UCLA
UCLA Electronic Theses and Dissertations

Title
Using the Individual to Enhance Treatment Efficiency for Disorders of Negative Affect

Permalink
https://escholarship.org/uc/item/7h08h03b

Author
Dour, Halina

Publication Date
2016

Peer reviewed|Thesis/dissertation

eScholarship.org Powered by the California Digital Library
University of California

UNIVERSITY OF CALIFORNIA

Los Angeles

Using the Individual to Enhance Treatment Efficiency for Disorders of Negative Affect

A dissertation submitted in partial satisfaction of the requirements for the degree of Doctor of Philosophy in Psychology

by

Halina Jacqueline Dour

2016

ABSTRACT OF DISSERTATION

Using the Individual to Enhance

Treatment Efficiency for Disorders of Negative Affect

by

Halina Jacqueline Dour

Doctor of Philosophy in Psychology

University of California, Los Angeles, 2016

Professor Michelle Craske, Chair

Background: There is an over proliferation of evidence-based psychotherapies for anxiety and depression. These treatments share strategies, and yet, knowledge of which order of components is most efficacious and what mediators explain change within each strategy is limited. Greater understanding can help improve patient outcomes. ***Methods:*** Six patients with elevated levels of anxiety and/or depression were treated with a component of behavior therapy and a component of cognitive therapy using a series of multiple-baseline single-case experimental designs. Patients were randomized to receive behavior therapy first or cognitive therapy first, and they were administered self-report and behavioral measures daily, weekly, and at pre-, mid-, post- and follow-up assessments. Individual and aggregate effect size estimates were calculated for each outcome measure to assess order and incremental effects. Visual inspection was conducted to

identify potential mediators. ***Results:*** Patients improved from therapy from pre- to post-treatment. Effect size estimates suggest that patients experience greater improvement in reducing negative outcomes from receiving the behavioral component first and greater improvement in increasing positive outcomes from receiving the cognitive component first. Incremental effects followed these trends with the cognitive component demonstrating a greater incremental effect over the behavioral component in reducing negative outcomes than the converse, and with the behavioral component demonstrating a greater incremental effect over the cognitive component in increasing positive outcomes than the converse. ***Implications:*** Results suggest that patients likely exhibit greatest improvement in reducing negative outcomes from receiving behavioral therapy followed by cognitive therapy and greatest improvement in increasing positive outcomes from receiving cognitive therapy followed by behavior therapy. Further implications of mediators are discussed.

This dissertation of Halina Jacqueline Dour is approved.

