Kiecolt-Glaser et al., 2003; Laurent et al., 2013; Laurent & Powers, 2007; Powers et al., 2006). Partner support has been shown to be a protective factor to stress response, lowering cortisol reactivity in response to a laboratory-induced stressor (Ditzen et al., 2007). Higher stress within marriage, by contrast, has been related to higher waking cortisol and
a flatter decline in the expected natural decrease in cortisol over the day, indicating
greater health risk (Barnett, Steptoe, & Garceis, 2005). The quality of a marriage and
interactions within it, therefore, have effects on cortisol functioning.

Few studies have examined attunement processes in cortisol (Saxbe & Repetti,
2010; Papp, Pendry, Simon, & Adam, 2013). Saxbe and Repetti (2010) conducted a study
examining spousal mutual influence processes in salivary cortisol. Using an observational
design, they examined what they termed cortisol “coregulation,” in 30 married couples
over a three-day period. They sampled salivary cortisol four times per day over three
days, generating 12 cortisol samples per spouse. They regressed each spouses’ cortisol
value on the other spouses’ value at each measurement, controlling for time of day
effects. They reported the average relationship between husbands’ and wives’ cortisol
over the 12 samples. Although they termed the prediction of one spouses’ cortisol from
another “coregulation,” in fact the modeling can be best described as capturing
covariation in spousal cortisol across all measurements.¹ Thus, their study examined
average similarity, but not change in similarity, in spousal cortisol, and found evidence
that spouses’ cortisol levels significantly covaried with one other. They also found that
time spouses spent in proximity on a given day increased the strength of the covariation
in their cortisol levels.

Building on this study using similar methodology in a sample of 47 couples, Papp
et al. (2013) also found evidence of spousal covariation (termed “synchrony” in their

¹ If this modeling technique was applied to the present study, I would follow the guidelines outlined in Bolger and
Laurenceau (2013), and decompose the within-subject variation in the partner cortisol covariate into a separate
between- and within-subject version of the variable. This is accomplished in an HLM model by including the
aggregate or mean of the variable at level 2 and the daily deviation from that mean at level 1.
study). They also found that greater “connectedness,” including more time in the presence of a spouse and lower subjective loneliness predicted greater covariation.

3. Predictors of Variability in Couples’ Cortisol Similarity and Attunement

Both theory and emerging evidence suggest that marital closeness may differentiate the degree to which spouses are influenced by one another. In a review of the importance of interpersonal relationships Berscheid posits that “[M]any of the processes that underlie relationship phenomena...are believed to be causally linked to the closeness of the relationship” (Berscheid, 1994, p. 81). There is reason to believe that closeness may be an important factor to consider in predicting couples’ cortisol attunement. Although closeness has not been related to cortisol attunement within marriage, studies of cortisol attunement in mother-infant dyads lend support to the theory that closeness may be an important predictor of differences between couples in their degree of cortisol attunement.

Several studies of cortisol attunement indicate that greater maternal sensitivity is associated with higher levels of cortisol coregulation in mother-infant dyads (e.g. van Bakel & Riksen-Walraven, 2008; Sethre-Hofstad, Stansbury, & Rice, 2002). Ditzen, Hoppmann, and Klumb (2008) found that greater intimacy was associated with lower cortisol levels in dual-earner couples, but their study did not test for effects of partner stress level. Thus, greater relationship closeness may have positive effects on an individual’s health, but may also leave them more vulnerable to be influenced by their partner’s reactivity. These findings suggest that closeness within marriage may at times buffer spouses from negative influences, and at times engender greater sensitivity to negative partner effects. In this vein, other researchers found that the transmission of
depressive symptomatology across partners is stronger in closer couples, as compared to couples rating themselves as less close (Tower & Kasl, 1996; Tower & Krasner, 2006).

This issue is addressed in my study, in which I hypothesize that cortisol attunement is more pronounced in closer couples. Thus, closeness serves as both a protective factor for those with healthy cortisol regulation, and as a vulnerability in couples where either partner exhibits cortisol dysregulation. Couples with a high degree of closeness may receive benefits from it when the partner is well, but may also be more vulnerable to more reactive stress response by their partner, indicative of cortisol dysregulation. Thus, relationship closeness is an important variable to consider when studying predictors of cortisol attunement. Although there have been no known studies examining the effect of closeness on cortisol attunement over time, work in other areas, particularly the depression contagion literature, suggests that the attunement process will be stronger in couples with a higher degree of felt closeness.

The finding of greater health concordance in longer-married couples also suggests that relationship length is likely to be related to attunement processes as well. Ask et al., (2013) found that couples’ gradual attunement appeared to be nonlinear, such that increased similarity was evident in the early years of the relationship, followed by slowing similarity and greater divergence about 20 years into marriages, followed by increased convergence in later years of intact marriages. Given the present sample’s developmental stage (couples in the early years of relationship formation and maintenance), the literature suggests a positive relationship between relationship length and attunement. Similarly, as physical proximity has been associated with greater
attunement (Saxbe & Repetti, 2010; Papp et al., 2013), it follows that length of cohabitation will be positively related to similarity and attunement in cortisol functioning.

Finally, gender differences will be examined, as several studies have indicated that there are meaningful gender differences in HPA patterns (Almela et al., 2011; Kiecolt-Glaser & Newton, 2001; Powers et al., 2006). There is some evidence that husbands’ cortisol levels are more sensitive to influence from their wives (Saxbe & Repetti, 2010), while other studies find that wives’ cortisol levels are associated with their husbands’ negative behavior while husbands are not similarity affected (Kiecolt-Glaser, Newton, Cacioppo, MacCallum, Glaser, & Malarkey, 1996). These conflicting findings suggest that gender differences may well be important, but do not provide clear evidence for the nature of this difference.

4. The Present Study

The present study expands on prior studies of spousal influence in cortisol in two key ways. First, it examines changes in spousal similarity over the early years of marriage, rather than over a period of days as in prior studies (Saxbe & Repetti, 2010; Papp et al., 2013). Second, the present study examines couples’ similarity in reacting to and recovering from an interpersonal stressor, while prior studies have only examined covariation in cortisol level fluctuation. In addition, with a larger sample size than prior studies, the present inquiry allows for the addition of predictors that may account for differences in cortisol similarity and attunement across couples. Based on the literature review, I developed the following research questions and hypotheses:

**Research Question 1**: Do couples vary in how similar they are in their cortisol entry level, reactivity, and recovery within each lab visit? Significant variability, if found,
provides evidence that couples may vary around the average in systematic and meaningful ways.

**Hypothesis 1:** I predict that there will be significant variability around the average cortisol discrepancy between men and women. Not all couples are expected to have the same pattern as the average couple.

**Research Question 2:** Is there evidence that spouses become more similar in their cortisol functioning across the first years of marriage?

**Hypothesis 2:** I predict that that couples’ cortisol similarity will increase on average from the first to the second lab visit, indicative of convergence or attunement.

**Research Question 3:** Is there significant variability around average cortisol attunement over time, indicating that couple-level characteristics may be reliably associated with differing degrees of similarity and attunement?

**Hypothesis 3:** I predict that there will be significant variability around the average increase in similarity from the first to the second lab visit, indicating that some couples may be more likely to converge in their stress response than others.

**Research Question 4:** If there is significant variability around couples’ average cortisol similarity and convergence over time, are there couple-level predictors that are associated with more or less similarity and attunement?

**Hypothesis 4:** Relationship-level characteristics will be associated with the degree of cortisol similarity and attunement. Specifically, I predict that

a. Higher closeness will be associated with higher cortisol average similarity and attunement over time.
b. Those in longer relationships, and who cohabited longer, will exhibit greater cortisol average similarity and attunement over time.

c. There will be gender differences in attunement, although the direction is not hypothesized based on conflicting findings in the literature.

B. Method

1. Study Design and Procedures

Data for the present study are drawn from a larger study of the biopsychosocial factors that can influence spousal health in newlywed couples.² That study had the following design: Couples were invited to participate in a lab session, described below. These lab visits were repeated two more times, separated by intervals of about a year and a half, for a total of 3 lab visits. For the sake of simplicity, I refer to the lab visits as “Years 1 & 2.”

Lab visits took place during the late afternoon to early evening (between 4 p.m. and 7 p.m.) to control for the diurnal rhythm of cortisol (Dickmeis, 2009; Dorn, Lucke, Loucks, & Berga, 2007). Sessions lasted about 3 hours. During each visit, couples provided demographic information and filled out questionnaires relating to their physical and mental health and relationship quality. Participants were asked to identify and rate the intensity of three topics of unresolved conflict in their relationship. For each couple’s upcoming conflict discussion, the experimenter chose a topic that both partners listed,

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² Research supported by a grant from the National Cancer Institute, Paula Pietromonaco, P.I., Sally Powers, Co-P.I. (R01CA133908); Biopsychosocial Factors in Depression and Marriage; Implications for Cancer.
and that had the highest combined intensity rating. Couples then participated in a 15 minute conflict discussion, in which they attempted to resolve the selected ongoing conflict in their relationship. Five saliva samples were collected before, during, and after the conflict discussion, to capture cortisol reactivity in response to the stress associated with the conflict discussion. These were collected via “passive drool” into tubes and sent to Salimetrics to be assayed. Each sample was split into two samples prior to assay to increase the reliability of measurement of cortisol (Salimetrics, PA). Participants were compensated for each laboratory visit, $50 each for the initial session, and $70 each for the follow-up sessions about a year and a half later. See Figure 2.1 for a schematic representation of the lab visit, which includes timing of saliva sample collection.

2. Participants

Marriage license records filed in western Massachusetts were used to identify recently married couples, and they were recruited via phone and mail invitations to participate in the study. This sampling methodology ensured that couples were representative of the population of western Massachusetts. To be eligible for participation, it was required that both partners were never previously married, had no children, and were between the ages of 18 and 50 years old. Couples were also required to be within the first 7 months of their marriage and not expecting a child at the time of the first lab visit. A total of 225 couples completed their first lab visit. At the time of the present analyses, 183 of the original 225 couples had returned for their second lab visit. As one of the primary questions in the present study related to longitudinal increase in cortisol similarity, only couples who had completed the second lab visit were retained in
the present study. Thus the final sample size for this study is 183 couples; all descriptive statistics that follow are based on this analytic subsample.

At the first lab visit, wives’ average age was 27.98 ($SD = 4.77$) and husbands’ average age was 29.36 ($SD = 5.25$). Most participants had obtained a bachelor’s degree (51% for wives, 45% for husbands) or higher graduate degree (34% of wives, 20% of husbands). The vast majority of participants identified as White (92% of wives, 96% of husbands). Average relationship length from the time couples began dating was 5.13 years ($SD = 3.07$). A majority of couples (85%) reported living together before they married, 2.79 years on average ($SD = 2.31$).

3. Measures

Cortisol. Saliva samples were gathered from both spouses at five timepoints during the lab session. Cortisol levels in saliva actually reflect reactivity from about 15 to 20 min prior to the time of measurement. Saliva samples were collected with this lag time in mind, with the aim of capturing key points of reactivity to and recovery from the conflict discussion stressor. Figure 2.1 depicts the timing of saliva sample collection, and provides a graphic representation for the periods capturing the “reactivity” and “recovery” processes. As it shows, the first sample was given about 30 min into the lab visit, and reflected couples’ level of anticipation of the conflict discussion on entry into the lab (Sample 1). Couples provided the second anticipatory sample about 15 minutes after being informed more explicitly of the nature of the conflict discussion (Sample 2). Sample 3 was provided 10 min after the conflict discussion ended and captured cortisol level during the conflict discussion. Sample 4 was given about 30 min after the discussion, and was the first to capture cortisol recovery after the conflict stressor. The
final sample (Sample 5) was provided 60 min after the conflict discussion. Both the fourth and fifth samples reflect the recovery process as cortisol levels decrease following the conflict discussion. The five samples provided the repeated measures on which the cortisol trajectories are based.

Cortisol values that were greater than or equal to 4 μg/dl were excluded from analyses, as these are beyond the normative range (Aardal & Holm, 1995). The cortisol values were quite positively skewed and kurtotic, and required a base-10 logarithmic transformation in order to symmetrize the data to meet the assumption of normality required for statistical analysis (Tukey, 1977). Skewness and kurtosis measures of the transformed variables were within acceptable levels (no magnitude exceeding +/- 1). Cortisol values beyond 3 SDs were considered outliers and removed from the final dataset, as these measurements exceeded the normal range of values and would unduly influence analyses. Table 2.1 shows the five observed cortisol values for both spouses at both lab visits, in the raw and transformed metric. In the table, focus on the raw cortisol values and note that there is a general descriptive increase in cortisol levels from the first to the second year lab visits. Paired samples t tests were conducted to determine whether these differences were statistically significant. Because a majority of the cortisol samples were not significantly different from one another, it was assumed that attunement between partners was not affected by slight differences in level from the first to the second lab visit.

**Relationship closeness.** Each spouse rated their relationship closeness on 2 subscales of the Perceived Relationship Quality Components scale (PRQC; Fletcher, Simpson, & Thomas, 2000). The Intimacy subscale is a 3 item scale, with each item
measured on a 7-point Likert scale. An example for the intimacy subscale is *How close do you feel toward your partner?* (anchored at 0-Not at all and 7-Extremely). Items from the Intimacy subscale were averaged to create an index of felt-closeness in the relationship. I included both partner’s self-report ratings in the analysis, as perspectives on relationship quality may vary by gender. The PRQC Intimacy subscale has adequate test-retest and internal reliability (α = .86; Fletcher, Simpson, & Thomas, 2000), as well as adequate construct validity (Acker & Davis, 1992). The Love subscale is 3 items, with each item measured on a 7-point Likert scale. An example for the Love subscale is *How much do you love your partner?* (anchored at 0-Not at all and 7-Extremely). The subscale has adequate inter-rater reliability (α = .89; Fletcher, Simpson, & Thomas, 2000).

Cohesion was also used as a proxy for relationship closeness. I used the dyadic cohesion subscale of the Dyadic Adjustment Scale (Spanier, 1976), a common measure of relationship adjustment. The subscale consists of five items each rated on a 6-point Likert scale. The scale describes couples’ level of positive collaborative activity. One item example is *How often would you say the following events occur between you and your mate?...work together on a project (anchored at 0- Never and 5-More often than once per day).* This subscale has adequate validity and reliability (α = .86; Spanier, 1976).

Because these indicators of closeness (love, intimacy, cohesiveness) were highly correlated, I used principal components analysis to create a maximally reliable composite measure of closeness. The principal component accounted for 56% of the variance among the original three variables for wives and 62% for husbands. The distribution of these
composites was approximately normally distributed. See Table 2.2 for relevant
descriptive and skewness statistics.

**Relationship length and cohabitation length.** Information about relationship
length and cohabitation length were collected from questionnaires the respondent filled
out prior to the conflict discussion. Spouses’ separate reports were averaged to create one
measure for each variable, measured in the metric of years. Both variables were
positively skewed, however, and required a square transformation to symmetrize the data.
See Table 2.2 for all descriptive information on the original and transformed variables.

**Gender differences.** Within-couple differences in cortisol value served as a
proxy for gender differences in cortisol functioning. Indicators were created to identify
which couple member had higher cortisol at entry, who had higher reactivity values, and
who had higher recovery values. The variable used as a predictor identified those couples
who had wives with higher values than husbands. This was a “dummy” variable, where
couples in which wives had higher cortisol values were given a value of “1,” and couples
where husbands had the higher value were given a value of “0.” This allowed for a
statistical test of whether couples in which wives had higher cortisol values differed from
couples where husbands had higher cortisol values in analyses. In this study, 43% of
couples had wives with higher entry cortisol than their husbands, 55% of couples had
wives with higher cortisol reactivity values than their husbands, and 43% had wives with
higher cortisol recovery values than their husbands.

4. **Analytic Strategy**

There were three steps in the analyses. In the first step, for each couple member,
growth curve modeling was used to estimate a trajectory of change in cortisol over the
five samples obtained during the first lab visit. Based on the study design of the lab visit, the conflict discussion represented an “intervention” expected to change the course of participants’ cortisol trajectories. The trajectories of change would be non-linear, or discontinuous. These trajectories were therefore modeled as a piecewise growth model, discussed in depth in the results section below. The slope that represents the reactivity process will differ from the slope that represents the recovery process.

In the second step, a difference score (or discrepancy) model was used to obtain estimates of the similarity between spouses’ cortisol trajectories. This model is based on a technique modeling discrepancies between partners’ scores at each measurement occasion using HLM (Sayer & Klute, 2005). The third step used a growth model to estimate change in cortisol similarity (modeled as discrepancy) over 1 year, from the first to the second lab visits. This model is an extension of cross-sectional discrepancy modeling, which allows for modeling change in discrepancy over time. Note that this model estimated similarity between spouses’ individual growth curve model parameters, to account for similarity (discrepancy) in cortisol levels at entry, cortisol reactivity, and cortisol recovery.

In the third step, this model was also used to test the longitudinal attunement convergence hypothesis: whether couples’ cortisol levels and reactivity patterns become more attuned over the course of their first year of marriage. Note that discrepancies can be considered an obverse measure of similarity in their cortisol functioning, where lower discrepancy scores are indicative of greater similarity. If discrepancy decreases, there is evidence of greater cortisol attunement over time.
I used the HLM7 program (Raudenbush, Bryk, & Congdon, 2011) to estimate the models in each step. HLM offers advantages for the study of individual change over time as well as the study of dyads because it explicitly models the two sources of non-independence: the correlations among the repeated measures, and the correlation in the outcome scores between members of the same dyad or couple. HLM can account for differences in the number of timepoints per couple, thus accommodating any potential missing values. HLM does not allow for missingness at level 2. Because there were very few missing data points, variable means were used in the place of missing values in the level 2 file.

C. Results

1. Descriptive Evidence of Dependency Years 1 & 2

Intraclass correlations (ICCs) are descriptive statistics that relate the proportion of variance between couples to the total variance, which is the sum of within- and between-couple differences (Raudenbush & Bryk, 2002). The ICC is also defined as the correlation between the two scores within each couple, and therefore can be used as an estimate of the degree of dependency within couples. To estimate the ICC, I fit an unconditional HLM model. This model provides estimates of the 2 variances (within and between) that are necessary to calculate the ICC. To determine the degree of dependency within couples, on average, intraclass correlations were estimated across all cortisol samples per lab visit. In the first lab visit, the average correlation within couples was 40.22% (ICC=.4022). In the second lab visit, the average was 43.34% (ICC=.4334). The increase in the ICC is in line with my attunement hypotheses in that the correlation, or dependency, increased from the first to the second lab visit. These descriptive statistics
are limited in that they do not provide a test to determine whether this increase in dependency is statistically significant.

2. Modeling of Spouses’ Cortisol Trajectories Within Each Lab Visit

The first research question was: within each lab visit, is there evidence of cortisol similarity in couples? Hypothesis 1 speaks to the average couple, and hypothesis 2 speaks to the variability in the sample around the average couple. To answer this question we first had to model growth curves to the husband and wife cortisol measurements, using separate models for the two lab visits. The form of the growth model was chosen to capture reactivity to and recovery from the conflict stressor. This is a version of the statistical model used in Beck et al. (2013). A piecewise (spline) growth model was chosen that was defined by three parameters: an intercept that represented the expected value of cortisol when time is equal to zero (“entry”); a linear slope that represented the change in cortisol from entry up to and including the conflict discussion (“reactivity”); a linear slope that represented the change in cortisol from the conflict discussion to the end of the lab visit (“recovery”). Piecewise models are often used when an intervening event or experimental manipulation shift the expected growth rate (Swartberg, Seltzer, Stiles, Khoo, 1995; Raudenbush & Bryk, 2002). An important issue in modeling piecewise growth is the choice of the transition point (or knot), which is the timepoint at which the linear slope is permitted to vary. Because the conflict discussion was a planned manipulation with a hypothesized effect on cortisol change across the laboratory session, the trajectory of cortisol change was allowed to vary at that point.

The coding for time used in these models is displayed in schematic form in Table 2.3. The first row is the saliva sample number. The second row shows the original time of
each sample, coded as fractions of an hour from onset. The third row displays the recoding of time to facilitate interpretation of the model intercept, which is defined as the cortisol value when time is zero. By subtracting .5 from each original value of time, the time of the first sample is now equal to zero. This means that the intercept value of each growth curve represented the cortisol level when the participant entered the lab ("entry"). The forth row displays the coding for time that represents Piece 1, the first linear segment, or the reactivity slope. The fifth row displays the coding for time that represents Piece 2, the second linear segment or the recovery slope.

Using a multivariate outcomes model (see Raudenbush, Brennan, & Barnett, 1995) husbands' and wives' cortisol trajectories were estimated simultaneously per lab visit to appropriately account for the dependency in their cortisol scores. The equations for the multivariate piecewise trajectory models were:

Level 1

\[ \text{Cortisol}_{ij} = \]
\[ \beta_{ij}(M_{Entry}) + \beta_{2j}(M_{Piece1}) + \beta_{3j}(M_{Piece2}) + \]
\[ \beta_{4j}(F_{Entry}) + \beta_{5j}(F_{Piece1}) + \beta_{6j}(F_{Piece2}) + r_{ij} \]

Level 2

\[ \beta_{1} = \gamma_{10} + u_{1} \]
\[ \beta_{2} = \gamma_{20} + u_{2} \]
\[ \beta_{3} = \gamma_{30} + u_{3} \]
\[ \beta_{4} = \gamma_{40} + u_{4} \]
\[ \beta_{5} = \gamma_{50} + u_{5} \]
\[ \beta_{6} = \gamma_{60} + u_{6} \]

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3 See Appendix A, Figure A.1 for the "stacked" data setup in the level 1 file that allows for both the dependency within dyads as well as the dependency of cortisol scores within individuals.
The level-1 model is the within-couple model that uses three separate parameters to capture the trajectory for each individual: level at lab visit entry (Entry), change in cortisol up until the conflict discussion (Piece 1), and change in cortisol after the conflict discussion (Piece 2). The $r_j$'s represent the residual deviation, or the extent to which the trajectories are not a perfect fit to the observed cortisol scores. The level-2 model is the between-couple model, where each coefficient from level 1 becomes an outcome variable at level 2. For example, at level 2, $\beta_j$ represents the variability in the male intercepts (entry score) as a function of a grand mean ($\gamma_{10}$), which represents the average male entry score across all couples. The $u$'s represent the random effects, which are the couple-specific deviations from the average score. Model estimates for both the fixed effects (the averages), and the variance of the random effects (the variability) for both lab visit are presented in Table 2.4.

As displayed under the first row of “Fixed Effects” in Table 2.4, both male and female entry cortisol levels were significantly different from zero, at both lab visits. As displayed in row 2 of the Fixed Effects in the table, the reactivity slope was negative at both lab visits, indicating that cortisol was decreasing from entry up to the conflict discussion. As displayed in row 3 of the Fixed Effects in the table, the recovery slope at

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4 I tested whether use of a piecewise model was supported in the data. To do this, I used model comparison to test linear change in cortisol across the lab session to a model which allowed this trajectory to vary after the conflict discussion. If the model allowing this variation was a better fit to the data, the use of a piecewise model would be supported. I therefore fit a linear model to cortisol and obtained a deviance statistic for the linear model, $\chi^2(45$ parameters) = -3608.230. I then allowed the trajectory to vary after the conflict discussion and obtained the more complex model's deviance statistic $\chi^2(91$ parameters) = -4279.933. A model comparison test confirmed that the more complex model, which allowed for the conflict discussion's influence on the cortisol change trajectory, was a significantly better fit to the data than the simple linear model, $\chi^2(\Delta df=46) = 671.708$. This provided support for the use of piecewise modeling of the cortisol trajectories. The same procedure was applied to the second lab visit with the same improvement in model fit for the piecewise versus linear model, $\chi^2(\Delta df=46) = 456.580$.

5 These residuals pick up both the extent to which the model fit differs from the observed data, and the dependency within couples. See Bolger and Shroout (2007) for examples of a model that using correlated residuals to address this issue.
both lab visits was also negative but flatter than the reactivity slope, indicating that the change in cortisol slowed during the recovery phase. Reliabilities for all parameters were good, ranging from .804-.940 (lab visit 1) and .743-.967 (lab visit 2). The cortisol trajectory parameters are graphed in Figure 2.2, representing the average couple. Note that the average couple’s cortisol trajectories in the first year appear more discrepant than in year 2. In the next step of analyses, we tested whether these discrepancies were significantly different from zero. As displayed in the variance components section of Table 2.4 the 12 parameters (six at each lab visit) were all significantly different from zero. This finding indicates that couples were different from the average couple.

As a final step, the level-1 post-estimation scores, or Empirical Bayes (EB) coefficients, were estimated for each spouses’ parameters and were output to a residual file. These represent the model-based estimates of the entry, reactivity, and recovery for each couple.6

3. Hypothesis 1 Results: Variability Around Average Cortisol Discrepancy

In the second step of analyses, the discrepancy between each spouses’ cortisol qualities were estimated simultaneously in a multivariate outcomes model. For each cortisol growth parameter (level at entry, cortisol reactivity, cortisol recovery), the estimates of the EB coefficients from the individual cortisol trajectories were used as outcomes. At level-1, the basic discrepancy model is represented as a difference score model characterized by two coefficients, one capturing the couple average (the model

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6 For the purposes of identifying the model in the following step, a more complex trajectory model was estimated with two trajectories per spouse (per lab visit) rather than one. The two trajectories per spouse were obtained by using the two measures of cortisol provided by Salimetrics separately, rather than averaging them together. Having two estimates of cortisol trajectories for each spouse allowed for a properly identified model in Step 2. As this was a statistical issue, it was decided that it would be more straightforward to present the simpler dual trajectory model for each lab visit. Tables of coefficients for the more complex four-trajectory models for lab visits 1 and 2 are presented in Appendix C.
intercept) and one capturing the couple discrepancy (the slope). To estimate the
discrepancy for each couple, discrepancy indicators were used, with wives’ values coded
as .5 and husbands’ values coded as -.5 Since the three growth parameters were modeled
simultaneously there are a total of 6 parameters at level-1.
The equations for this model were:
Level 1
\[
\text{Cortisol}_{ij} = \\
\beta_{1j}(\text{Entry\_Average}) + \beta_{2j}(\text{Entry\_Discrepancy}) + \\
\beta_{3j}(\text{Piece1\_Average}) + \beta_{4j}(\text{Piece1\_Discrepancy}) + \\
\beta_{5j}(\text{Piece2\_Average}) + \beta_{6j}(\text{Piece2\_Discrepancy}) + r_{ij}
\]
Level 2
\[
\beta_1 = \gamma_{10} + u_1 \\
\beta_2 = \gamma_{20} + u_2 \\
\beta_3 = \gamma_{30} + u_3 \\
\beta_4 = \gamma_{40} + u_4 \\
\beta_5 = \gamma_{50} + u_5 \\
\beta_6 = \gamma_{60} + u_6
\]
This modeling procedure was applied to the two lab visits separately. Estimates
for the average couple and the variability across couples for both models are presented
together in Table 2.5. For each of these two models, Empirical Bayes’ (EB) coefficient
estimates of average and discrepancies in cortisol qualities were output as residuals for
use in the final step of analyses. See Figure 2.3, which displays a sample of EB estimates
output from the above discrepancy model. Note the variability in recovery estimates,
some are negative and some are positive. Negative values indicate couples in which the
husband had higher cortisol values than wives on that parameter, while positive
discrepancy values indicated that wives had higher cortisol values than husbands on that parameter. See Appendix C for predicted values in transformed and original metrics.

4. Hypotheses 2 and 3 Results: Modeling Cortisol Attunement Over Time

The second research question was: Across the lab visits from year 1 to year 2, is there evidence of increased cortisol attunement over time? To answer this question, estimates of male-female discrepancy in each growth parameter were used as input for the final modeling step, in which discrepancy in cortisol parameters at year 1 were compared with the discrepancies from the second lab visit. The EB coefficients for each couple were transformed into absolute discrepancies. The distribution of the absolute values was positively skewed; I used a square root transformation to symmetrize the distribution and to meet normality assumptions (see Figure 2.4).

These scores became our measure of cortisol attunement, where lower values indicated more similarity (less discrepancy), and higher scores indicated less similarity (more discrepancy). These scores were then used as dependent variables in a model testing for change in discrepancy over time. Because there were only two timepoints, this growth model is a difference score model, where the trajectory for each type of discrepancy (entry, reactivity, recovery) is characterized by an intercept and a slope that represents the change over time. The independent variable in this model was Time, coded -.5 to represent lab visit 1, and +.5 to represent lab visit 2. This coding ensures that the intercept (the value of the outcome when time is equal to zero) will reflect the

---

7 The sign of the discrepancy for each parameter indicated couples with the property of having a wife with a higher cortisol level versus a husband with a higher cortisol level. However, this quality changed in about half of the couples from the first to the second lab visit. For example, for entry level cortisol, 104 of the 183 couples had male members with higher entry cortisol at the first lab visit (negative discrepancy scores). At the second lab visit, however, 70 couples had discrepancy values that were different from their time 1 scores (sign of discrepancy score “flipped” from the first to the second lab visit). To avoid the complexity of accounting for multiple types of change, the absolute discrepancy was calculated for couples at each of the lab visits, and these scores were compared across lab visits.
discrepancy score halfway between year 1 and year 2, referred to here as the average score. The equations for the baseline model testing for cortisol attunement over time were:

A multivariate outcomes model was used to estimate change in cortisol similarity (discrepancy) from the first to the second lab visit, for entry cortisol, change prior to conflict (Reactivity), and change following the conflict (Recovery). The equations for this model were:

Level 1:

\[
\text{Cortisol\_Similarity}_{ij} = \beta_1(\text{Entry\_Average}) + \beta_2(\text{Entry\_Attunement}) + \\
\beta_3(\text{Reactivity\_Average}) + \beta_4(\text{Reactivity\_Attunement}) + \\
\beta_5(\text{Recovery\_Average}) + \beta_6(\text{Recovery\_Attunement})
\]

Level 2:

\[
\begin{align*}
\beta_1 &= \gamma_{10} + u_1 \\
\beta_2 &= \gamma_{20} + u_2 \\
\beta_3 &= \gamma_{30} + u_3 \\
\beta_4 &= \gamma_{40} + u_4 \\
\beta_5 &= \gamma_{50} + u_5 \\
\beta_6 &= \gamma_{60} + u_6
\end{align*}
\]

Where \( \beta_1, \beta_2, \) and \( \beta_5 \) (“Average”) represent average level of similarity (discrepancy) across lab visits for each cortisol parameter, and \( \beta_2, \beta_4, \) and \( \beta_6 \) (“Attunement”) represent change in this discrepancy from the first to the second lab visit.

Table 2.6, column 1 displays the parameter estimates from the model. The key estimate

---

8 Because only two timepoints were available, there was not enough information to identify the model and obtain estimates of the level-1 variance. Therefore, I calculated the level-1 measurement error variance using the following formula from classical test theory: measurement error, \( - (1 - \text{reliability})^2 \) variance. I used the reliability estimates in from the Step 2 models and obtained the variance of scores in the output residual files.
to test my hypothesis is the estimate for change from year 1 to year 2. As displayed in
column 1 of Table 2.6, this was negative and significantly different from zero, for all of
the cortisol parameters. This indicates that the absolute discrepancies decreased over
time, and provided evidence for attunement over time on average, in entry cortisol,
reactivity slopes, and recovery slopes. These findings confirmed Hypothesis 2, showing
evidence of significant attunement. Figure 2.5 presents graphs of the average attunement
(thick line) for cortisol entry (Panel A), reactivity (Panel B), and recovery (Panel C).

Confirming Hypothesis 3, all of the variance components were significantly
different from zero, as seen in Table 2.7. This suggests that differences between couples
might account for greater or lesser average similarity, or for more or less attunement
change across the first year of marriage. Figure 2.5 displays attunement trajectories for 20
couples in the study for each cortisol parameter to demonstrate variability in the sample.

5. Predictors of Cortisol Similarity and Attunement

Finally, I added predictors to the baseline model, to test whether couple-level
characteristics were predictive of similarity and attunement in cortisol parameters. For
each variable, I conducted preliminary analyses in which I tested that variable’s
relationship to all six outcomes (i.e. average similarity and attunement on entry,
reactivity, and recovery cortisol similarity). I then trimmed any non-significant predictors
from the model. Two sets of models were conducted: the first tested the relationship
quality variables’ prediction of cortisol similarity and attunement, the second tested for
gender differences in expected similarity and attunement.

a. Relationship characteristic predictors: Hypotheses 4a and 4b results. The
first series of predictor models tested the relative impact of relationship predictors on
couples’ cortisol similarity and attunement. Each predictor was tested separately, trimmed from parameters where non-significant. Significant predictors were retained in a model that was compared to baseline to test whether the addition of that predictor provided improved model fit. The model with the closeness predictor is referred to as Model 2a, while the model with the relationship and cohabitation length variables is Model 2b. In a final model (Model 3), significant predictors were added simultaneously and compared to the previous models.

The two measures (husband and wife reports) representing relationship closeness were each added separately as predictors for Model 2a. Only significant predictors were retained for the final model. Husband-reported closeness was not significantly related to any of the cortisol similarity outcomes. Wife-reported closeness was predictive of greater similarity in cortisol reactivity, and this effect approached statistical significance ($\gamma_{31} = -.013, p = .08$). Closeness related to average similarity in the expected direction, with higher levels of closeness predicting greater cortisol reactivity similarity within couples. The model with the closeness predictor provided a better fit to the data than the baseline model, though this effect only approached statistical significance ($p = .08$). Refer to column 2 of Table 2.6 for estimates from this model (M2a).

Relationship length and length of cohabitation before marriage were both significant predictors of cortisol attunement in reactivity when entered separately as predictors as the preliminary step for Model 2b. They related to reactivity attunement in the expected direction, with longer relationship length or cohabitation length associated with greater attunement (approached statistical significance). When used as predictors in the same model, however, neither was a significant (or near-significant) predictor of any
parameter of cortisol attunement. This was suggestive of multicollinearity of these variables, and so relationship length was selected for reporting and for the final model. As seen in column 3 of Table 2.6 (M2b), relationship length was related to degree of cortisol reactivity attunement in the expected direction, such that longer time together was associated with a higher degree of cortisol attunement (i.e. decreased discrepancy), and this association approached statistical significance ($\gamma_{11} = -.046$, $p = .059$).

In Model 3, both predictors were entered simultaneously to predict cortisol similarity and attunement. As displayed in column 4 of Table 2.6, these predictors retained their prediction on cortisol reactivity similarity and attunement parameters respectively, even when controlling for the effects of the other predictor. Model 3 provided a significantly better fit to the data than either Model 2a ($\Delta \chi^2 [1] = 3.781, p = .049$) or 2b ($\Delta \chi^2 [1] = 3.075, p = .076$). Figure 2.6 provides a graphic representation of these findings, showing expected average cortisol reactivity similarity and attunement over time for couples at varying levels of relationship closeness and relationship length.

b. Testing for gender differences: Hypothesis 4c results. The final models compared two types of couples: those who had a wife with higher cortisol level at the time of the first lab visit and those who had a husband with a higher value at the time of the first visit. Recall that gender differences were defined by a couple-level variable which had a value of “1” if the wife had a higher cortisol level at the time of the first lab visit. The value of the intercept where this indicator was a predictor, therefore, was the estimated value of that parameter for couples where the men were higher than wives (where the value of the “wife higher” indicator variable was “0”). Thus, significance values for this predictor indicate a significant difference between two kinds of couples,
those characterized by wives having higher cortisol values versus husbands. Results showed significant differences between these types of couples on average cortisol similarity in reactivity and recovery. Column 2 in Table 2.8 displays coefficients from the model trimmed to show only significant predictors. Results showed that couples in which wives had higher cortisol at year 1 had lower average cortisol similarity in reactivity and recovery than couples in which husbands had higher cortisol levels at the time of the first lab visit. The model including the gender-specific patterns of cortisol parameters provided a significantly better fit to the data than the baseline model, Δχ²(2) = 8.792, p = .012. Figure 2.7 graphs the expected level of cortisol similarity for each kind of couple, for both reactivity (top panel) and recovery (bottom panel). Note that there was no significant difference in change in expected attunement over time, but the overall level of similarity in these parameters was different by couple type.

D. Discussion

Several findings from this study supported the primary hypotheses. First, there was evidence of significant similarity in spousal cortisol patterns. Second, similarity in spousal cortisol patterns significantly increased from the first to the second lab visit, indicative of attunement. Third, there was significant variability around average cortisol similarity and around the rate of attunement between lab visits. Finally, the study found some evidence that couples differed in their degree of cortisol similarity and attunement depending on characteristics of their relationship.