Bruce L. Baker

John Carl Piacentini

Theodore Francisco Robles

Michelle Craske, Committee Chair

University of California, Los Angeles

2016

Table of Contents

Introduction …….....1

Increasing treatment efficiency……..……..……..…….…...…...3

Treatment designs…….……3

History of single-case experimental designs……..……..4

New directions……..…….……...5

Components……..5

Cognitive therapy component…….……..5

Behavior therapy component…….……...6

Current study…….…….……...7

Incremental and order effects…….……...8

Incremental effects……...…….…….....8

Order effects…….…….…….…….....9

Current study…….…….…….……..9

Mechanisms…….……10

Cognitive mechanisms…….…….…..……..10

Behavioral mechanisms…….……..…….……...11

Non-specific mechanisms…….…….……..……11

Mechanism research - limitations and gaps..……..……12

Hypotheses……..…….....13

Incremental effects……..….....13

Order effects……13

Mediators of change…….....14

Cognitive…….14

Behavioral……...14

Non-specific…….....14

Method……....14

Study design…….....14

Participants…….…….....14

Therapists…….16

Procedure…….17

Treatment…….18

Measures……..19

Participant daily measures…….....19

Participant daily measures assessing outcome…….19

Participant daily measures assessing mediators…….....19

Participant weekly measures…….…...20

Participant weekly measures assessing outcome…….....20

Participant weekly measures assessing mediators……21

Participant measures of covariates…….....27

Participant pre-, mid-, post-, follow-up measures……..…….....27

Participant pre-, mid-, post-, follow-up measures assessing mediators……..….27

Participant pre-, mid-, post-, follow-up measures assessing covariates…….……..27

Therapist session measures…….....28

Data analysis……..….....28

Overall effects…….…..28

Order effects…….…....30

Incremental effects……31

Mediators…….....32

Missing data…….....32

Results…….….....33

Overall effects…….….....33

Visual Inspection of PMPF data for overall effects…...…….....33

Visual Inspection of daily data for overall effects……...……..…33

Overall effect sizes……...……...33

Test of predictors in fixed effect model…….35

Order effects…….……..35

Visual Inspection of PMPF data for order effects……...……...35

Visual Inspection of daily data for order effects…….....35

Order effect sizes……..……..35

Test of predictors in fixed effect model…….36

Incremental effects…….37

Visual Inspection of PMPF data for incremental effects……..…….37

Visual Inspection of daily data for incremental effects……...……..37

Incremental effect sizes of first component compared to baseline….....37

Incremental effect sizes of second component compared to first component…….....38

Test of predictors in fixed effects model……...…...39

Mediators……..39

Cognitive mediators……..39

Behavioral mediators……39

Non-specific mediators……40

Discussion……41

Mediators……..56

Homework compliance……47

Emotional avoidance……48

Emotion regulation……...50

Self-efficacy and treatment expectancy……....52

Stress appraisal and frequency of automatic thinking……..54

Other potential mediating factors……55

Limitations……....57

Implications……..59

Summary……..60

Table 1……..61

Table 2……..63

Table 3……..66

Table 4……..67

Table 5……..68

Table 6……..69

Table 7……...……...……..70

Table 8……..…...……..……..71

Figure 1……..…….……72

Figure 2……..…….73

Appendix A……..……...74

Appendix B……...……...93

Appendix C……...……...98

References……...……108

Acknowledgements: This doctoral dissertation was funded by the NSF Graduate Research Foundation.

VITAE

EDUCATION

2010-2011 M.A., **University of California, Los Angeles**, Department of Psychology
Thesis: Sudden gains as a long-term predictor of treatment improvement among children in community mental health organizations

2004-2008 B.A., **Wellesley College**, *cum laude,* Department of Psychology
Honors thesis: Examining the relations among level of voice, eating attitudes, perfectionism, and internalization of the "superwoman ideal"

2006-2007 **University College London**, non-degree program, study abroad

2006 **Yale University**, non-degree Russian language program

2000-2004 **Academy for the Advancement of Science & Technology**, magnet high school

AWARDS / FELLOWSHIPS

2016 Outstanding SSCP Student Clinician Award

2016 APA Div 12 Distinguished Student Practice in Clinical Psychology Award

2015 Senior Clinical Scientist Award

2012-2015 National Science Foundation Fellowship

2012 Graduate Summer Research Mentorship

2011-2012 Ursula Mandel Stipend Award

2011-2012 UCLA Affiliates Award

2011-2012 Philip & Aida Siff Award

2011-2012 Graduate Research Mentorship

2011 Graduate Summer Research Mentorship

2010-2011 University Distinguished Fellowship

2008 Sigma Xi, Awarded membership to the Scientific Research Society

2008 Psi Chi, Awarded membership to the National Honor Society in Psychology

RELEVANT RESEARCH / WORK EXPERIENCE

2011-2016 **UCLA Anxiety Disorders Research Center**, Department of Psychology, University of California, Los Angeles, Graduate Student
Supervisor: Michelle Craske, Ph.D.

2010-2011 **Child F.I.R.S.T.**, Department of Psychology, University of California, Los Angeles, Graduate Student
Supervisor: Bruce F. Chorpita, Ph.D.

2009-2010 **Center for Anxiety and Related Disorders (CARD)**, Department of Psychology, Boston University, Research Volunteer
Supervisors: David Barlow, Ph.D., ABPP; Todd Farchione, Ph.D.

2008-2010 **Laboratory for Clinical and Developmental Research**, Department of Psychology, Harvard University, Laboratory Manager / Research Assistant
Supervisor: Matthew Nock, Ph.D.

2007-2008 **Honors Thesis,** Department of Psychology, Wellesley College
Principal Investigator
Supervisor: Sally Theran, Ph.D.

PEER-REVIEWED MANUSCRIPTS

Dour, H. J., Cohen, Z., Wiley, J., Mesri, B., Craske, M., & Rubeis, R. To ACT or to CBT? (Submitted). Using prediction models to identify which treatment to implement for an anxious patient.

Craske, M. G., Meuret, A., Ritz, T., Treanor, M., & **Dour, H. J.** (Submitted). Treatment for Anhedonia: A Neuroscience Driven Approach.