1. Cortisol Similarity and Variability

Consistent with previous research (Saxbe & Repetti, 2010), the study found evidence of a high degree of dependency in spouses’ cortisol. This finding suggests that
physiological functioning of spouses, like psychological and other social constructs, is subject to the same principle of interdependence. Interdependence in spouses’ cortisol levels and reactivity at a single timepoint could be due to any of the theoretically derived explanatory processes: either from selection of similar partners ( assortative mating), shared environmental influences (shared resources), or from spouses’ bidirectional influence of one another through repeated interaction over time in their relationship (mutual influence). Methodologically, the finding of significant dependency within dyads also supports the use of dyadic modeling of spouses’ physiological data.

2. Cortisol Attunement Across the Early Years of Marriage

Converging methods showed evidence of increased attunement in spouses’ cortisol level at entry, reactivity in anticipation of a stressor, and recovery from that stressor, in the early years of marriage. First, the intraclass correlation, or degree of dependency between spouses’ observed cortisol scores, increased from the first to the second lab visit. Increased cortisol dependency over time suggests a process of mutual influence, in which spouses become more similar to one another in their stress response in the early years of marriage. It is also possible that spouses became more similar in their HPA functioning because of living in a shared environment with similar influences. Assortative mating, however, does not adequately account for the increase in attunement found in this study. Assortative mating suggests that we choose partners who are similar to us as mates, but is not suggestive of an increase in similarity after this selection has taken place. The finding of significant cortisol attunement between spouses in the first lab visit could have been due to assortative mating, shared resources, mutual influence, or some combination of these three. The finding of increased attunement, however, implies
convergence, a move toward greater similarity. This finding is crucial for those interested in intervention to improve physiological or psychological outcomes. It suggests that important patterns of physiological and psychological functioning can be affected by context, either relationship context (mutual influence) or the larger social context (shared resources). If such patterns can be identified, then they can be targeted for intervention and changed with the aim of improving health outcomes.

By modeling spouses’ cortisol trajectories, this study modeled spousal attunement not only in cortisol level, but also cortisol reactivity to and recovery from the laboratory situation and conflict discussion stressor. To the best of my knowledge, this is the first study to examine attunement in spouses’ cortisol patterns of reactivity and recovery. Previous studies have found evidence of spousal similarity only in cortisol level, not acute stress response.

3. Variability Around Average Cortisol Attunement

Significant variability was found around all cortisol similarity parameters (level, reactivity, and recovery). In addition, there was variability around the increase in couples’ attunement from the first to the second lab visit. Variability around average indicates that there are significant differences between couples in their degree of cortisol similarity and attunement. This finding supports the hypotheses that couple-level characteristics can help researchers and clinicians understand which couples are more versus less influential on one another’s stress response. Indeed, in the final step of analyses we found several predictors of this variability.
4. Couple-Level Differences in Cortisol Similarity and Attunement

As hypothesized, relationship length was marginally related to one aspect of cortisol attunement, such that the longer couples had been dating, the greater their cortisol reactivity attunement increased from the first to the second lab visit. This finding suggests that couples become more similar not in their general cortisol level, but in their physiological response to a stressor, in the early years of marriage. Relationship length was not a significant predictor of any of the other attunement parameters.

Relationship closeness also related to cortisol similarity in the expected direction, such that higher levels of closeness were related to greater similarity, on average. Higher closeness was related to higher average similarity in cortisol reactivity. This finding dovetails well with prior research which shows a relationship between HPA response and interpersonal behavior.

We did find evidence of different cortisol similarity patterns depending on gender differences in cortisol level within the couples. For couples’ average reactivity and recovery similarity, those with a wife with higher cortisol values at the first lab visit had lower similarity, on average, than couples in which the husbands’ cortisol level was higher. This could potentially suggest that husbands with higher cortisol are more likely to influence their wives cortisol than vice versa. If cortisol reactivity is considered a risk factor, this would suggest that wives are more vulnerable to negative influence from their husbands than husbands are of wives. This is in line with several recent studies, which have found similar gender differences and hypothesized that differences may be due to gender role socialization (Lee, Fiske, Glick, & Chen, 2010). These studies showed that wives were affected by husbands when husbands were not similarly affected by wives or
that the magnitude of husbands’ effect on wives’ outcomes was greater than vice versa (e.g. Knoll et al., 2009; Kouros & Cummings, 2010; Stimpson, et al., 2006; Whisman & Uebelacker, 2009). Further research on gender differences will be needed in order to clarify the role gender may play in couples’ cortisol coregulation processes. It is possible, for example, that the differences observed related to the nature of the laboratory stressor, which may itself pulls for differential responding based on gender role socialization (Powers et al., 2006).

5. Conclusions and Limitations

This study is limited in that there are only two waves of data available for analysis at this time, as data collection is ongoing. It was notable that though the study found variability in cortisol similarity and around the findings of average attunement over time, predictors only approached statistical significance. It is possible that the weak effects were due to the constraints associated with only having two measurement points. The findings from this study are in some sense preliminary, and may signal which predictors will be implicated in the attunement process once longer term analysis of all three lab visits is possible. On the other hand, it is possible that the cortisol attunement process takes place for other reasons not captured by the selected predictor variables, and that other predictors should be considered.

In addition, future studies should use information about how relationship closeness predicts cortisol attunement to identify those couples at risk of negative health outcomes. Although the present study will provide useful information about which couples may be more likely to influence one another, it does not directly examine the
question of maladaptive versus adaptive profiles of couples’ cortisol attunement in early marriage.

As in any observational study, it is possible that some of the longitudinal effects observed in this study were due to other factors, such as habituation to the laboratory visit, rather than to true changes in the relationship. Couples did engage in a new conflict discussion of their own choosing in the second lab visit, so it could be argued that the argument was equally activating as an interpersonal stressor. In addition, the cortisol values were slightly higher, not lower in the second lab visit, though these differences were generally not statistically significant. But the possibility that the increased attunement in spouses’ stress reactivity was due to practice effects should also be noted.

Finally, while this study provides a framework for testing whether significant attunement change took place, it did not speak directly to the mechanism responsible for increased attunement. It did not measure or test variables that might illuminate the mechanism through which couples become more similar in cortisol functioning over time. Studies that directly examine possible confounding variables or mediational processes that explain physiological attunement are needed.
Table 2.1. Raw and transformed values for wives’ and husbands’ cortisol samples for lab visits 1 and 2.

<table>
<thead>
<tr>
<th>Wives’ Samples</th>
<th>Lab Visit 1</th>
<th></th>
<th>Lab Visit 2</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>SD</td>
<td>Min</td>
<td>Max</td>
</tr>
<tr>
<td>1</td>
<td>ug/dl</td>
<td>.106</td>
<td>.119</td>
<td>.017</td>
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<tr>
<td></td>
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<td>-1.769</td>
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<td>.073</td>
<td>.014</td>
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<tr>
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<td>-1.876</td>
</tr>
<tr>
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<td>ug/dl</td>
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<td>.067</td>
<td>.005</td>
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<td>-2.274</td>
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<td>ug/dl</td>
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<td>.064</td>
<td>.004</td>
</tr>
<tr>
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<td>.052</td>
<td>.004</td>
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<td>Husbands’ Samples</td>
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</tr>
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<td>.122</td>
<td>.011</td>
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<td>ug/dl</td>
<td>.096</td>
<td>.120</td>
<td>.003</td>
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<td>.318</td>
<td>-1.942</td>
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<td>3</td>
<td>ug/dl</td>
<td>.071</td>
<td>.060</td>
<td>.007</td>
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<td>.006</td>
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<td>.110</td>
<td>.004</td>
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<td>1.365</td>
<td>.356</td>
<td>-2.397</td>
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Note: Significant differences between a participant’s 1st and 2nd visit cortisol samples are bolded.
Table 2.2. Descriptive statistics for all predictors.

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<tr>
<th></th>
<th>Mean</th>
<th>SD</th>
<th>Min</th>
<th>Max</th>
<th>Skew</th>
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<tr>
<td><strong>Averages of Couple Reports</strong></td>
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<td></td>
<td></td>
<td></td>
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<tr>
<td>Relationship Length (Years)</td>
<td>5.13</td>
<td>3.07</td>
<td>.5</td>
<td>16.58</td>
<td>1.10</td>
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<td>Relationship Length (Transformed)</td>
<td>2.17</td>
<td>.66</td>
<td>.71</td>
<td>4.07</td>
<td>.32</td>
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<tr>
<td>Cohabitation Length (Years)</td>
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<td>2.36</td>
<td>0</td>
<td>13.75</td>
<td>1.98</td>
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<td>Cohabitation Length (Transformed)</td>
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<td>.80</td>
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<td>.11</td>
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<td><strong>Wife Self Report</strong></td>
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<tr>
<td>Intimacy</td>
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<td>.62</td>
<td>4.67</td>
<td>7.00</td>
<td>-.87</td>
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<tr>
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<td>7.00</td>
<td>-2.31</td>
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<tr>
<td>Cohesion</td>
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<td>2.78</td>
<td>8.00</td>
<td>24.00</td>
<td>-.36</td>
</tr>
<tr>
<td>Closeness (Composite of standardized Intimacy, Love, &amp; Cohesion)</td>
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<td>-3.90</td>
<td>1.49</td>
<td>-.91</td>
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<tr>
<td>Satisfaction</td>
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<td>3.57</td>
<td>28.00</td>
<td>49.00</td>
<td>-.81</td>
</tr>
<tr>
<td><strong>Husband Self Report</strong></td>
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</tr>
<tr>
<td>Intimacy</td>
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<td>7.00</td>
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<td>4.33</td>
<td>7.00</td>
<td>-1.78</td>
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<td>Cohesion</td>
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<td>2.72</td>
<td>8.00</td>
<td>24.00</td>
<td>-.36</td>
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<tr>
<td>Closeness (Composite of standardized Intimacy, Love, &amp; Cohesion)</td>
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<td>1.00</td>
<td>-3.49</td>
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<td>-.81</td>
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<td>28.00</td>
<td>49.00</td>
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Table 2.3. Timing of saliva samples with piecewise trajectory coding.

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<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
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<tbody>
<tr>
<td>Time elapsed from study onset (hours)</td>
<td>.5</td>
<td>1.17</td>
<td>1.71</td>
<td>2.04</td>
<td>2.54</td>
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<tr>
<td>Time elapsed centered on 1st saliva sample</td>
<td>0</td>
<td>.67</td>
<td>1.21</td>
<td>1.54</td>
<td>2.05</td>
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<tr>
<td>Piece 1 coding Reactivity</td>
<td>0</td>
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<td>1.21</td>
<td>1.21</td>
<td>1.21</td>
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<tr>
<td>Piece 2 coding Recovery</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>.33</td>
<td>.83</td>
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</table>
Table 2.4. Parameter estimates and associated standard errors for wives’ and husbands’ cortisol trajectories for lab visits 1 and 2.\textsuperscript{a}

<table>
<thead>
<tr>
<th>Fixed Effects</th>
<th>Lab Visit 1</th>
<th></th>
<th>Lab Visit 2</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Husband</td>
<td>Wife</td>
<td>Husband</td>
<td>Wife</td>
</tr>
<tr>
<td>Entry</td>
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<td>-1.077</td>
<td>-1.033</td>
<td>-1.070</td>
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<tr>
<td></td>
<td>(.024)</td>
<td>(.020)</td>
<td>(.024)</td>
<td>(.017)</td>
</tr>
<tr>
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<td>-.150</td>
<td>-.123</td>
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<tr>
<td></td>
<td>(.014)</td>
<td>(.011)</td>
<td>(.011)</td>
<td>(.011)</td>
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<tr>
<td>Piece 2 (Recovery)</td>
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<td>-.081</td>
<td>-.085</td>
<td>-.082</td>
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<tr>
<td></td>
<td>(.019)</td>
<td>(.017)</td>
<td>(.015)</td>
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</table>

<table>
<thead>
<tr>
<th>Variance Components</th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Entry</td>
<td>.100</td>
<td>.070</td>
<td>.107</td>
<td>.051</td>
</tr>
<tr>
<td>Piece 1</td>
<td>.029</td>
<td>.021</td>
<td>.016</td>
<td>.017</td>
</tr>
<tr>
<td>Piece 2</td>
<td>.059</td>
<td>.043</td>
<td>.030</td>
<td>.046</td>
</tr>
<tr>
<td>Level 1</td>
<td>.008</td>
<td>.008</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Model Deviance (df)</td>
<td>-4252.624 (28)</td>
<td>-4482.397 (28)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

\textsuperscript{a} All values were statistically significant, \( p < .001 \)
Table 2.5. Parameter estimates, confidence intervals, and standard errors for dyadic average and dyadic discrepancy of cortisol trajectory parameters, by lab visit.

<table>
<thead>
<tr>
<th></th>
<th>Lab Visit 1</th>
<th></th>
<th>Lab Visit 2</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Coefficient</td>
<td>SE</td>
<td>Coefficient</td>
<td>SE</td>
</tr>
<tr>
<td></td>
<td>[95% CI]</td>
<td></td>
<td>[95% CI]</td>
<td></td>
</tr>
<tr>
<td>Fixed Effects</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Entry Average</td>
<td>-.1052***</td>
<td>.017</td>
<td>-.1052***</td>
<td>.016</td>
</tr>
<tr>
<td></td>
<td>[-1.085, -1.019]</td>
<td></td>
<td>[-1.083, -1.021]</td>
<td></td>
</tr>
<tr>
<td>Entry Discrepancy(^a)</td>
<td>-.049(^\dagger)</td>
<td>.026</td>
<td>-.037</td>
<td>.025</td>
</tr>
<tr>
<td></td>
<td>[-.100, .002]</td>
<td></td>
<td>[.086, .012]</td>
<td></td>
</tr>
<tr>
<td>Piece 1 Average</td>
<td>-.167***</td>
<td>.008</td>
<td>-.137***</td>
<td>.006</td>
</tr>
<tr>
<td></td>
<td>[-.183, -.151]</td>
<td></td>
<td>[-.149, -.125]</td>
<td></td>
</tr>
<tr>
<td>Piece 1 Discrepancy(^b)</td>
<td>.027(^\dagger)</td>
<td>.015</td>
<td>.027*</td>
<td>.011</td>
</tr>
<tr>
<td></td>
<td>[.002, .056]</td>
<td></td>
<td>[.005, .049]</td>
<td></td>
</tr>
<tr>
<td>Piece 2 Average</td>
<td>-.110***</td>
<td>.010</td>
<td>-.083***</td>
<td>.008</td>
</tr>
<tr>
<td></td>
<td>[-.130, -.090]</td>
<td></td>
<td>[.099, -.067]</td>
<td></td>
</tr>
<tr>
<td>Piece 2 Discrepancy(^c)</td>
<td>.057*</td>
<td>.024</td>
<td>.003</td>
<td>.019</td>
</tr>
<tr>
<td></td>
<td>[.010, .104]</td>
<td></td>
<td>[-.034, .040]</td>
<td></td>
</tr>
<tr>
<td>Variance Components</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Entry Average</td>
<td>.050***</td>
<td></td>
<td>.046***</td>
<td></td>
</tr>
<tr>
<td>Entry Discrepancy</td>
<td>.126***</td>
<td></td>
<td>.118***</td>
<td></td>
</tr>
<tr>
<td>Piece 1 Average</td>
<td>.011***</td>
<td></td>
<td>.007***</td>
<td></td>
</tr>
<tr>
<td>Piece 1 Discrepancy</td>
<td>.041***</td>
<td></td>
<td>.023***</td>
<td></td>
</tr>
<tr>
<td>Piece 2 Average</td>
<td>.017***</td>
<td></td>
<td>.014***</td>
<td></td>
</tr>
<tr>
<td>Piece 2 Discrepancy</td>
<td>.103***</td>
<td></td>
<td>.067***</td>
<td></td>
</tr>
<tr>
<td>Level 1</td>
<td>.0001</td>
<td></td>
<td>.0004</td>
<td></td>
</tr>
<tr>
<td>Model Deviance (df)</td>
<td>-6503.174 (28)</td>
<td></td>
<td>-5505.750 (28)</td>
<td></td>
</tr>
</tbody>
</table>

Note: \(^\dagger\) p < .10, \(^*\) p < .05, \(^**\) p < .01, \(^***\) p < .001.
\(^a\) Recall that negative discrepancy values indicate that wives have lower cortisol values, on average, than husbands. This coefficient is the discrepancy of two logs, and can be rewritten as the log of the ratio of wives to husbands. This ratio can be converted to a percentage through exponentiation to clarify their interpretation. For lab visit 1, the Entry discrepancy of -.049 means that husbands had cortisol values that were 12% greater than those of wives. For lab visit 2, the -.037 coefficient means that husbands had cortisol values that were 9% greater than wives.
\(^b\) For both lab visits 1 and 2, the Piece 1 discrepancy coefficient of .027 means that wives had cortisol values that were 6% greater than those of husbands.
\(^c\) For lab visit 1, the Piece 2 discrepancy coefficient of .057 means that wives had cortisol values that were 14% greater than those of husbands. For lab visit 2, the Piece 2 discrepancy coefficient of .003 means that wives had cortisol values that were .7% greater than those of husbands.

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Table 2.6. Parameter estimates and associated standard errors (in parentheses) for relationship closeness and length as a predictors of cortisol similarity and cortisol attunement over time.

<table>
<thead>
<tr>
<th>Entry Cortisol</th>
<th>M1</th>
<th>M2a</th>
<th>M2b</th>
<th>M3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dyadic Average Similarity, $\gamma_{10}$</td>
<td>.46***</td>
<td>.46***</td>
<td>.46***</td>
<td>.46***</td>
</tr>
<tr>
<td>(01)</td>
<td>(01)</td>
<td>(01)</td>
<td>(01)</td>
<td></td>
</tr>
<tr>
<td>Attunement from Y1 to Y2, $\gamma_{20}$</td>
<td>-.04*</td>
<td>-.04*</td>
<td>-.04*</td>
<td>-.04*</td>
</tr>
<tr>
<td>(02)</td>
<td>(02)</td>
<td>(02)</td>
<td>(02)</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Cortisol Reactivity</th>
<th>M1</th>
<th>M2a</th>
<th>M2b</th>
<th>M3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dyadic Average Similarity, $\gamma_{30}$</td>
<td>.33***</td>
<td>.33***</td>
<td>.33***</td>
<td>.33***</td>
</tr>
<tr>
<td>(01)</td>
<td>(01)</td>
<td>(01)</td>
<td>(01)</td>
<td></td>
</tr>
<tr>
<td>Closeness, $\gamma_{31}$</td>
<td>-</td>
<td>-.013†</td>
<td>-</td>
<td>-.014†</td>
</tr>
<tr>
<td>(008)</td>
<td>(008)</td>
<td>(008)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Attunement from Y1 to Y2, $\gamma_{40}$</td>
<td>-.05**</td>
<td>-.05**</td>
<td>-.05**</td>
<td>-.05**</td>
</tr>
<tr>
<td>(02)</td>
<td>(02)</td>
<td>(02)</td>
<td>(02)</td>
<td></td>
</tr>
<tr>
<td>Relationship Length, $\gamma_{41}$</td>
<td>--</td>
<td>--</td>
<td>-.046†</td>
<td>-.046†</td>
</tr>
<tr>
<td>(024)</td>
<td>(024)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Cortisol Recovery</th>
<th>M1</th>
<th>M2a</th>
<th>M2b</th>
<th>M3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dyadic Average Similarity, $\gamma_{50}$</td>
<td>.40***</td>
<td>.40***</td>
<td>.40***</td>
<td>.40***</td>
</tr>
<tr>
<td>(01)</td>
<td>(01)</td>
<td>(01)</td>
<td>(01)</td>
<td></td>
</tr>
<tr>
<td>Attunement from Y1 to Y2, $\gamma_{60}$</td>
<td>-.042†</td>
<td>-.042†</td>
<td>-.042†</td>
<td>-.042†</td>
</tr>
<tr>
<td>(022)</td>
<td>(022)</td>
<td>(022)</td>
<td>(022)</td>
<td></td>
</tr>
</tbody>
</table>

| Model Fit Statistics | | | | |
|----------------------|----------------|----------------|----------------|
| Deviance (df) | 9404.08 (27) | 9401.06 (28) | 9400.35 (28) |
| Δ Deviance (Δdf) | -- | 3.02† (1) | 3.73† (1) |
| | | | 3.78‡ (1) |
| | | | 3.08§ (1) |

Note: † $p < .10$, * $p < .05$, ** $p < .01$, *** $p < .001$.

a Model 1 versus Model 2a 
b Model 1 versus Model 2b 
c Model 2a versus Model 3 
d Model 2b versus Model 3
Table 2.7. Variance components for models 1 through 3.

<table>
<thead>
<tr>
<th>Variance Components</th>
<th>M1</th>
<th>M2a</th>
<th>M2b</th>
<th>M3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Entry Dyadic Average Similarity, $\tau_{11}$</td>
<td>.02910</td>
<td>.02910</td>
<td>.02910</td>
<td>.02910</td>
</tr>
<tr>
<td>Entry Attunement, $\tau_{22}$</td>
<td>.07823</td>
<td>.07823</td>
<td>.07823</td>
<td>.07823</td>
</tr>
<tr>
<td>Reactivity Dyadic Average Similarity, $\tau_{33}$</td>
<td>.01173</td>
<td>.01156</td>
<td>.01173</td>
<td>.01156</td>
</tr>
<tr>
<td>Reactivity Attunement, $\tau_{44}$</td>
<td>.04939</td>
<td>.04939</td>
<td>.04855</td>
<td>.04855</td>
</tr>
<tr>
<td>Recovery Dyadic Average Similarity, $\tau_{55}$</td>
<td>.01937</td>
<td>.01937</td>
<td>.01937</td>
<td>.01937</td>
</tr>
<tr>
<td>Recovery Attunement, $\tau_{66}$</td>
<td>.08664</td>
<td>.08664</td>
<td>.08664</td>
<td>.08664</td>
</tr>
</tbody>
</table>

*All values were statistically significant, $p < .001$*
Table 2.8. Parameter estimates and associated standard errors (in parentheses) for gender differences model. Cortisol reactivity and recovery similarity predicted by within-couple gender differences in cortisol level observed in year 1.

<table>
<thead>
<tr>
<th></th>
<th>M1</th>
<th></th>
<th>M2</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Entry Cortisol</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dyadic Average Similarity, $\gamma_{10}$</td>
<td>.46***</td>
<td>.01</td>
<td>.01</td>
<td>.01</td>
</tr>
<tr>
<td>Attunement from Y1 to Y2, $\gamma_{20}$</td>
<td>-.04*</td>
<td>.02</td>
<td>-.04*</td>
<td>.02</td>
</tr>
<tr>
<td><strong>Cortisol Reactivity</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dyadic Average Similarity, $\gamma_{30}$</td>
<td>.31***</td>
<td>.01</td>
<td>.31***</td>
<td>.01</td>
</tr>
<tr>
<td>Female Higher Cort Couples, $\gamma_{31}$</td>
<td>.03*</td>
<td>.02</td>
<td>.03*</td>
<td>.02</td>
</tr>
<tr>
<td>Attunement from Y1 to Y2, $\gamma_{40}$</td>
<td>-.05**</td>
<td>.02</td>
<td>-.05**</td>
<td>.02</td>
</tr>
<tr>
<td><strong>Cortisol Recovery</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dyadic Average Similarity, $\gamma_{50}$</td>
<td>.40***</td>
<td>.01</td>
<td>.378***</td>
<td>.02</td>
</tr>
<tr>
<td>Female Higher Cort Couples, $\gamma_{51}$</td>
<td>.04*</td>
<td>.02</td>
<td>.04*</td>
<td>.02</td>
</tr>
<tr>
<td>Attunement from Y1 to Y2, $\gamma_{60}$</td>
<td>-.04†</td>
<td>.02</td>
<td>-.04†</td>
<td>.02</td>
</tr>
<tr>
<td><strong>Variance Components</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Entry Dyadic Similarity, $\tau_{11}$</td>
<td>.02910***</td>
<td></td>
<td>.02910***</td>
<td></td>
</tr>
<tr>
<td>Entry Attunement, $\tau_{22}$</td>
<td>.07823***</td>
<td></td>
<td>.07823***</td>
<td></td>
</tr>
<tr>
<td>Reactivity Dyadic Similarity, $\tau_{33}$</td>
<td>.01173***</td>
<td></td>
<td>.01148***</td>
<td></td>
</tr>
<tr>
<td>Reactivity Attunement, $\tau_{44}$</td>
<td>.04939***</td>
<td></td>
<td>.04939***</td>
<td></td>
</tr>
<tr>
<td>Recovery Dyadic Similarity, $\tau_{55}$</td>
<td>.01937***</td>
<td></td>
<td>.01906***</td>
<td></td>
</tr>
<tr>
<td>Recovery Attunement, $\tau_{66}$</td>
<td>.08664***</td>
<td></td>
<td>.08664***</td>
<td></td>
</tr>
<tr>
<td><strong>Model Fit Statistics</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Deviance (df)</td>
<td>9404.08 (27)</td>
<td></td>
<td>9395.29 (29)</td>
<td></td>
</tr>
<tr>
<td>$\Delta$ Deviance ($\Delta$df)$^a$</td>
<td></td>
<td></td>
<td>8.80* (2)</td>
<td></td>
</tr>
</tbody>
</table>

Note: $^a p < .10$, * $p < .05$, ** $p < .01$, *** $p < .001$.  

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Figure 2.1. Schematic of lab visit, including time of saliva sample collection.
Figure 2.2. Average cortisol trajectories for each spouse at lab visits 1 & 2.
Figure 2.3. EB estimates of couple average and couple discrepancy for each component of their cortisol trajectories, displayed for first 10 couples.

<table>
<thead>
<tr>
<th>CoupleD</th>
<th>EntryAverage</th>
<th>EntryDiscrep</th>
<th>ReacAverage</th>
<th>ReacDiscrep</th>
<th>RecovAverage</th>
<th>RecovDiscrep</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>-1.279</td>
<td>-0.278</td>
<td>-0.121</td>
<td>-0.190</td>
<td>0.021</td>
<td>0.328</td>
</tr>
<tr>
<td>2</td>
<td>-1.254</td>
<td>-0.406</td>
<td>-0.005</td>
<td>-0.024</td>
<td>-0.158</td>
<td>-0.229</td>
</tr>
<tr>
<td>3</td>
<td>-1.148</td>
<td>-0.095</td>
<td>-0.186</td>
<td>0.055</td>
<td>-0.258</td>
<td>0.349</td>
</tr>
<tr>
<td>5</td>
<td>-1.212</td>
<td>-0.090</td>
<td>-0.664</td>
<td>0.007</td>
<td>-0.293</td>
<td>-0.061</td>
</tr>
<tr>
<td>6</td>
<td>-1.004</td>
<td>-0.212</td>
<td>-0.094</td>
<td>0.087</td>
<td>-0.139</td>
<td>0.144</td>
</tr>
<tr>
<td>9</td>
<td>-0.866</td>
<td>-0.270</td>
<td>-0.309</td>
<td>0.303</td>
<td>-0.035</td>
<td>-0.006</td>
</tr>
<tr>
<td>10</td>
<td>-0.802</td>
<td>0.325</td>
<td>-0.123</td>
<td>-0.037</td>
<td>-0.106</td>
<td>-0.012</td>
</tr>
</tbody>
</table>
Figure 2.4. Distributions of EB estimates of cortisol discrepancy values, absolute value of these discrepancies, and the transformation of the absolute values.

<table>
<thead>
<tr>
<th>Distribution</th>
<th>Skewness</th>
<th>Kurtosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cortisol Discrepancies</td>
<td>-.164</td>
<td>3.842</td>
</tr>
<tr>
<td>Absolute Cortisol Discrepancies</td>
<td>2.313</td>
<td>7.746</td>
</tr>
<tr>
<td>Transformation of Absolute Cortisol Discrepancies</td>
<td>.727</td>
<td>7.85</td>
</tr>
</tbody>
</table>
Figure 2.5. Average dyadic attunement trajectory (thick line) superimposed on the couple-specific trajectories for a sample of 20 couples, displayed for cortisol entry (Panel A), reactivity (Panel B), and recovery (Panel C).
Figure 2.6. Change in couples’ similarity in cortisol reactivity as a function of relationship length and closeness, displayed at low (25th percentile) and high (75th percentile) values.
Figure 2.7. Cortisol similarity as a function of within-couple gender differences in cortisol level. The top panel presents differences in recovery, and the bottom panel presents differences in reactivity.
CHAPTER III

STUDY 2: PATIENT- THERAPIST ALLIANCE SIMILARITY AND ATTUNEMENT AS PREDICTORS OF OUTCOME IN CHRONIC DEPRESSION TREATMENT

A. Introduction

1. The Alliance in Psychotherapy Research

The concept that there is no significant difference in outcome due to treatments from distinct theoretical orientations is an empirically tested phenomenon (Luborsky, Singer, & Luborsky, 1975; Wampold et al., 1997). Although still somewhat controversial, this idea is accepted by psychotherapy researchers across theoretical orientations. This has led researchers to theorize that there are common factors at work across psychotherapy modalities (including adherence, therapist approach and personality, and therapeutic alliance), which partially account for improvement in client outcomes across psychotherapy modalities (Castonguay & Beutler, 2006; Kazdin, 2005; Wampold, 2001). The quality of the therapeutic relationship is a powerful curative mechanism across multiple types of treatment and disorders (Norcross, 2002; Norcross & Lambert, 2011). Norcross & Lambert (2011) estimated that 12% of outcome change in psychotherapy is due to variables related to the patient-therapist relationship. Facets of the psychotherapy relationship include distinct but overlapping constructs such as empathy, responsiveness, and working alliance between patients and therapists, also referred to as the therapeutic alliance, or simply the alliance (Constantino et al., 2010; Elvins & Green, 2008). Although there are differences in conceptualization and measurement of this construct (Constantino et al., 2002), it is generally agreed that the alliance is a term describing a positive, collaborative working relationship between
patients and therapists comprising both affective and cognitive components (Horvath & Bedi, 2002; Elvins & Green, 2008; Hougaard, 1994). The working alliance is a pan-theoretical construct that captures the positive bond between patients and therapists, as well as agreement between patients and therapists on the tasks and goals of psychotherapy (Bordin, 1979). Agreement on goals is defined as patients and therapists sharing a view of the purpose of the therapy, such as patient symptom reduction, improved family relationships, or increased quality of life. Agreement on tasks refers to feeling that actions within the psychotherapy process will help the patient reach the defined goal. Drawing from these definitions, the present study defines the alliance as including both positive affective bond between patients and therapists, as well as attention to and agreement on the goals and tasks of psychotherapy.

2. The Alliance-Outcome Link

The relationship between alliance and psychotherapy outcome has been well-established. A recent meta-analysis of 190 psychotherapy studies found that the alliance had a small but robust effect on outcome (weighted $r = .275, p < .001$) across multiple treatment modalities and types of psychopathology (Horvath et al., 2011). There has been some debate as to the casual direction of this relationship, with some arguing that symptom change early in psychotherapy is responsible for increases in alliance, rather than the alliance being responsible for improved outcomes (e.g. DeRubeis & Feeley, 1990; Strunk, Brotman, & DeRubeis, 2010). Some studies have found no link between alliance and outcome, once initial symptom change was accounted for (e.g. Feeley et al., 1999), whereas other studies found evidence that early alliance was predictive of outcome, even when controlling for early symptom change (Klein et al., 2003; Arnow et
al., 2013). Although there is still a debate about the direction of the alliance-outcome link, the majority of studies report this link is present, and a growing body of literature supports the idea that good alliances predict better outcomes, rather than early symptom change producing stronger alliances.

There is growing consensus that measures of the alliance early in psychotherapy, in particular, are more predictive of outcome than later measurements (Constantino et al., 2002; Horvath & Bedi, 2002). Predicting outcome from early alliance measure also establishes temporal precedence between the hypothesized process and outcome (DeRubeis & Feeley, 1990; Kraemer, Wilson, Fairburn, & Agras, 2002; Barber, 2009). This allows for the inference, if not direct test, of a causal process. The importance of measuring alliance-related constructs early in psychotherapy, then, is indicated by the literature.

3. The Alliance as Dyadic Construct

The alliance is a construct rooted in the idea of patient-therapist agreement on, and shared experience of, psychotherapy. As Bordin (1979) pointed out long ago, the concept is fundamentally dyadic in nature. There has historically been a mismatch between the dyadic nature of the construct and the way it is measured. The Working Alliance Inventory (WAI) asks several questions about dyadic properties of the relationship. For example, it asks the patient to rate the following statement “[My therapist] and I agree about the things I will need to do in counseling to help improve my situation” (WAI; Horvath & Greenberg, 1989). The therapist version of the question similarly asks the therapist to rate “[My client] and I agree about the steps to be taken to improve his situation.”
While it is possible to incorporate both patient and therapist ratings of the alliance, the vast majority of studies only collect patient ratings of it (Elvins & Green, 2008; Horvath et al., 2011). The bias toward collecting patient ratings may be due to research-based recommendations that patient ratings were more predictive of psychotherapy outcome (Castonguay, Constantino, & Holtforth, 2006; Santiago et al., 2002). An early meta-analysis of the alliance-outcome link did find evidence that patient ratings of the alliance were more strongly associated with outcome than therapist ratings (Horvath & Symonds, 1991). This finding has been refuted in more a recent meta-analysis of the literature, however, that found no significant difference in the strength of the link between therapist versus patient reports of alliance in predicting outcome (Horvath et al., 2011).

4. Differences in Patient and Therapist Views of the Alliance

While of equal predictive importance, patient and therapist ratings of the alliance are not interchangeable (Horvath & Bedi, 2002; Stiles, Agnew-Davies, Hardy, Barkham, & Shapiro, 1998). Each is equally associated with outcome, but likely carries distinct information because of the inherent asymmetry found in the psychotherapy relationship. This corresponds with empirical findings that patient and therapist ratings of the alliance are only moderately associated with one another (Constantino et al., 2010). Taken together, these findings suggest that the difference in patient versus therapist reports is worthy of investigation.

The practice of only using patient ratings of the alliance may be obscuring important information about the therapeutic relationship: the difference between patient and therapist perspectives may capture clinically relevant information. This notion
dovetails with research distinguishing different sources of variability in therapeutic alliance (Baldwin, Wampold, & Imel, 2007; DeRubeis et al., 2005). One is the notion, described above, that good alliances are the result of prior symptom change. Other sources are patient and therapist contributions, or characteristics that make them more or less likely to form a good collaborative working relationship. The last identified source of variability is related to the complex interaction between patients and therapists.

I suggest that the discrepancy between patient and therapist views on the relationship may approximate part of the dynamic interplay that occurs during psychotherapy. The differences between patient and therapist ratings of the alliance may indicate a lack of mutual clarity and shared understanding of the therapy, and as such may be related to poorer outcome. If therapists and patients are widely divergent in their views of how the therapy is progressing, whether they agree on the course of therapy, and how strong a bond they feel for one another, such divergence in report could represent a kind of empathic break that is not beneficial to a productive therapeutic encounter.

Researchers have made similar calls for using both patient and therapist ratings to better capture the alliance (Elvins & Green, 2008; Laurenceau, Hayes, & Feldman, 2007; Kivlighan, 2007). Kivlighan (2007) has argued that the alliance is by its nature a relational construct that is co-created by the therapist and patient, and is inappropriately operationalized when only one rater’s perspective is used. Therefore, he argues, both perspectives on the alliance are essential to understanding it fully.

Joining together, cocreation, partnership, and collaboration all speak to the dyadic nature of the alliance. The alliance is the shared perception and shared creation of the client and counselor. Using alliance ratings from only the client or counselor, even if these ratings are of perceptions of collaboration, misses the dyadic and interactional nature of the working alliance. I propose that to fully capture the
alliance researchers need to analyze the alliance as a dyadic phenomenon. (Kivlighan, 2007, p. 424)

There is a small literature addressing this theoretical issue, including studies that examined the degree of similarity (or discrepancy) between patient and therapist views on the alliance. In a study of 270 patient-therapist dyads, Hersoug et al. (2001) found evidence that similarity in patient and therapist perspectives on the alliance increased over the course of psychotherapy. Gunderson, Najavits, Leonhard, Sullivan, and Sabo (1997) found that attunement in therapist and patient ratings over the course of therapy was predictive of psychotherapy outcome. This suggests that examining a dyadic measure of the alliance over time, not just cross-sectionally, may be important to the question of more fully understanding alliance formation (Kramer, de Roten, Beretta, Michel, & Despland, 2008).

Elvins and Green (2008) note that few research studies capture interpersonal change processes that are responsible for generating good working alliances. Measuring the alliance at one timepoint may capture it descriptively, but does little to explain the interpersonal processes that occurred over time to create the alliance. It has been proposed that data collected at multiple times during psychotherapy can illuminate interpersonal processes by modeling how the construct changes throughout the therapy (Laurenceau et al., 2007). Such modeling of the change process brings researchers closer to identifying how the therapeutic alliance is formed (Constantino, Catongan, & Schut, 2002).