Dour, H. J. (2015). Decide How to Say it: Communication Skills and Interpersonal Effectiveness. In L. Rith-Najarian (Ed), *TheHappinessChallenge.org Intervention Manual* (Module 4). http://thehappinesschallenge.org/Manual.php

Dour, H. J., Brown, L. A., & Craske, M. (2015). The effect of valence training on return of spider fear. *Journal of Behavior Therapy and Experimental Psychiatry.*

Niles, A., **Dour, H. J.,** Stanton, A. L., Roy-Byrne, P. P., Stein, M. B., Sullivan, G., Sherbourne, C. D., Rose, R. D., & Craske, M. G. (2015). Anxiety and depressive symptoms and medical illness among adults with anxiety disorders. *Journal of Psychosomatic Research, 78,* 109-115.

Wolitzky-Taylor, K., **Dour, H. J.,** Zinbarg, R., Mineka, S., Vrshek-Schallhorn, S., Epstein, A., Bobova, L., Griffith, J., Waters, A., Nazarian, M., Rose, R., & Craske, M. G. (2014). Experiencing core symptoms of anxiety and unipolar mood disorders in late adolescence predicts disorder in early adulthood. *Depression and Anxiety, 31*, 207-213.

Dour, H. J., Wiley, J., Roy-Byrne, P., Stein, M. B., Sullivan, G., Sherbourne, C., Bystrisky, A., Rose, R. D., & Craske, M. (2013). Social support mediates anxiety and depressive symptom changes following primary care intervention. *Depression and Anxiety, 31,* 436-442.

Dour, H. J., Chorpita, B. F., Lee, S., & Weisz, J. R. & the Research Network on Youth Mental Health (2013). Sudden gains as a long-term predictor of treatment improvement among children in community mental health organizations. *Behavior Research and Therapy, 51* 564-572.

Dour, H. J., Cha, C. B., & Nock, M. K. (2011). Evidence for an emotion-cognition interaction in the statistical prediction of suicide attempts. *Behavior Research and Therapy, 49,* 294-298.

Dour, H. J. & Theran, S. A. (2011). The interaction between the superhero ideal and maladaptive perfectionism as predictors of unhealthy eating attitudes and body esteem. *Body Esteem, 8,* 93-96.

Nock, M. K., Cha, C. B., & **Dour, H. J.** (2011). Disorders of impulse-control and self-harm. In D. H. Barlow (Ed.), *The Oxford Handbook of Clinical Psychology*. New York: Oxford University Press.

Nock, M. K., Park, J. L., Finn, C. T., Deliberto, T. L., **Dour, H. J.,** & Banaji, M. R. (2010). Measuring the "suicidal mind:" Implicit cognition predicts suicidal behavior. *Psychological Science, 21,* 511-517.

Nock, M. K., Mendes, W. B., **Dour, H. J.,** & Deliberto, T. L. (Submitted). Self-harm as a form of self-help.

Using the Individual to Enhance Treatment Efficiency for Disorders of Negative Affect

In the mid-1990s, the American Psychological Association published a document calling for the development and systematic examination of standardized treatments (American Psychological Association, 1995; APA Presidential Task Force on Evidence-Based Practice, 2006; Chambless et al., 1996). The response was extraordinary with subsequent implementation of hundreds of randomized clinical trials (Web of Knowledge, 2012). These studies, which compare the outcomes of manualized therapies to other treatments or waitlist control, has been a foundational step in the development of more efficacious treatments, resulting in an explosion of evidence-based psychotherapies for anxiety and depressive disorders in the last two decades.

Unfortunately, the scale of development has become too large. Psychologists now suffer from choosing from an over proliferation of psychotherapeutic treatments for their patients. Over 500 treatments for child disorders alone (Chorpita & Daleiden, 2009; Kazdin, 2000) have been identified, and the numbers for adult treatments likely match that of children, if not are greater. Further, development of more treatments does not yield greater benefits for patients. Diminishing returns begin after adoption of a given number of therapies (Chorpita, Bernstein, & Daleiden, 2011), such that the more treatments that are created, the greater difficulty clinicians have in knowing which treatment to use and in learning and implementing each in practice.