5. Relevance of Dyadic Processes to Depression Treatment

The dyadic interplay between patients and therapists may be of particular relevance in treatment of depression, as interpersonal issues are often salient in depressed
individuals. Researchers who have examined the interactional nature of depression also posit that interpersonal processes are at the core of the etiology and maintenance of depression (Joiner, Coyne, & Blalock, 1999; Joiner, 2002). Chronically depressed patients are characterized by interpersonal wariness, distortions in perception of interpersonal encounters, and impaired empathy and responsiveness (McCullough, 2000). This suggests that examining similarity or divergence in patient-client views of the relationship may be particularly relevant to the treatment of chronic depression. Two studies of chronic depression have provided evidence for a causal process of the prediction of outcome from alliance. In a study of an interpersonally informed, skills-based treatment for chronic depression, Klein et al. (2003) found that patient-rated early alliance was predictive of lower depression at the end of treatment, even when controlling for initial symptomatology. Arnow et al. (2013) confirmed the prediction of early alliance on depression outcome when controlling for symptom change. Taken together, these findings provide support for the notion that the alliance is important to outcome in treatment in chronic depression.

6. The Present Study

The present study addresses this gap between clinical knowledge and research methodology by using dyadic statistical modeling techniques to appropriately account for both the therapist and patient reports of the alliance and the interdependence between them (Laurenceau, Hayes, & Feldman, 2007). I model similarity in patient and therapist perspectives on the alliance, as well as the progressive attunement in their views on the alliance, to predict treatment outcome in a sample of patients undergoing treatment for
chronic depression. Based in the prior studies of alliance similarity and attunement, I put forward the following research questions and hypotheses:

**Research Question 1:** Is there evidence of alliance attunement across psychotherapy?

**Hypothesis 1:** Based on prior research finding increased alliance similarity across psychotherapy (Hersoug et al., 2001), I hypothesize that similarity in alliance ratings by patients and therapists will increase across psychotherapy, indicative of alliance attunement.

**Research Question 2:** Does similarity in patients’ and therapists’ initial alliance ratings predict outcome?

**Hypothesis 2:** Given findings that alliance measured early in psychotherapy is generally predictive of outcome (Horvath et al., 2011; Constantino et al., 2010), I hypothesize that the degree of alliance similarity measured in early therapy sessions will predict:

a) Lower levels of depression at end of therapy

b) A steeper rate of decrease in depression across psychotherapy

**Research Question 3:** Is attunement of patients’ and therapists’ alliance ratings over time predictive of outcome?

**Hypothesis 3:** Given the theorized importance of the interpersonal process in predicting treatment outcome, I hypothesize that alliance attunement over therapy will predict:

a) Lower levels of depression at end of therapy

b) A steeper rate of decrease in depression across psychotherapy

**Research Question 4:** Is the association between alliance similarity and outcome moderated by the therapy dyad’s level of early alliance? Is the association between alliance attunement and outcome moderated by the therapy dyad’s level of early alliance?
Hypothesis 4: Given that attunement is considered a healthy process, and that higher alliances early in psychotherapy are related to better outcomes as well, I hypothesize that the interaction of the two will be predictive of better outcome. That is, the relationship between attunement and outcome will be stronger in dyads with higher early alliance.

B. Methods

1. Sample and Procedure

Participants for the present study are a subsample of a large multisite study of chronic depression entitled the Research Evaluating the Value of Augmenting Medication with Psychotherapy (REVAMP) trial (Kocsis et al., 2009). The REVAMP trial had two phases, each of 12 weeks duration. In the first phase, 808 patients defined as chronically depressed received only medication according to a pharmacology algorithm, and were monitored for treatment response. Those in less than full remission (N=491) were randomized to phase 2 of the study, in which patients received either medication alone (N=96), or were treated with both medication and psychotherapy. Those receiving psychotherapy were treated either with the cognitive-behavioral analysis system of psychotherapy plus medication (CBASP; N=200) or brief supportive psychotherapy plus medication (BSP; N=195) for 12 additional weeks. For the current study, those who received the medication-alone treatment were excluded (N=395). Patients who had no alliance ratings from either patients or therapists (N=28) were also excluded, leaving a final sample of 357 psychotherapy dyads. The sample was primarily Caucasian, college-educated, and middle-aged. Specific demographic information is presented in Table 3.1. Patients received 12 weeks of psychotherapy in phase 2, and were assessed for depressive
symptomatology every two weeks. Patients and therapists rated their perceptions of the working alliance at weeks 2 ("early"), 6 ("middle"), and 12 ("late").

2. Measures

**Working Alliance Inventory-short form.** The Working Alliance Inventory (WAI; Horvath & Greenberg, 1989) is the most widely used self-report measure of the therapeutic alliance (Elvins & Green, 2008; Horvath et al., 2011). It is based on Bordin’s (1979) pantheoretical conceptualization of the therapeutic alliance. A 12-item short form adapted by Tracey and Kokotovic (1989; Appendix E) provides an acceptable and valid abbreviated version of the larger measure. Three domains (goals, tasks, and bond) of the therapeutic relationship are assessed. Questions for the goals subscale assess whether the rater believes that there is agreement on the overall problem the therapy is addressing, and the outcome that is desired. A sample item is *We are working towards mutually agreed-upon goals.* Questions for the tasks subscale assess whether the rater believes that there is agreement on whether the specific tasks within the therapy will address the goals of the therapy. A sample item is *My client believes the way we are working with his/her problem is correct (therapist rating) or I believe the way we are working with my problem is correct (patient rating).* Questions for the bond subscale assess whether the rater believes that there is a strong affective bond between the therapist and the patient. A sample item is *I appreciate my client as a person (therapist rating), or I feel that my therapist appreciates me (patient rating).* Each subscale has 4 items, with each item measured on a 7-point Likert scale (anchored at Never=1 to Always=7. Total alliance ratings summed across the three subscales have a theoretical range from 12-84, with higher values indicating a better working relationship. Reliability for the WAI was quite
good across the multiple measurement points throughout the therapy: patient alphas were
(early = .903, mid = .928, late = .926) and therapist alphas were (early = .932, mid = .963,
late = .957).

**Hamilton Rating Scale for Depression (HAMD).** The Hamilton Rating Scale for Depression (HAMD; Hamilton, 1967: Appendix E) is a 24 item therapist-rated
measure of patient depressive symptomatology (such as sleep disturbance, mood, appetite
disruption, and suicidality). The HAMD is a commonly used measure of depression in
randomized clinical trials, with items rated on a 0-2, 0-3, or 0-4 scale depending on the
symptom assessed. Items are summed to create a total score with a theoretical range from
0 to 75, with higher values indicating greater depressive symptomatology. In the current
study, the HAMD was administered every two weeks of psychotherapy, for a total of six
measurements. The HAMD showed adequate internal consistency reliability; alphas
ranging from .899 to .845 across the biweekly assessments.

**Quick Inventory of Depressive Symptomatology-Clinician Version (QIDS-C).**
The Quick Inventory of Depressive Symptomatology (QIDS; Rush et al., 2003) is the
brief version of the Inventory of Depressive Symptomatology (IDS; Rush et al., 1986,
1996: Appendix E). The clinician-rated version was used in the present study (QIDS-C)
and was administered every two weeks of psychotherapy. The QIDS-C is a 16-item
measure designed to assess depressive symptom severity in the seven-day period prior to
assessment. It assesses all symptom domains that diagnose a major depressive episode as
stated in the American Psychiatry Association Diagnostic and Statistical Manual of
Mental Disorders-4th edition (DSM-IV; APA 1994). The domains assessed are mood,
concentration, self-criticism, suicidal ideation, interest, energy/fatigue, sleep disturbance,
appetite or weight decrease or increase, psychomotor agitation or retardation. For each
domain, patients were rated on a scale of 0-3 to connote symptom frequency and severity
depending on the symptom assessed. Item responses were summed to create an index,
with higher values indicating greater depressive symptomatology. The QIDS-C has
adequate psychometric properties in this sample, with alphas ranging from .722-.785
across the 6 assessments.

3. **Analytic Plan**

There were three steps in these analyses. In the first step, a difference score (or
discrepancy) model was used to obtain estimates of the similarity between patients’ and
therapists’ alliance ratings at each time of measurement. This model was used by Lyons
and Sayer (2005) to model discrepancies in caregivers and care recipients’ ratings of
functionality. The second step used a linear growth model to estimate change in alliance
similarity (modeled as discrepancy) over 12 weeks of psychotherapy. This model is an
extension of the cross-sectional difference score model and has been used by Lyons and
Sayer (in press) to investigate change in discrepant reports of pain and other symptoms in
a sample of lung cancer patients and their spouses. This growth model tested the
longitudinal attunement hypothesis: whether therapy dyads become more similar in their
views of the alliance over the course of psychotherapy. Discrepancy can be considered an
obverse measure of similarity; that is, lower discrepancy scores are indicative of greater
similarity. If discrepancy decreases, there is evidence of greater alliance attunement over
time. In the third and final modeling step, measures of alliance similarity and attunement
were used as predictors of depressive symptom trajectories over the course of
psychotherapy.
I used the HLM7 program (Raudenbush et al., 2011) to estimate the models in each step. The key statistical advantage is that it controls for the two sources of dependency in the models: the correlation in the pair of outcome scores associated with each dyad (e.g., one for the patient and one for the therapist) and the correlation among the repeated measures over time.

C. Results

1. Hypothesis 1: Evidence of Alliance Attunement

The first research question asked if there was evidence of alliance attunement across psychotherapy in the present study, as indicated by patients’ and therapists’ ratings of the alliance becoming more similar over time. I first examined the raw scores of each therapy dyad member’s alliance scores across the therapy.

   a. Descriptive evidence of attunement. Figure 3.1 displays the mean WAI scores for both patient and therapist at weeks 2, 6, and 12. It is clear from the graph that mean discrepancies were largest at week 2 (Mean raw discrepancy = 7.759, SD = 12.469) and smallest at Week 12 (Mean raw discrepancy = 5.165, SD = 12.016). Based on this descriptive evidence I can infer that observed discrepancies decreased in magnitude over time. However, observed data contains a degree of measurement error that may distort the true underlying discrepancies. In the steps that follow, I turned to models that first estimate the true discrepancy and then, using a growth model, test whether it increases or decreases over the course of psychotherapy. Specifically, I first created measures of dyadic alliance similarity at each of the three time points (Step 1), and then tested whether they changed to become increasingly similar, on average, over time (Step 2).
b. Step 1: Creating dyadic measures of the alliance at each timepoint. The average level of alliance and the discrepancy of patients’ and therapists’ alliance ratings were modeled at weeks 2 (“early”), 6 (“middle”), and 12 (“late”) of psychotherapy for each dyad. At level-1, the basic discrepancy model is represented as a difference score model characterized by two coefficients, one capturing the dyad average (the model intercept) at each measurement occasion, and one capturing the dyad discrepancy (the slope) at each occasion. Since the three occasions were modeled simultaneously to account for the between-dyad correlations among the scores, there are six parameters at level-1.

Level 1

\[ WAI_{ij} = \]

\[ \beta_{ij}(E_{\text{Average}}) + \beta_{2j}(E_{\text{Discrepancy}}) + \]

\[ \beta_{3j}(M_{\text{Average}}) + \beta_{4j}(M_{\text{Discrepancy}}) + \]

\[ \beta_{5j}(L_{\text{Average}}) + \beta_{6j}(L_{\text{Discrepancy}}) + r_{ij} \]

Level 2

\[ \beta_1 = \gamma_{10} + u_1 \]
\[ \beta_2 = \gamma_{20} + u_2 \]
\[ \beta_3 = \gamma_{30} + u_3 \]
\[ \beta_4 = \gamma_{40} + u_4 \]
\[ \beta_5 = \gamma_{50} + u_5 \]
\[ \beta_6 = \gamma_{60} + u_6 \]

\[ WAI_{ij} \] represents the alliance score \( i \) in dyad \( j \) (\( i = 1, \ldots, 6 \) parallel scores per dyad).

Under this formulation, “Average” (\( \beta_1, \beta_2, \beta_3 \)) represent the expected value of the outcome “WAI” when the predictor “Discrepancy” is zero. The discrepancy variable was effects-coded to ensure that the zero value represented the dyad average; that is, therapist scores were coded (-.5), while patient scores were coded (.5). The model “Discrepancy”
coefficients ($\beta_2, \beta_c, \beta_{10}$) represent the discrepancy or gap in WAI between the two members of the therapy dyad. I used three parallel scales per dyad member, at each measurement occasion, to address the issue of limited information and avoid model identification problems (Cano, Johansen, & Franz, 2005). The parallel scales were the three subscale scores of the WAI.

Results from these analyses indicated significant discrepancy, on average, in patients and therapists WAI ratings at each measurement occasion. There was also significant variability around the average discrepancy, on each occasion. In addition, the dyad average WAI score was significantly different from zero at each occasion, with significant variability around the dyad average. Parameter estimates for the WAI dyad levels and dyad discrepancies are presented in Table 3.2. Post-estimation coefficients (in HLM, Empirical Bayes or EB coefficients) of the average and discrepancy for each dyad, at each occasion, were output from the HLM residual file. The EB estimates were used as dependent variables in the next step of analyses that tested whether WAI discrepancy significantly decreased over the course of the 12 weeks.

**c. Step 2: Modeling alliance discrepancy change over time.** Prior to fitting the HLM model, the data required transformation. At each occasion of measurement, patients’ alliance scores were generally higher than therapist scores. Given the coding of the discrepancy indicator as described previously, this resulted in a positive discrepancy score (when patient scores were higher) and a negative score (when therapist scores were higher. There were 290 dyads with positive discrepancies and 67 dyads with negative discrepancy scores. More importantly, there were some dyads whose discrepancy scores “flipped” signs between timepoints; for example, a therapist rated the alliance higher at
Week 2, but the patient rated it higher at Weeks 6 and 12. Since the primary question of this study was simply to test for similarity in alliance ratings, and to model convergence in similarity, I elected to take the absolute value of the discrepancies obtained for each measurement occasion to circumvent the problem of changing signs. The EB estimates of the discrepancies were transformed by first taking the absolute value, which resulted in a positively skewed distribution. I then applied a square root transformation to correct the skew. Figure 3.2 shows the histogram of the output discrepancy scores, absolute value, and root transformation for the early alliance measure. Similar transformations were applied successfully to the middle and late alliance measures.

The transformed scores were then used in a model testing for alliance similarity change over time. This was a growth curve model, with the outcome (the EB Discrepancies) measured at three timepoints (weeks 2, 6, and 12). The original coding for time was rescaled to capture the total change over the entire therapeutic period (Bolger & Laurenceau, 2013). That is, the value of the first session (week 2) was set to 0, and the value of the final session (week 12) was set to 1, with the intervening weeks spaced equally across the 0 to 1 interval. Thus, a one unit change in the linear slope parameter would represent change across the entire psychotherapy period. This resulted (after rescaling to move the zero point to represent Week 2, the onset of time in the present study) in values of 0, .4, and 1.0 for the time variable. The equations for the absolute discrepancy change model were as follows:

Level 1:

\[ \text{Alliance\_AbsDiscrepancy}(Y)_{ij} = \beta_0 + \beta_1 (\text{Time}_E) + r_{ij} \]
Level 2:

Early Alliance AbsDiscrepancy ($\beta_0$) = $\gamma_{00} + u_{0j}$

Alliance AbsDiscrepancy Change Over Time ($\beta_1$) = $\gamma_{10} + u_{1j}$

At level 1, the within-dyad model, $\beta_{0j}$ is the model intercept and represents the expected value of discrepancy early in therapy (at Session 2). $\beta_{0j}$ is the model slope and represents the change in discrepancy for a 1-unit change in time, or the change over the course of psychotherapy. At level 2, the between-dyad model, these coefficients become outcomes, where $\gamma_{00}$ represents the mean discrepancy at Week 2 and $\gamma_{10}$ represents the linear change in discrepancy over the course of 12 weeks. The $u$’s are the level-2 random effects and represent the dyad-specific deviation from the average dyad score. The variance in these $u$’s is an estimate of the heterogeneity or variability around the average dyad.

d. Hypothesis 1 results. Results from this model indicated that alliance discrepancy significantly decreased over the course of psychotherapy. Alliance discrepancy early in therapy was estimated as $\gamma_{00} = 2.829 \ (p < .001)$, which decreased significantly by the end of psychotherapy ($\gamma_{10} = -.342, \ p < .001$). Results from this model are presented in Table 3.3. It is important to note that, while the majority of psychotherapy dyads demonstrated increased alliance attunement, there were some dyads who became more divergent in their views of the alliance as the therapy progressed. As in the previous step, EB coefficients of each dyad’s estimated alliance discrepancy early in therapy and the change across the therapy were output for use in the final step of analyses. Figure 3.3 displays the results on average (Panel A) and the variability around the average (Panel B).
2. Hypotheses 2 & 3: Early Alliance Similarity and Alliance Attunement Predict Depression Outcome

I used the EB estimates generated from the previous analyses as predictors in a model that tested the two hypotheses relating alliance attunement to outcome. I modeled change in depression over time using a linear growth curve model, characterized by two parameters (intercept and slope) similar to the model in Step 2. The time variable was rescaled from 0 to 1, so that the linear change coefficient would reflect the change across the entire therapy. I then centered the time variable so that it was zero at the tenth therapy session. Thus, the intercept of this model reflects the patient’s level of depressive symptomatology at week 10 of psychotherapy.

Level 1:
\[
\text{Depression}(Y)_{ij} = \beta_{0j} + \beta_{1j} (\text{Week}_{10}) + r_{ij}
\]

Level 2:

Week 10 Depression \((\beta_0) = \gamma_{00} + u_{0j}\)

Depression Change Across Psychotherapy \((\beta_1) = \gamma_{10} + u_{1j}\)

Under this formulation, the level-1 intercept \((\beta_0)\) represents the expected value of the outcome “Depression” at Week 10. This was meant to represent depression late in psychotherapy. The level-1 slope \((\beta_1)\) represents the change in depression across the psychotherapy. At level 2, these coefficients become outcomes in separate equations, to be explained by a grand mean across all dyads plus a deviation score unique to each dyad. This model was estimated twice, first using the QIDS ratings, and then the HAMD depression ratings, as the repeated measures. These results are reported in turn below.
a. QIDS results. Results of the baseline model of depressive symptomatology as measured by the QIDS-C are displayed in column 1 (Model 1) of Table 3.4. The average depression level at Week 10 of psychotherapy was estimated to be 6.91 ($p < .001$), and there was significant negative change in depressive symptomatology, on average, across psychotherapy ($\gamma_{10} = -2.85$, $p < .001$).