Further, many of these psychotherapies require resources that are not available in the community setting (Gunter & Whittal, 2010; Hawley & Weisz, 2005; Weisz, Doss, & Hawley, 2005). Current knowledge of research outcomes requires time and skill. Training of psychotherapies calls for manpower, time, and cost. Knowledge of which therapies to implement for whom requires additional skill, time, and consultation. Additionally, some treatments have long durations and sessions requiring resources that are not available to clinics.

Thus, many of these evidence-based treatments are not adopted in the community. When they are implemented and tested in such settings, they demonstrate limited effectiveness, and even when administered in controlled research settings, they often yield only marginal efficacy (Cuijpers, Smit, Bohlmeijer, Hollon, & Andersson, 2010; Loerinc, Meuret, Twohig, & Craske; Westen & Morrison, 2001; Westen, Novotny, & Thompson-Brenner, 2004).

The need to generate fewer, more efficient treatments is palpable. Nearly all evidence-based treatments for anxiety and depression share common components – cognitive, behavioral, monitoring, psychoeducation, and homework, among others. The development of more efficient treatments that include essential strategies across all these evidence-based treatments would relieve clinicians of some of the burden of training and decision-making. Unfortunately, knowing which of these components (e.g., cognitive, behavioral, monitoring) and which adaptation of these components (e.g., cognitive restructuring, cognitive defusion) leads to greatest change for whom is limited.

Further, there are movements in the research domain (the most recognized of which is the Research Domain Criteria, better known as RDoC) to transition from a categorical diagnostic framework to a dimensional conceptualization of psychopathology. These changes can have great benefits on intervention development. Evidence-based theories suggest that anxiety and depression lie on continua of affect – positive and negative, such that anxiety and depression are characterized by high levels of negative affect (or threat sensitivity) and depression additionally is characterized by low levels of positive affect (or reward sensitivity). Therefore, treatments targeting deficits in these two domains rather than treatments targeting each disorder can greatly reduce the number of interventions from which clinicians have to choose (Barlow et al., 2010; Chorpita & Daleiden, 2009).

Increasing treatment efficiency

Enhancement of treatment efficiency can be achieved by the following: removal of unnecessary strategies, tweaking of current components, and the addition of more effective elements. Removal of unnecessary strategies can be achieved via examination of which components predict change. Such examination can be conducted using dismantling studies or single-case experimental designs investigating incremental effects (i.e., does a second component lead to greater gain in outcomes?) and order effects (i.e., in which order do elements have the greatest effect on outcome? Does a given order yield significant or non-significant incremental effects?).

Mechanism research can generate knowledge of how to fine-tune treatments. Mechanisms are processes through which interventions cause change. Understanding the processes that drive change in therapy allows for adjustment of strategies so that emphasis is placed on active change ingredients. Thus, translation of mechanism research to interventions can yield more efficient treatments.

Finally, adding to a treatment can greatly reduce the cost-benefit ratio of interventions. A complementary strategy may produce heightened gains such that the additional benefit outweighs the cost of that additional element. Experimental procedures testing the effect of these strategies in basic research, and further examination of incremental effects, can aid in identification of these outcome-enhancing strategies.

Treatment designs

For the past few decades, researchers most commonly have relied on randomized clinical trials to examine treatment outcomes and, less frequently, mechanisms. That is, one group of patients receiving an active treatment would be compared to another group receiving a control or

another active treatment to assess whether the active treatment of one group fared better than its comparison. After replication, if the active psychotherapy indeed did lead to better outcomes, it was considered to be an evidence-based treatment and ready to be tested in the community.

Although these methods have led to great achievements in improving clinical care in the mental health field, limitations as a result of these methods remain. First, they require vast resources, including time (often greater than a year's duration), cost, staff and therapists, and massive recruitment for large sample sizes. Second, given the aforementioned expenses and resource drain, little room remains for good idiographic assessment or frequent repeated measurement, which is necessary for good research on mechanisms. Similarly, questions of incremental and order effects are too specific and require sample sizes too large to be answered with a group design.

An alternative method is the single-case experimental design. Single-case designs require only one participant, thus one therapist and minimal resources. Given its economy, frequent repeated measurement (e.g., ambulatory monitoring), idiographic assessment, and examination of specific mechanisms are feasible. Although not a replacement to the treatment trial, the single-case design can provide answers to questions unexamined with group designs and provide a springboard for development of future treatment trials. These study designs are ideal for pilot testing multiple hypotheses.

History of single-case experimental designs

Historically, single-case designs were the universal method of understanding the mind (Barlow, Nock, & Herzen, 2009). From the study of physiology with Johannes Müller to animal research of Pavlov and Skinner and human research with Wundt's introspection, the study of psychology (and statistics) originated with the individual. With the discovery of individual

differences came the development and use of the group design, resulting in diminished implementation – and even distaste – of the single-case design (Barlow et al., 2009). The notion of the average response without consideration of individual differences became the doctrine of this era, sometimes resulting in misleading findings. A classic example is that of Eysenck's group design analyses, suggesting that psychotherapy does not work. Bergin's reexamination of Eysenck's data demonstrated that while some individuals improved, others had not, and thus the average response blunted the true effect (Barlow et al., 2009). Although group designs should and will always have a place in applied settings, single-case experimental designs can have a tremendous impact on the field via initial examination of mediators, incremental effects, and order effects. Their use in pilot studies and by clinicians who have a desire to contribute to the research field without the necessary resources for a group design can be invaluable.

New directions

The current study sought to add to the existing field by beginning to address the question of 'how can treatment efficiency be maximized for disorders of negative affect?' Using a series of single-case experimental designs, patients who reported high levels of negative affect were treated biweekly with a cognitive component and a behavioral component, during which time they completed daily, weekly, and pre-, mid-, post-treatment and follow-up assessments to assess mechanisms. The order of these components was randomized. Theoretical reasons for this design and further explanation of procedures are provided.

Components

Cognitive Therapy Component. In the 1950s and 60s, Albert Ellis and Aaron T. Beck initiated a revolution in clinical psychology with their establishment of testable psychotherapies – rational emotive therapy and cognitive therapy – that focused on patients' present cognitions

rather than their prior upbringing or unconscious processes. After years of research to support their clinical efficacy and after development of multiple adaptations, these therapies, often referred to as cognitive therapies, have become some of the leading mental health treatments among clinicians for the past three decades (J. S. Beck, 2011). Yet, how this therapy works and whether it has any incremental effect over other evidence-based therapies is unclear.

Cognitive therapy is a treatment that requires the patient to monitor, manipulate, and/or change one's relationship to his/her thoughts in service of decreasing symptoms or increasing functioning. It is based on the assumption that one's cognitive bias or interpretation of events can influence an individual's behavioral response (Ching & Dobson, 2010). Some psychologists argue that direct manipulation of cognitions is the therapy's core intervention, while others assert that changing one's relationship to his/her thoughts is another viable strategy of cognitive therapy. Nonetheless, these psychotherapies share the following evidence-based tenets: cognitive activity can be monitored, and cognitive activity affects behavior (Dobson, 2013).

Behavior Therapy Component. While Ellis and Beck were developing forms of cognitive therapy, a group of psychologists turned their attention to behavior to understand human psyche. Behaviorism arose in earlier decades from clinical and non-clinical psychologists as part of a movement to study observable behavior. From this movement, researchers like Pavlov, Watson, Skinner, Bandura, and Wolpe contributed to the development of what is now known as behavior therapy. Pavlov unintentionally established the foundation of our most influential treatment for anxiety – exposure therapy – from his work on salivating dogs and subsequent research on classical conditioning. Origins of operant conditioning began with Skinner. Bandura developed social learning theory (i.e., people learn from observable behavior) from the principles of operant and classical conditioning. Watson suggested that all behavior can be described through

learning, and he is famous for his research on fear learning with young "Albert". Wolpe developed systematic sensitization, which is one of the first forms of behavior therapy (Hothersall, 2004).

The origin of behavior therapy differs from that of cognitive therapy in that behavior therapy was developed from a scientific understanding of learning and behavior in a bottom-up formulaic process, in which mechanisms of learning were first identified and understood and then translated into an intervention to have been tested with a clinical population. On the contrary, cognitive therapy developed from a top-down process, where theory led to development of treatment, resulting in a limited understanding of change mechanisms. Behavior therapy has become a leading evidence-based treatment for anxiety and depressive disorders and may be the most commonly implemented form of evidence-based treatment for these disorders. Numerous studies have demonstrated the efficacy of exposure (e.g., Ougrin, 2011) and behavioral activation (e.g., Mazzucchelli, Kane, & Rees, 2009) for anxiety and depression. These therapies encourage patient engagement in situations that are avoided with an overall goal to increase functioning despite having difficult emotions and/or symptoms.