I added discrepancy and attunement predictors simultaneously to the next model. Results from this model are displayed in column 2 (Model 2) of Table 3.4. Contrary to my hypotheses, discrepancy in alliance ratings early in psychotherapy was not a significant predictor of either depression level late in treatment ($\gamma_{01} = -.105$, $p = .59$), or rate of depression decline across treatment ($\gamma_{11} = .008, p = .97$). Consistent with hypotheses, I found evidence that decreased attunement (increased discrepancy) in alliance ratings across psychotherapy was related to higher depression scores by week 10 ($\gamma_{02} = 1.28$, $p = .03$) as well as slower rates of change across psychotherapy ($\gamma_{12} = 1.53$, $p = .02$). Put in terms of healthy process, increased attunement between therapists and patients in their alliance ratings across therapy was predictive of better outcome. The addition of these predictors accounted for 2.0% of the variance in depression level, and 3.2% of the variance depression change across psychotherapy.\(^9\)

b. HAMD results. Results of the baseline model of the HAMD showed an average depression level of 13.42 ($p < .001$) at session 10 of psychotherapy, and showed significant negative change in depressive symptomatology, on average, across psychotherapy ($\gamma_{10} = -5.62$, $p < .001$). There was also significant variability around these

\(^9\) These were calculated as pseudo-$R^2$ difference in unexplained variance from the baseline model to the model with predictors, over the original unexplained variance from the baseline model.
average values; all variance components were significantly different from zero. These results are displayed in Table 3.5, column 1.

I next added similarity (discrepancy) and attunement predictors to the model. Contrary to hypotheses, discrepancy in alliance ratings early in psychotherapy was not a significant predictor of either depression level late in treatment ($\gamma_{01} = -.41$, $p = .31$), or rate of depression decline across treatment ($\gamma_{11} = .49$, $p = .23$). Similarly, no significant relationship between alliance attunement over time was found for either depression level at week 10 ($\gamma_{02} = 1.58$, $p = .19$) or change ($\gamma_{12} = 1.84$, $p = .13$). Note that as in QIDS models, the direction of the attunement coefficients were positive, and the $p$ values were between .10 and .20. These results may suggest a similar, though weaker, signal in the relationship between alliance attunement and outcome as measured by the HAMD.

3. Hypothesis 4 Results: Early Alliance Level Interacts with Alliance Similarity and Attunement to Predict Depression Outcome

To test the final hypothesis that the early alliance level moderates the associations between treatment outcomes and alliance similarity and attunement, I created two interaction terms. Specifically, I created the cross-product of early alliance and alliance similarity, as well as the cross-product of early alliance and alliance attunement.$^{10}$ I fit a model that included all main effects and an interaction term and tested whether there were significant interaction effects on the intercept (the Week 10 depression score) or the slope (the change in depression over time).

---

$^{10}$ As recommended by Aiken and West (1991), the main effects (alliance similarity, alliance attunement, and the dyad average early alliance score) were mean-centered prior to creating the respective cross-product term. This removes the collinearity between the main effects and the interaction terms and allows for more sensitive detection of the interaction effect if it is present.
a. QIDS results. I found no significant improvement in model fit with the addition of the similarity interaction above a model with only the main effects of alliance similarity and alliance level, $\Delta \chi^2 (2) = 1.47, p = .48$. The model testing alliance attunement also did not provide a significantly better fit to the data than the model with the main effects, $\Delta \chi^2 (2) = 1.97, p = .37$.

b. HAMD results. Results were similarly non-significant for the depression outcome as measured by the HAMD. I found no significant improvement in model fit with the addition of the similarity interaction above a model with only the main effects of alliance similarity and alliance level, $\Delta \chi^2 (2) = .05, p = .97$. The model testing alliance attunement also did not provide a significantly better fit to the data than the model with the main effects, $\Delta \chi^2 (2) = 1.29, p = .52$.

c. Exploratory analyses. Since I hypothesized that greater agreement on the tasks and goals of psychotherapy, in particular, are likely related to a healthy process, I conducted exploratory analyses to attempt to understand characteristics of therapy dyads who viewed the alliance more similarly over time versus those who actually became more discrepant in their views of the alliance over time. To do this, I compared EB mean scores on the dyad alliance level at each of the three timepoints (early, middle, and late in therapy) for two subgroups: those dyads who became more discrepant ($N=64$) and those who became more attuned ($N=293$). The alliance levels for the group that became more attuned were higher at each measurement point than those in the group that became more discrepant over therapy. Table 3.6 presents the mean scores for each group. Thus, there was some descriptive support for the notion that greater attunement is associated with
better working alliances, though the interaction terms failed to attain statistical significance.

D. Discussion

1. Primary Findings

Findings from this study supported the hypothesis that patients and therapists became more attuned in their views of the therapeutic alliance as their psychotherapy relationship developed. This progressive attunement in alliance ratings significantly related to outcome, such that greater attunement in patients’ and therapists’ views of their alliance over time was related to lower depression levels at the end of psychotherapy and faster rates of change across psychotherapy. Implications for each of these findings are addressed below.

First, I found evidence that patients and therapists became more similar in their ratings of the alliance as therapy progressed. Their views of the alliance became more attuned, suggesting a therapeutic process of increased collaboration and clarity of the therapeutic task as the therapist and patient grew to know one another and work together over time. Counter to hypotheses, however, alliance similarity early in therapy was not predictive of outcome. This finding runs counter to the hypothesis - and findings of previous studies (Hersoug et al., 2001) - that consensus on the therapeutic process and strength of the therapeutic bond early in therapy would be associated with better treatment outcomes. By contrast, attunement in alliance ratings over time was predictive of outcome.
2. Implications of Alliance Similarity and Attunement Findings

Taken together, these findings have implications for effective therapeutic practice and clinical training. In the earliest sessions of psychotherapy, alliance agreement is not necessarily required. Working toward patient-therapist agreement and transparency on therapy goals and bond as the therapy progresses, however, does appear to be important. The findings suggest that divergent perspectives about both the therapy bond and the goals and tasks of the therapy is not in itself a cause for concern.

For example, early in therapy a depressed patient may view the goal of his therapy to be to change his unsupportive family, while a therapist may view the goal of the therapy to be increasing the patient’s motivation to address his own emotions and behaviors that may be negatively affecting familial relationships. In this case, the early alliance goal similarity would be low. Applying the findings to this example suggests, however, that a positive therapy outcome can still be achieved provided that over the course of the therapy patients and therapists come to view the goals of the therapy more similarly. These findings suggest that therapists should pay close attention to, and make explicit reference to, the patient’s view of the alliance, because doing so would allow for discussion about areas of disagreement or misunderstanding. These findings can be understood in light of the multitude of differences across therapy relationships. While perhaps there is no optimal level of alliance similarity early in treatment on average, what is important is that in the process of psychotherapy, therapists and patients move toward a greater shared understanding relative to their own perspective differences early on.
3. Implications for Relating Alliance Attunement to Alliance Level

It is clear from the results of my study that coming toward agreement on the alliance is predictive of greater therapeutic gains in terms of symptom improvement. Further support for the idea that progressive alliance convergence is important was provided by descriptive post-hoc comparisons of alliance ratings across subgroups defined by differences in attunement. Those therapy dyads with more divergent views of the alliance by the end of therapy also tended to have lower alliance ratings throughout the therapy, while those showing greater attunement had higher ratings. Higher alliance levels appear to be present in psychotherapy dyads that demonstrate this move toward attunement across the psychotherapy. This suggests that progressive alliance attunement co-occurs with—or is a function of—better alliances throughout the therapy. This claim is only speculative, however, because my study did not find statistical support for an interaction between alliance attunement and alliance level. This suggests that a more nuanced relationship between the three variables (early alliance level, attunement in alliance, and outcome) may exist than was modeled in the present study. For example, a mediational conceptualization may be more appropriate. Perhaps higher early alliances are predictive of an attunement process between patients and therapists, which in turn is predictive of outcome. Future studies should explore a potential mediational process.

4. Methodological Implications

This study also has implications for how psychotherapy researchers conceptualize and measure the therapeutic alliance. First, the study suggests that the use of both patient and therapist data—rather than patient data alone, the standard in many prior studies of the importance of the alliance—can help researchers better approximate the dynamic
interplay that occurs between patients and therapists in the arc of their psychotherapy. In addition, the collection of alliance data at multiple timepoints is indicated. At a practical methodological level, our study would not have been possible with only one rater’s perspective on the alliance, or if the alliance had only been measured at one timepoint. RCTs do not routinely include multiple repeated measures, and often do not assess both patient- and therapist-rated measures (Laurenceau et al., 2007). Because of the repeated measures and dyadic data available in the present study, I was able to model the process of psychotherapy in a more refined way than simple pre- and post- data would have allowed.

5. Future Directions and Study Limitations

Although not a primary question of this study, results did show that there was significant variability around average attunement, indicating that there may be variables that could predict more versus less attunement. It is possible, for example, that different therapeutic modalities may be more likely to lead to alliance attunement processes than others. Those that privilege direct and regular communication about the therapeutic relationship may be more likely than strictly behavioral treatments to be related to attunement patterns across the therapy. Further examination of this and other potential sources of the variability in average alliance attunement over time appear to be warranted.

Some psychotherapy researchers have found differential effects on treatment outcome for the bond versus tasks and goals components of the alliance (Webb et al. 2011). This suggests that future studies may find utility in disaggregating and testing for differential effects of the affective versus cooperative work-related components of
alliance as they relate to therapeutic outcome. It may be, for example, that agreement on
the strength of the therapeutic bond is important from the beginning of the therapy, while
the attunement process is more indicative of the complex negotiation and move toward
consensus of therapeutic tasks during the course of psychotherapy.

My findings are also unexpected in that they showed different strength of effects
for the two measures of depression used, such that only symptomatology as measured by
the QIDS was predicted by alliance attunement. There is some research that has shown
that the Inventory of Depressive Symptomatology (IDS), the longer version of the QIDS,
shows more sensitivity to changes in and severity of depressive symptomatology than the
HAMD (Helmreich et al., 2011). Another study found that the psychometric properties of
the HAMD were poor, in that responses tended to be in a more restricted range than the
scale was constructed to assess (Rush et al., 2006). Still, even with psychometric
limitations the HAMD is considered the “gold-standard” measure of clinician-rated
depressive symptomatology (Bagby, Ryder, Schuller, & Marshall, 2004). The
implications of my findings, therefore, should be interpreted with some caution.

Another limitation is that the generalizability of the results is restricted to samples
that mirror the participants in the study, who were primarily well-educated white adults.
Results may be very different in studies of disadvantaged minorities or other age groups
in the population.

Finally, this study is limited in that the primary construct of interest, the
therapeutic alliance, is measured only by self-report. In addition, although this study
accounts for the clustering inherent in dyadic data, it does not address the second issue of
clustering common in psychotherapy studies—that of multiple patients being seen by the
same therapist. Still, due to the large sample size and multiple measurements of the alliance from both patient and therapist perspectives, I believe the strengths outweigh the limitations. I argue that this study will provide both researchers and practitioners with useful information about how attunement in therapists’ and patients’ ratings of the alliance influence outcome in psychotherapy.
Table 3.1. Characteristics of study participants.

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>357</td>
</tr>
<tr>
<td>Age, $M(SD)$</td>
<td>46.96 ($11.62$)</td>
</tr>
<tr>
<td>Gender, % Female</td>
<td>56%</td>
</tr>
<tr>
<td>Ethnicity$^a$</td>
<td></td>
</tr>
<tr>
<td>African American</td>
<td>5.6%</td>
</tr>
<tr>
<td>Caucasian</td>
<td>92.4%</td>
</tr>
<tr>
<td>Other</td>
<td>5.0%</td>
</tr>
<tr>
<td>Hispanic ethnicity$^b$</td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>7.6%</td>
</tr>
<tr>
<td>No</td>
<td>91%</td>
</tr>
<tr>
<td>Education in years, $M (SD)$</td>
<td>16 (3)</td>
</tr>
<tr>
<td>Marital Status</td>
<td></td>
</tr>
<tr>
<td>Divorced, widowed or separated</td>
<td>27.7%</td>
</tr>
<tr>
<td>Married or cohabitating</td>
<td>41.7%</td>
</tr>
<tr>
<td>Never married</td>
<td>30.5%</td>
</tr>
<tr>
<td>Baseline HAMD, $M (SD), range</td>
<td>16.79 (7.82)</td>
</tr>
<tr>
<td></td>
<td>(1 - 39)</td>
</tr>
<tr>
<td>Baseline QIDS-C, $M (SD), range</td>
<td>8.90 (3.97)</td>
</tr>
<tr>
<td></td>
<td>(0 - 21)</td>
</tr>
</tbody>
</table>

$^a$Percentages are greater than 100 because participants could select more than one category.

$^b$Percentages do not total 100 due to missing data.
Table 3.2. Parameter estimates and associated standard errors for dyad average and dyad discrepancy for early, middle, and late alliance.a

<table>
<thead>
<tr>
<th></th>
<th>Coefficients</th>
<th>SE</th>
</tr>
</thead>
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<tr>
<td><strong>Fixed Effects</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Early Alliance</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Average, $\gamma_{10}$</td>
<td>63.65</td>
<td>.57</td>
</tr>
<tr>
<td>Discrepancy, $\gamma_{20}$</td>
<td>7.52</td>
<td>.82</td>
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<tr>
<td>Middle Alliance</td>
<td></td>
<td></td>
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<tr>
<td>Average, $\gamma_{30}$</td>
<td>66.38</td>
<td>.61</td>
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<tr>
<td>Discrepancy, $\gamma_{40}$</td>
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<td>.82</td>
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<tr>
<td>Late Alliance</td>
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<td></td>
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<tr>
<td>Average, $\gamma_{50}$</td>
<td>68.96</td>
<td>.62</td>
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<tr>
<td>Discrepancy, $\gamma_{60}$</td>
<td>5.28</td>
<td>.76</td>
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<td><strong>Variance Components</strong></td>
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<td></td>
</tr>
<tr>
<td>Early Average, $\tau_{11}$</td>
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<td>Early Discrepancy, $\tau_{22}$</td>
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<td></td>
</tr>
<tr>
<td>Middle Average, $\tau_{33}$</td>
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<td></td>
</tr>
<tr>
<td>Middle Discrepancy, $\tau_{44}$</td>
<td>130.69</td>
<td></td>
</tr>
<tr>
<td>Late Average, $\tau_{55}$</td>
<td>112.69</td>
<td></td>
</tr>
<tr>
<td>Late Discrepancy, $\tau_{66}$</td>
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<td></td>
</tr>
<tr>
<td>Level, $\sigma^2$</td>
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<td>6.57</td>
</tr>
<tr>
<td><strong>Model Fit Statistics</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Deviance (df)</td>
<td>31122.570 (28)</td>
<td></td>
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</tbody>
</table>

*aAll values were statistically significant, $p < .001
Table 3.3. Parameter estimates and associated standard errors for the baseline alliance discrepancy (similarity) growth model.

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<tr>
<th>Fixed Effects</th>
<th>Coefficient</th>
<th>S.E.</th>
</tr>
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<tbody>
<tr>
<td>Early Alliance Discrepancy, $\gamma_{00}$</td>
<td>2.83***</td>
<td>.06</td>
</tr>
<tr>
<td>Change in Alliance Discrepancy, $\gamma_{10}$</td>
<td>-.29***</td>
<td>.05</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Variance Components</th>
<th>Estimate</th>
<th>$\chi^2$ Statistic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Early Alliance Discrepancy, $\tau_{00}$</td>
<td>1.15***</td>
<td>2393.86</td>
</tr>
<tr>
<td>Change in Alliance Discrepancy, $\tau_{11}$</td>
<td>.32***</td>
<td>574.30</td>
</tr>
<tr>
<td>Level 1, $\sigma^2$</td>
<td>.51</td>
<td></td>
</tr>
</tbody>
</table>

Model Fit Statistics

Deviance (df) 2703.18 (6)

Note: *** $p < .001$. 
Table 3.4. Parameter estimates and associated standard errors for predicting level and change in QIDS depression scores from alliance similarity and attunement.

<table>
<thead>
<tr>
<th></th>
<th><strong>Model 1</strong></th>
<th></th>
<th><strong>Model 2</strong></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Coefficient</td>
<td>S.E.</td>
<td>Coefficient</td>
<td>S.E.</td>
</tr>
<tr>
<td><strong>Fixed Effects</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Depression Level</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Week 10 Depression, $\gamma_{00}$</td>
<td>6.91***</td>
<td>.20</td>
<td>6.91***</td>
<td>.19</td>
</tr>
<tr>
<td>Alliance Similarity Week 2, $\gamma_{01}$</td>
<td>--</td>
<td>--</td>
<td>-.10</td>
<td>.19</td>
</tr>
<tr>
<td>Alliance Attunement Over Time, $\gamma_{02}$</td>
<td>--</td>
<td>--</td>
<td>1.28*</td>
<td>.57</td>
</tr>
<tr>
<td>Depression Change</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Change in Depression Over Time, $\gamma_{10}$</td>
<td>-2.85***</td>
<td>.22</td>
<td>-2.85***</td>
<td>.22</td>
</tr>
<tr>
<td>Alliance Similarity Week 2, $\gamma_{11}$</td>
<td>--</td>
<td>--</td>
<td>.008</td>
<td>.22</td>
</tr>
<tr>
<td>Alliance Attunement Over Time, $\gamma_{12}$</td>
<td>--</td>
<td>--</td>
<td>1.53*</td>
<td>.63</td>
</tr>
<tr>
<td><strong>Variance Components</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Level, $\tau_{00}$</td>
<td>11.70***</td>
<td>2668.84</td>
<td>11.47***</td>
<td>2622.49</td>
</tr>
<tr>
<td>Change, $\tau_{11}$</td>
<td>8.07***</td>
<td>694.34</td>
<td>7.81***</td>
<td>683.44</td>
</tr>
<tr>
<td>Level 1, $\sigma^2$</td>
<td>5.57</td>
<td></td>
<td>5.57</td>
<td></td>
</tr>
<tr>
<td><strong>Model Fit Statistics</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Deviance (df)</td>
<td>10154.90 (6)</td>
<td></td>
<td>10145.72 (10)</td>
<td></td>
</tr>
<tr>
<td>$\Delta$Deviance ($\Delta$df)</td>
<td>9.18*</td>
<td>(4)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note: * $p < .10$, ** $p < .05$, *** $p < .01$, **** $p < .001$
Table 3.5. Parameter estimates and associated standard errors for predicting level and change in HAMD depression from alliance similarity and attunement.

<table>
<thead>
<tr>
<th></th>
<th>Model 1</th>
<th></th>
<th>Model 2</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Coefficient</td>
<td>S.E.</td>
<td>Coefficient</td>
<td>S.E.</td>
</tr>
<tr>
<td><strong>Fixed Effects</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Depression Level</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Week 10 Depression, ( \gamma_{00} )</td>
<td>13.42***</td>
<td>.41</td>
<td>13.42***</td>
<td>.41</td>
</tr>
<tr>
<td>Alliance Similarity Week 2, ( \gamma_{01} )</td>
<td>--</td>
<td>--</td>
<td>- .41</td>
<td>.41</td>
</tr>
<tr>
<td>Alliance Attunement, ( \gamma_{02} )</td>
<td>--</td>
<td>--</td>
<td>1.58</td>
<td>1.19</td>
</tr>
<tr>
<td>Depression Change</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Change in Depression, ( \gamma_{10} )</td>
<td>-5.62***</td>
<td>.41</td>
<td>-5.63***</td>
<td>.41</td>
</tr>
<tr>
<td>Alliance Similarity Week 2, ( \gamma_{11} )</td>
<td>--</td>
<td>--</td>
<td>.49</td>
<td>.41</td>
</tr>
<tr>
<td>Alliance Attunement, ( \gamma_{12} )</td>
<td>--</td>
<td>--</td>
<td>1.84</td>
<td>1.20</td>
</tr>
<tr>
<td><strong>Variance Components</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Level, ( \tau_{00} )</td>
<td>52.42***</td>
<td>3076.90</td>
<td>51.86***</td>
<td>3046.64</td>
</tr>
<tr>
<td>Change, ( \tau_{11} )</td>
<td>27.04***</td>
<td>666.91</td>
<td>26.40***</td>
<td>660.26</td>
</tr>
<tr>
<td>Level 1, ( \sigma^2 )</td>
<td>21.16</td>
<td></td>
<td>21.17</td>
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</tr>
<tr>
<td><strong>Model Fit Statistics</strong></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Deviance (df)</td>
<td>12833.06 (6)</td>
<td></td>
<td>12826.2411 (10)</td>
<td></td>
</tr>
<tr>
<td>( \Delta )Deviance (( \Delta )df)</td>
<td></td>
<td></td>
<td>6.82 (4)</td>
<td></td>
</tr>
</tbody>
</table>

Note: \( ^* \ p < .10, \ ^* *\ p < .05, \ ^* * *\ p < .01, \ ^* * * *\ p < .001 \)
Table 3.6. Mean alliance ratings (EB estimates) for therapy dyads that became less versus more attuned across psychotherapy.

<table>
<thead>
<tr>
<th>Alliance Level (Dyad Average)</th>
<th>Became More Discrepant Across therapy (N=64)</th>
<th>Became More Attuned Across Therapy (N=293)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Early</td>
<td>60.99</td>
<td>64.24</td>
</tr>
<tr>
<td>Middle</td>
<td>63.25</td>
<td>67.06</td>
</tr>
<tr>
<td>Late</td>
<td>65.70</td>
<td>69.67</td>
</tr>
</tbody>
</table>
Figure 3.1. Patient and therapist mean Working Alliance Inventory scores across treatment.

Note: Error bars represent standard error of the mean.
Figure 3.2. Distributions of EB estimates of alliance discrepancy values, absolute value of these discrepancies, and the transformation of the absolute values.
Figure 3.3. Average dyadic attunement trajectory (thick line) superimposed on the dyad-specific trajectories for a random sample of 40 therapy dyads.
Figure 3.4. Change in depressive symptomatology as a function of alliance attunement, displayed at low (10th percentile) and high (90th percentile) values.
CHAPTER IV
CONCLUSION TO THE DISSERTATION

The two studies of this dissertation both offer substantive evidence that attunement processes are important to health outcomes in distinct types of personal relationships. In both studies, dyadic indices of similarity were constructed from individuals within each dyad at multiple timepoints, and then were modeled over time to test for attunement processes.

Study 1 demonstrated significant attunement in the stress hormone cortisol over the early years of marriage in newlywed couples. This finding is suggestive of bidirectional spousal influence over a longer term than previously tested, as other studies have only inferred attunement processes by finding covariation in spousal cortisol over a matter of days. This study also disaggregated cortisol fluctuations into discrete parts, allowing for tests of spousal attunement not only in cortisol level, but also in physiological response to a stressor.

In Study 2, patient-therapist alliance attunement over time was predictive of better outcome for patients receiving psychotherapy for chronic depression. Here, attunement captured a communication process, in which growing similarity in perceptions of the work of psychotherapy predicted outcome. While the size of this effect was small, I argue that it has meaningful implications for clinical training and for more comprehensive measurement of the alliance construct.

Both studies utilized dyadic modeling techniques, which appropriately account for the interdependency in these data (from repeated measurements over time within individuals as well as from individuals nested within dyads). By accommodating these
sources of interdependency, the studies were able to model and test for attunement over time. Study 1 demonstrated an example of attunement as an outcome, while Study 2 demonstrated how attunement can be estimated and output for use as a predictor of another outcome of interest. In both studies, the key findings were captured through modeling of dyadically-measured constructs over time.

The major contribution of these studies was that they modeled the theorized relationship processes at the level of the dyad, rather than emphasizing individual outcomes. This dyadic level modeling of bidirectional influence in turn related to outcomes relevant to psychological health, which may have been obscured or undetectable with other modeling techniques. Taken together, these findings suggest that relationship processes have import for psychological and health outcomes.
APPENDIX A

STUDY 1 DATA FILE SETUP

Figure A.1. Level 1 data file setup for piecewise dual trajectory model at one lab visit for one couple.

<table>
<thead>
<tr>
<th>Couple</th>
<th>C0t1</th>
<th>MINT_T1</th>
<th>MPc1T1</th>
<th>MPc2T1</th>
<th>FNc1T1</th>
<th>FNc2T1</th>
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<td>.00</td>
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</table>
Figure A.2. Level 1 data file setup for multivariate outcomes model of couples’ average and discrepancy on each of the three cortisol parameters, at one lab visit for one couple.

<table>
<thead>
<tr>
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<th>Cortqual</th>
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<th>Int_DscT1</th>
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<th>Pc1_DscT1</th>
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<th>Pc2_DscT1</th>
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</table>
Figure A.3. Level 1 data file setup for multivariate outcomes model of couples’ cortisol average similarity on each of the three trajectory parameters, and change in this similarity over time.

<table>
<thead>
<tr>
<th>Couple</th>
<th>Crt_Sim</th>
<th>Ent_Avg</th>
<th>Time_Ent</th>
<th>Pc1_Avg</th>
<th>Time_Pc1</th>
<th>Pc2_Avg</th>
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</tbody>
</table>
APPENDIX B

STUDY 1 MEASURES

Perceived Relationship Quality Components (PRQC; Fletcher et al., 2000)

Each statement is answered on a 7-point Likert-type scale (ranging from 1 = not at all to 7 = extremely). Instructions are to rate the current partner and relationship on each item. Component categories are shown as subheadings (which are omitted when the scale is administered).

Relationship Satisfaction
1. How satisfied are you with your relationship?
2. How content are you with your relationship?
3. How happy are you with your relationship?

Commitment
4. How committed are you to your relationship?
5. How dedicated are you to your relationship?
6. How devoted are you to your relationship?

Intimacy
7. How intimate is your relationship?
8. How close is your relationship?
9. How connected are you to your partner?

Trust
10. How much do you trust your partner?
11. How much can you count on your partner?
12. How dependable is your partner?

Passion
13. How passionate is your relationship?
14. How lustful is your relationship?
15. How sexually intense is your relationship?

Love
16. How much do you love your partner?
17. How much do you adore your partner?
18. How much do you cherish your partner?
Dyadic Adjustment Scale (DAS, Spanier et al., 1976)

<table>
<thead>
<tr>
<th>Question</th>
<th>Frequency of Interaction</th>
</tr>
</thead>
<tbody>
<tr>
<td>23. Do you kiss your mate?</td>
<td></td>
</tr>
<tr>
<td>24. Do you and your mate engage in outside interests together?</td>
<td></td>
</tr>
<tr>
<td>How often would you say the following events occur between you and your mate?</td>
<td></td>
</tr>
<tr>
<td>25. Have a stimulating exchange of ideas</td>
<td></td>
</tr>
<tr>
<td>26. Laugh together</td>
<td></td>
</tr>
<tr>
<td>27. Calmly discuss something</td>
<td></td>
</tr>
<tr>
<td>28. Work together on a project</td>
<td></td>
</tr>
</tbody>
</table>

Note: Cohesion subscale items shown below. Items 24-28 comprise the scale, disregard item 23.
## APPENDIX C
### STUDY 1 SUPPLEMENTAL TABLES

Table C.1. Parameters for cortisol growth curve trajectory models (lab visit 1)\(^a\)

<table>
<thead>
<tr>
<th>1(^a) Lab Visit</th>
<th>Reliability Estimates</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Coefficients</td>
</tr>
<tr>
<td></td>
<td>(Robust Standard Errors)</td>
</tr>
<tr>
<td></td>
<td>Variance Components</td>
</tr>
<tr>
<td>Reliability of measure</td>
<td></td>
</tr>
<tr>
<td>Male cortisol measure 1</td>
<td>.936  .756  .750</td>
</tr>
<tr>
<td>Male cortisol measure 2</td>
<td>.936  .756  .736</td>
</tr>
<tr>
<td>Female cortisol measure 1</td>
<td>.913  .687  .668</td>
</tr>
<tr>
<td>Female cortisol measure 2</td>
<td>.914  .708  .683</td>
</tr>
<tr>
<td>Fixed Effects</td>
<td></td>
</tr>
<tr>
<td>Male cortisol measure 1</td>
<td>-1.023**  -.182***  -.138***</td>
</tr>
<tr>
<td></td>
<td>(.024)  (.014)  (.020)</td>
</tr>
<tr>
<td>Male cortisol measure 2</td>
<td>-1.033**  -.179***  -.138***</td>
</tr>
<tr>
<td></td>
<td>(.024)  (.014)  (.020)</td>
</tr>
<tr>
<td>Female cortisol measure 1</td>
<td>-1.072***  -.152***  -.081***</td>
</tr>
<tr>
<td></td>
<td>(.020)  (.012)  (.017)</td>
</tr>
<tr>
<td>Female cortisol measure 2</td>
<td>-1.082***  -.154***  -.081***</td>
</tr>
<tr>
<td></td>
<td>(.020)  (.012)  (.018)</td>
</tr>
<tr>
<td>Random Effects</td>
<td></td>
</tr>
<tr>
<td>Male cortisol measure 1</td>
<td>.100***  .029***  .061***</td>
</tr>
<tr>
<td>Male cortisol measure 2</td>
<td>.100***  .029***  .057***</td>
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<tr>
<td>Female cortisol measure 1</td>
<td>.070***  .020***  .041***</td>
</tr>
<tr>
<td>Female cortisol measure 2</td>
<td>.070***  .023***  .044***</td>
</tr>
</tbody>
</table>

Note: *p < .05, **p < .01, ***p < .001.

\(^a\) These are tables reflecting true measurement of cortisol modeling to properly model measurement error (two trajectories per spouse per lab visit, not one as reported in text).
Table C.2. Parameters for step 1 cortisol growth curve trajectory models (lab visit 2)\textsuperscript{a}

<table>
<thead>
<tr>
<th>2\textsuperscript{nd} Lab Visit</th>
<th>Reliability Estimates</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reliability of measure</td>
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<tr>
<td>Male cortisol measure 1</td>
<td>.940</td>
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<tr>
<td>Male cortisol measure 2</td>
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<tr>
<td>Female cortisol measure 1</td>
<td>.890</td>
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<tr>
<td>Female cortisol measure 2</td>
<td>.878</td>
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</tbody>
</table>

Coefficients (Robust Standard Errors)

Fixed Effects

| Male cortisol measure 1          | -1.020***  | -.147***  | -.087*** |
| Male cortisol measure 2          | -1.046***  | -.152***  | -.082*** |
| Female cortisol measure 1        | -1.064***  | -.117***  | -.090*** |
| Female cortisol measure 2        | -1.077***  | -.129***  | -.073*** |

Variance Components

| Male cortisol measure 1          | .105***    | .015***   | .029***  |
| Male cortisol measure 2          | .110***    | .018***   | .032***  |
| Female cortisol measure 1        | .054***    | .017***   | .045***  |
| Female cortisol measure 2        | .048***    | .017***   | .048***  |

Note: * $p < .05$, ** $p < .01$, *** $p < .001$.

\textsuperscript{a} These are tables reflecting true measurement of cortisol modeling to properly model measurement error (two trajectories per spouse per lab visit, not one as reported in text).
Table C.3. Predicted values for wives and husbands in log_{10} and raw score metrics, lab visits 1 & 2.

<table>
<thead>
<tr>
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<th>Log_{10} Predicted Value</th>
<th>Exponentiation to Original Raw Data Metric (µg/dl)</th>
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<td>Wives</td>
<td>Husbands</td>
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<tr>
<td><strong>Lab Visit 1</strong></td>
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<td></td>
</tr>
<tr>
<td>Entry Cortisol</td>
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</tr>
<tr>
<td>Piece 1 Slope</td>
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<td>-.1805</td>
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<tr>
<td>Piece 2 Slope</td>
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</tr>
<tr>
<td><strong>Lab Visit 2</strong></td>
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<td></td>
</tr>
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<td>Entry Cortisol</td>
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<tr>
<td>Piece 1 Slope</td>
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<td>Piece 2 Slope</td>
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<td>-.0845</td>
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</table>

Note: The normative range for salivary cortisol values is .003 – 4 µg/dl.
### APPENDIX D

#### STUDY 2 DATA FILE SETUP

Figure D.1. Level 1 data file setup for multivariate outcomes model of alliance average and discrepancy at early, middle, and late timepoints, for one therapy dyad.

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<td>.50</td>
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<td>.50</td>
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<td>0.00</td>
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<td>.50</td>
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<td>1.00</td>
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<td>.50</td>
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Figure D.2. Level 1 data file setup for growth curve model of therapy dyad alliance discrepancy over time.

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<td>2</td>
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</table>
Figure D.3. Level 1 data file setup for growth curve model of depression change over time.

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<th>QTOT</th>
<th>Time10centered</th>
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<td>10</td>
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<td>22</td>
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<tr>
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<td>9</td>
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</table>
APPENDIX E

STUDY 2 MEASURES

Quick Inventory of Depressive Symptomatology-Clinician Version (QIDS-C; Rush et al., 2003)

1. **Sleep Onset Insomnia:**
   - 0 Never takes longer than 30 minutes to fall asleep.
   - 1 Takes at least 30 minutes to fall asleep, less than half the time.
   - 2 Takes at least 30 minutes to fall asleep, more than half the time.
   - 3 Takes more than 60 minutes to fall asleep, more than half the time.

2. **Mid-Nocturnal Insomnia:**
   - 0 Does not wake up at night.
   - 1 Restless, light sleep with few awakenings.
   - 2 Wakes up at least once a night, but goes back to sleep easily.
   - 3 Awakes more than once a night and stays awake for 20 minutes or more, more than half the time.

3. **Early Morning Insomnia:**
   - 0 Less than half the time, awakens no more than 30 minutes before necessary.
   - 1 More than half the time, awakens more than 30 minutes before need be.
   - 2 Awakes at least one hour before need be, more than half the time.
   - 3 Awakes at least two hours before need be, more than half the time.

4. **Hypersomnia:**
   - 0 Sleeps no longer than 7-8 hours/night, without naps.
   - 1 Sleeps no longer than 10 hours in a 24 hour period (include naps).
   - 2 Sleeps no longer than 12 hours in a 24 hour period (include naps).
   - 3 Sleeps longer than 12 hours in a 24 hour period (include naps).

**Enter the highest score on any 1 of the 4 sleep items (1-4 above):**

- 0
- 1
- 2
- 3

= page total
5. Mood (Sad):
   - □ 0 Does not feel sad.
   - □ 1 Feels sad less than half the time.
   - □ 2 Feels sad more than half the time.
   - □ 3 Feels intensely sad virtually all of the time.

Rate either 6 or 7 (not both)  If there is no change answer number 6 only.

6. Appetite (Decreased):
   - □ 0 No change from usual appetite.
   - □ 1 Eats somewhat less often and/or lesser amounts than usual.
   - □ 2 Eats much less than usual and only with personal effort.
   - □ 3 Eats rarely within a 24-hour period, and only with extreme personal effort or with persuasion by others.
   - OR -

7. Appetite (Increased):
   - □ 0 No change from usual appetite.
   - □ 1 More frequently feels a need to eat than usual.
   - □ 2 Regularly eats more often and/or greater amounts than usual.
   - □ 3 Feels driven to overeat at and between meals.

Rate either 8 or 9 (not both)  If there is no change answer number 8 only.

8. Weight (Decrease) Within the Last Two Weeks:
   - □ 0 Has experienced no weight change.
   - □ 1 Feels as if some slight weight loss has occurred.
   - □ 2 Has lost 2 pounds or more.
   - □ 3 Has lost 5 pounds or more.
   - OR -

9. Weight (Increase) Within the Last Two Weeks:
   - □ 0 Has experienced no weight change.
   - □ 1 Feels as if some slight weight gain has occurred.
   - □ 2 Has gained 2 pounds or more.
   - □ 3 Has gained 5 pounds or more.

Enter the highest score on any 1 of the 4 appetite/weight change items (6-9 above):

□ 0
□ 1
□ 2
□ 3

+ item 5 = [__] page total.
10. Concentration/Decision Making:
   - 0. No change in usual capacity to concentrate and decide.
   - 1. Occasionally feels indecisive or notes that attention often wanders.
   - 2. Most of the time struggles to focus attention or make decisions.
   - 3. Cannot concentrate well enough to read or cannot make even minor decisions.

11. Outlook (Self):
   - 0. Sees self as equally worthwhile and deserving as others.
   - 1. Is more self-blaming than usual.
   - 2. Largely believes that he/she causes problems for others.
   - 3. Rumination over major and minor defects in self.

12. Suicidal Ideation:
   - 0. Does not think of suicide or death.
   - 1. Feels life is empty or is not worth living.
   - 2. Thinks of suicide/death several times a weeks for several minutes.
   - 3. Thinks of suicide/death several times a day in depth, or has made specific plans, or attempted suicide.

13. Involvement:
   - 0. No change from usual level of interest in other people and activities.
   - 1. Notices a reduction in former interests/activities.
   - 2. Finds only one or two former interests remain.
   - 3. Has virtually no interest in formerly pursued activities.

14. Energy/Fatigability:
   - 0. No change in usual level of energy.
   - 1. Tires more easily than usual.
   - 2. Makes significant personal effort to initiate or maintain usual daily activities.
   - 3. Unable to carry out most of usual daily activities due to lack of energy.

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= page total (total items 10-14)
15. **Psychomotor Slowing:**
   - □ 0 Normal speed of thinking, gesturing, and speaking.
   - □ 1 Patient notes slowed thinking, and voice modulation is reduced.
   - □ 2 Takes several seconds to respond to most questions; reports slowed thinking.
   - □ 3 Is largely unresponsive to most questions without strong encouragement.

16. **Psychomotor Agitation:**
   - □ 0 No increased speed or disorganization in thinking or gesturing.
   - □ 1 Fidgets, wrings hands, and shifts positions often.
   - □ 2 Describes impulse to move about and displays motor restlessness.
   - □ 3 Unable to stay seated. Faces about with or without permission.

Enter the highest score of either of the 2 psychomotor items (15 or 16 above):

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= page total.
Hamilton Rating Scale for Depression (HAMD; Hamilton, 1967)

I. Rate the item regardless of cause (except in the case of weight loss attributable to dieting).
II. Rate the item regardless of whether or not they represent a change from the patient’s usual condition.
III. Rate the item for the past week.
IV. Unless otherwise indicated, symptoms should be present for at least two days to be rated as present unless clinically significant (e.g., missing a day of work or having suicidal ideation for a single day)
V. If an individual falls in a gap between two ratings (i.e., it is too severe for a rating of 1 but doesn’t meet criteria for a rating of 2), the lower rating should be assigned.

1. Depressed Mood (dysphoria, low or blue mood).
   
   **Probes:** What’s your mood been like this past week?
   
   Have you been feeling down or depressed?
   
   If yes: In the last week, how often have you felt this way?
   
   Have you been crying at all this past week?

   □ 0  **ABSENT**
   
   □ 1  **MILD:** Feels sad, blue, unhappy 2 or 3 days.
   
   □ 2  **MODERATE:** Feels any symptoms more days than not, OR missed one day of work, OR experienced suicidal thinking during one day.
   
   □ 3  **MARKED:** Communicates depressive emotional status non-verbally (i.e. through facial expression, posture, voice and/or tendency to weep), OR reports being depressed ≥ 5 days.
   
   □ 4  **SEVERE:** Patient reports virtually ONLY depressive emotional status in verbal and non-verbal communication, OR suicidal ideation for ≥ 3 days, OR missed ≥ 3 days of work.

2. Feelings of Guilt.
   
   **Probes:** Were you particularly self-critical this past week?
   
   Did you feel that you were doing things wrong, had let others down, or let yourself down in some way?
   
   Would you say that you felt guilty about anything that you have done or not done in the past?
   
   Do you feel that you are being punished in some way?

   □ 0  **ABSENT**
   
   □ 1  Self-reproach, feels he/she has let other people/self down (has difficulty letting go of the feeling).
   
   □ 2  Guilt or rumination over past errors or sinful deeds.
   
   □ 3  Present illness is a punishment, OR has delusions of guilt.
   
   □ 4  Hears accusatory or denouncing voices and/or experiences threatening visual hallucinations.
3. Suicide.

Probes: During this past week, did you have thoughts that life is not worth living?
Did you think that you would be better off dead?
Have you had thoughts of hurting or killing yourself? If yes: What have you thought about doing?
Have you actually tried to hurt yourself?

☐ 0 ABSENT

☐ 1 Feels that life is not worth living.

☐ 2 Wishes that he/she was dead, or had thoughts about hurting self (wishes that he/she were dead but would not do it because of the kids, family, friends, etc.).

☐ 3 Suicidal ideas or gesture (has a definite plan, or cuts self, or begins to carry out suicide but stops for some reason).

☐ 4 A suicide attempt during the past week.

4. Insomnia - Early.

Probes: Did you have difficulty going to sleep during the past week?
How many nights last week did it take you longer than one-half hour to fall asleep?

☐ 0 No difficulty in falling asleep.

☐ 1 Complains of occasional difficulty falling asleep (> one-half hour) on 2-3 nights.

☐ 2 Complains of difficulty falling asleep (> one-half hour) on ≥ 4 nights.

5. Insomnia - Middle.

Probes: During the past week, after you fell asleep, what was the quality of your sleep?
Was it restless or disturbed?
How many nights was it restless or disturbed?
Did you wake up in the middle of the night?
How long did it take you to fall back to sleep?
Did you get out of bed?
How many nights did you get out of bed?
How many nights did you wake during the middle of the night?

☐ 0 No difficulty.

☐ 1 Complains of being restless and disturbed during the night on ≥ 2 nights but had no difficulty falling back to sleep.

☐ 2 Meets criteria for (1). Wakes during the night ≥ 4 times during the week and gets out of bed on ≥ 2 nights for reasons other than voiding.
6. **Insomnia - Late** (Last two hours of expected sleep).

**Probes:**

- Do you set an alarm?
- How many mornings did you wake earlier than you wanted?
- Were you able to go back to sleep, or did you just toss and turn?
- Did you just get up?

☐ 0  No difficulty.
☐ 1  Spontaneously awoke earlier than he/she wanted for reasons other than voiding, but was able to go back to sleep on 2-3 mornings.
☐ 2  Unable to fall back to sleep on ≥ 2 mornings.

7. **Work and Activities**

**Probes:**

- Did you miss any time from work or school or your activities this past week? Why?
- Have you tended to your household responsibilities this past week?
- Have you felt interested in your work and other activities or have you had to push yourself to get things done?
- Have others had to encourage you to get things done?
- Do you think you spent less time than usual (or than you should) on your work, household chores, or engaging in recreational activities?
- How much less time did you spend on these activities?
- Is there anything in your work or activities that you stopped doing altogether?

☐ 0  No difficulties.
☐ 1  Loss of interest/decreased pleasure in work/other significant activities but does not have to push oneself to do them.
☐ 2  Loss of interest/decreased pleasure in work and/or activities and having to push oneself to do them.
☐ 3  Decrease in actual time spent in work and/or activities or a decrease in productivity in 2 work/activity domains.
☐ 4  Stopped working or engaging in two or more other (non-work) domains of activity.
8. **Psychomotor Retardation** (slowness of thought and speech; impaired ability to concentrate; decreased motor activity).

   (This item should be rated based on the patient's behavior during the interview: slowness of thought and speech; impaired ability to concentrate during interview; decreased motor activity.)

   - 0 Normal thought, speech, and behavior.
   - 1 Slight retardation during interview. Movement, gesture, or verbal latencies are mildly slowed but barely noticeable; there is no interference with the pace of the interview.
   - 2 Obvious retardation during interview such as sighing, obvious difficulty concentrating and answering questions, and movement is somewhat forced and difficult; some interference with the pace of the interview.
   - 3 There is significant/great interference with the pace of the interview; interview is almost impossible to conduct.
   - 4 Complete Snoror.

9. **Psychomotor Agitation.**

   (This item should be rated based on the patient's behavior during the interview; agitated thought and speech; impaired ability to concentrate because of agitation.)

   - 0 Normal thought, speech, and behavior.
   - 1 Slight fidgetiness, moving part(s) of body such as finger tapping, or playing with a pencil or other inanimate object(s), etc.
   - 2 Obvious "playing with" hands, hair, etc., and mild difficulty sitting still; the pace of the interview is not interrupted.
   - 3 Moving about, can't sit still and moving whole torso; some interference with the pace of the interview.
   - 4 Hand-wringing, nail biting, hair-pulling, biting of lips, rubbing legs, pacing and walking about, etc.; normal pace of the interview is significantly/greatly affected.

10. **Anxiety - Psychic.**

    **Probes:** Have you been feeling especially tense or irritable this past week?
    - If yes: How often have you been feeling this way?
    - What is making you so tense? (Is patient able to pinpoint realistic concerns?)
    - How many days have you been feeling this way?

    - 0 No difficulty.
    - 1 Anxiety is present on at least 2 days but it does not interfere with daily functioning. May be experienced as subjective tension and/or irritability, or free-floating anxiety.
    - 2 Anxiety is present on at least 2 days and it mildly interferes with daily functioning (e.g., transient difficulty concentrating). May be associated with worry or rumination.
    - 3 Anxiety mildly interferes with daily functioning on at least 4 days OR anxiety significantly interferes with daily functioning on 2 or 3 days.
    - 4 Anxiety significantly interferes with daily functioning on at least 4 days. May be experienced as extreme fear or anxiety that impairs function.
11. Anxiety - Somatic
(Physiological manifestations of psychic anxiety assessed in item 10.)

Probes: In the past week, have you been bothered by any physical symptoms that sometimes go along with being nervous?

- Ask for the presence of each symptom in category below:
  - Gastrointestinal: dry mouth, flatulence (wind), indigestion, diarrhea, cramps, belching.
  - Cardiovascular: Palpitations, headaches.
  - Respiratory: Hyperventilation, sighing.
  - Urinary: Frequency Increased.
  - Sweating (excessive perspiration)

☐ 0  No difficulty.
☐ 1  MILD: 2 days this week (≥ 1 symptom).
☐ 2  MODERATE: 3 days this week (≥ 1 symptom).
☐ 3  MARKED: > 4 days this week (≥ 1 symptom).
☐ 4  SEVERE: > 4 days this week and symptom(s) interfering with work, social, and/or family functioning.

12. Somatic Symptoms - Gastrointestinal

Probes: How has your appetite been this past week?

- Have you been skipping meals or have you had to force yourself to eat?
- Have others had to urge you to eat?
- When you eat, do you enjoy it (gustatory pleasure)?

☐ 0  Normal.
☐ 1  Loss of appetite but eating meals without encouragement or heavy feelings in abdomen.
☐ 2  Need to be encouraged to eat; difficulty eating without urging.

13. Somatic Symptoms - General

Probes: How has your energy been this past week? Have you felt tired?

- How often have you felt tired?
- Were you so tired that you felt as if you were often dragging through the day?
- Did you feel heaviness in your limbs, back or head? NOTE: Do not rate "headaches." This was assessed in item 11.

☐ 0  No symptoms.
☐ 1  MILD: Heaviness in limbs, back or head; backaches, muscle aches, loss of energy or fatigability present most of the day for 2-3 days during the past week.
☐ 2  MODERATE: Any clear-cut symptom(s) > 3 days.
14. Genital Symptoms
(symptoms such as loss of libido, menstrual disturbances).

Probes: Were you interested in sex this past week? We are not asking about sexual activity, but about your interest in sex - that is, your desire for sexual activity.

☐ 0 No symptoms present.
☐ 1 MILD: Some disinterest noticed.
☐ 2 SEVERE: No interest during the past week.

15. Hypochondriasis.
(Health should be defined broadly in rating this item; that is, the patient does not necessarily have to be concerned about having a specific illness.)

Probes: During the past week, did you notice any nagging aches or pains?
- Do you find yourself often concerned about your physical well-being?
- Do you frequently complain to others about how you feel?
- Did you find yourself asking for assistance because of your health concerns?
- Have you seen a physician for any of these problems? Was a diagnosis made?

☐ 0 No symptoms present.
☐ 1 Mild somatic concerns that are non-specific and fleeting.
☐ 2 Preoccupation with health concerns (brooding preoccupation)
☐ 3 Frequent verbal complaints to others, or requests for assistance, help, etc. because of health concerns.
☐ 4 Hypochondriacal delusions (e.g., "I have leukemia," in the absence of a doctor's diagnosis).

16. Loss of Weight
(Rate by patient self-report. NOTE: Ascertain if the patient is actively dieting in an effort to lose weight. IF SO, RATE THIS ITEM "0".)

Probes: Do you think you have lost any weight this past week?
- Do your clothes fit any more loosely?

Rating by history (by self-report):

☐ 0 No weight loss.
☐ 1 Probable weight loss reported by patient.
☐ 2 Definite weight loss reported by patient.
17. Insight.
(This item can generally be rated without having to ask specific questions. However, if there is some doubt, then ask the following questions.)

Probes: Do you think you are depressed?
What do you think is wrong with you?

□ 0 Acknowledges being depressed or NOT currently depressed.
□ 1 Acknowledges being depressed but attributes the cause to bad food, climate, overwork, virus, need for rest, etc.
□ 2 Denies being depressed at all.

18. Diurnal Variation (Depression intensity varies during morning versus evening.)

(If symptoms are worse in the morning after awakening OR in the evening before going to sleep, note which time period it is and rate the severity of the variation.)

Probes: In the past week, did you notice that your mood was regularly worse at a particular time of the day?
If yes: When during the day? Morning after awakening, or evening before going to sleep?
If yes: How much worse do you feel during this time?
A "little" worse, a moderate amount or a lot worse?
How many days did you notice this variation?

CHECK THE "TIME PERIOD" IF INTENSITY VARIABILITY IS PRESENT:
□ Worse after awakening
□ Worse before going to sleep

SELECT THE APPROPRIATE DESCRIPTOR:
□ 0 No variation between morning versus evening, OR not currently present during the past week.
□ 1 MILD: A "little bit" variation for 1-2 days during the past week.
□ 2 MODERATE: "Clearly noticeable" variation for 3 days during the past week.
□ 3 SEVERE: "Very significant" variation for ≥ 4 days during the past week.
19. Depersonalization and Derealization
(These two symptoms include feelings such as a sense that one is not real (sense of reality) or a sense that a part(s) of the external environment is/are not real, respectively)

**Probes:** In the past week, have you SUDDENLY had the feeling that everything is unreal, and/or feel that you were in a dream, or cut off from others or that others were cut off from you in some strange, unrealistic way?
If yes: How often did you feel this way?

- □ 0 Symptom absent.
- □ 1 MILD: Felt this way one time during the past week.
- □ 2 MODERATE: ≥ 2 times during the past week but it didn’t interfere with functioning.
- □ 3 SEVERE: ≥ 3 times during the past week, and it interfered somewhat with work, social, or family functioning.
- □ 4 Incapacitating.

20. Paranoid Symptoms

**Probes:** During the past week, did you feel that anyone was trying to give you a hard time or hurt you?
If yes: Tell me about that.
Does it appear that others are talking about you behind your back?
If yes: Tell me about that.

- □ 0 None.
- □ 1 Suspicious (e.g., excessive concerns about interview information usage or relatively constant concern about others’ motives).
- □ 2 Ideas of reference (but they do not interfere with daily functioning).
- □ 3 Delusions of reference and persecution (i.e., a paranoid system).

21. Obsessive and Compulsive Symptoms

**Probes:** (Obsessions)
During the past week, did you have any thoughts that did not make sense to you, and that kept going through your mind? If yes: Would you give me an example?

**Probes:** (Compulsions)
During the past week, have there been things that you’ve had to do over and over again, like checking the locks on the doors several times, or repeatedly washed your hands, etc.?

**COMMENT:** Depressed patients frequently ruminate over mood-congruent depressive themes such as guilt, inadequacy, life stresses, etc. Such ruminations should be discriminated from the true obsessions and should NOT be scored for this item.

- □ 0 Absent.
- □ 1 MILD/MODERATE: clearly present but not interfering with functioning.
- □ 2 SEVERE: intrusive and incapacitating.
22. Helplessness

Probes: During the past week, did you feel that you had trouble coping on a daily basis? Were there times when you felt unable to deal with your problems? Did others have to encourage you to tend to your work, school, or household duties? Were these feelings so bad that you would say you felt helpless? Would you say that you have given up trying to cope with your life?

☐ 0 Absent.

☐ 1 MILD: Some subjective feelings of helplessness present.

☐ 2 MODERATE: Pervasive feelings of helplessness present, but does not seek or require help to tend to responsibilities.

☐ 3 MARKED: Requires urging, guidance, reassurance to accomplish chores, or personal tasks. Others have noticed and commented.

☐ 4 SEVERE: Requires physical assistance for dress, grooming, eating, or personal hygiene.

23. Hopelessness

Probes: What are your thoughts about the future? During the past week, have you had an optimistic or pessimistic view of the future? If pessimistic ask: If I attempted to reassure you that things were going to improve for you, would you believe me? How many days during the past week do you think you felt this way (pessimistic)? (If optimistic, score "0")

☐ 0 Absent.

☐ 1 Intermittent doubts that "things will improve" but can be reassured.

☐ 2 Consistently feels "hopeless", but can accept reassurance.

☐ 3 Expresses feelings of discouragement, despair, and/or pessimism about the future that cannot be dispelled.

24. Worthlessness

(ranges from mild loss of self-esteem, feelings of inferiority, self-deprecation to delusional notions of worthlessness)

Probes: During the past week, have you felt that you're as good as others whom you know and respect? Do you believe that others, in some way, are better than you? If yes: Is what you're saying a loss of self-esteem or is it worse than this? Are you saying that you've felt completely worthless to yourself and/or others?

☐ 0 Absent.

☐ 1 MILD: Indicates mild feelings of worthlessness (some loss of self-esteem such as a "little down" on himself/herself).

☐ 2 MODERATE: Indicates moderate feelings of worthlessness and an unmistakable loss of self-esteem (e.g., feels very badly about himself/herself).

☐ 3 MARKED: Differes from (2) by degree: Patient states that he/she is "no good," "inferior," etc., or describes himself/herself as worthless.

☐ 4 SEVERE: Delusional notions of worthlessness (e.g., "I am a piece of garbage," or its equivalent).
Working Alliance Inventory (WAI –SF; Tracey and Kokotovic, 1989)

Therapist version:

1. _____________ and I agree about the steps to be taken to improve his situation.

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2. My client and I both feel confident about the usefulness of our current activity in counseling.

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3. I believe _____________ likes me.

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4. I have doubts about what we are trying to accomplish in counseling.

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5. I am confident in my ability to help _____________.

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6. We are working towards mutually agreed upon goals.

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7. I appreciate _____________ as a person.

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8. We agree on what is important for _____________ to work on.

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9. _____________ and I have built a mutual trust.

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10. _____________ and I have different ideas on what his real problems are.

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11. We have established a good understanding between us of the kind of changes that would be good for _____________.

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12. _____________ believes the way we are working with her problem is correct.

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Patient version:

1. __________ and I agree about the things I will need to do in counseling to help improve my situation.

2. What I am doing in counseling gives me new ways of looking at my problem.

3. I believe __________ likes me.

4. __________ does not understand what I am trying to accomplish in counseling.

5. I am confident in __________'s ability to help me.

6. __________ and I are working towards mutually agreed upon goals.

7. I feel that __________ appreciates me.

8. We agree on what is important for me to work on.

9. __________ and I trust one another.

10. __________ and I have different ideas on what my problems are.

11. We have established a good understanding of the kind of changes that would be good for me.

12. I believe the way we are working with my problem is correct.
REFERENCES


Luborsky, L., Singer, B., and Luborsky, L. (1975). Comparative studies of psychotherapies: Is it true that “Everyone has won and all must have prizes?” *Archives of General Psychiatry, 32*, 995-1008.