Current Study. Given strong evidence in support of these evidence-based therapies, a cognitive component and a behavioral component were the treatment elements utilized in this study. Examination of standardized specific (e.g., cognitive restructuring, behavioral activation) versus standardized broadly defined treatment elements (e.g., cognitive, behavioral) has benefits and limitations. Identification of specific strategies can provide information on more specific mediators or mechanisms. On the other hand, examination of broadly defined cognitive therapy or broadly defined behavioral therapy can lead to an understanding of how addressing one's thoughts or altering one's behavior – regardless of content – change outcome. Further, the latter

approach can provide answers as to whether there are mediators that are specific to each of these general components. Given that this study was intended to be the first of many that would attempt to understand how to maximize treatment efficiency, the current study utilized broadly defined, standardized protocols of these elements in an effort to understand the general changes that occur as a result of these treatments.

Incremental and order effects

Examination of incremental effects can provide information on which psychotherapy components are essential. Further, examination of order effects can demonstrate which order of elements requires the least amount of time to elicit change and leads to greatest outcome. Thus, analysis of both of these effects is critical for generating greater knowledge around treatment efficiency.

Incremental Effects. Evidence is inconclusive as to whether cognitive therapy alone leads to symptom change. Ample research suggests that cognitive therapy strategies in conjunction with behavioral strategies (e.g., cognitive behavioral therapy) and behavioral strategies alone are efficacious in treating patients with depression and anxiety (Dobson, 1989; Gloaguen, Cottraux, Cucherat, & Blackburn, 1998; Hollon, Thase, & Markowitz, 2002).

Results from component analysis and meta-analytic studies suggest that cognitive strategies demonstrate no incremental effect over behavioral strategies (Ching & Dobson, 2010; Jacobson et al., 1996; Longmore & Worrell, 2007; Raes, Koster, Loeys, & De Raedt, 2011), and for some populations, behavioral strategies demonstrate much stronger effects (Dimidjian et al., 2006; Olatunji et al., 2013). In a meta-analysis, Norton and Price (2007) found no differences in outcomes between cognitive therapy, exposure, relaxation, or their combination for anxiety.

Relatedly, definitions of cognitive therapy have ranged from the broad (e.g., any therapy that monitors thoughts) to the narrow (e.g., cognitive restructuring). Without universal definitions and labels, knowledge of whether a given strategy works, for whom it works, and how it works is limited. Often cognitive behavioral therapy is mislabeled as cognitive therapy, resulting in the aforementioned problems over whether cognitive strategies drive change. The types of cognitive strategies that are considered "cognitive therapy" also differ among researchers. For example, some psychologists argue that changing a patient's relationship to his/her thoughts is not a form of cognitive therapy.

Analysis of incremental effects of pure cognitive therapy (i.e., without inclusion of behavioral strategies) over pure behavioral therapy (i.e., without inclusion of cognitive strategies) would begin to shed light on some of these unanswered questions regarding what drives treatment change and whether cognitive therapy has an effect over an above the effect of behavior therapy.

Order Effects. To the author's current knowledge, no prior studies have examined order effects of cognitive and behavioral components for anxiety and depressive disorders.

Current Study. The current study was the first of its kind to examine order and incremental effects of broadly defined standardized cognitive therapy versus broadly defined standardized behavior therapy for disorders of negative affect. To assess order effects, participants were randomized to either cognitive first (henceforth, "cognitive group"; i.e., cognitive component first followed by behavioral component) or behavioral first (henceforth, "behavioral group"; i.e., behavioral component first followed by cognitive component). To examine incremental and order effects, participants were assessed daily, weekly, and at pre-, mid-, and post-treatment with a battery of questionnaires and behavioral tasks.

Mechanisms

An examination of mediators and mechanisms is a necessary step in answering the question of "how does a given treatment work." A mediator is any factor that explains the relation between two other factors. A mechanism is a mediator at a basic level (e.g., a biological process, intimate conversations with sibling) rather than a meta level (e.g., stress, social support) (Kazdin, 2007). In treatment research, mechanisms of change are factors that explain change in symptoms during treatment. In the current study, a variety of factors will be examined as potential mediators of change for the treatment components.

Cognitive Therapy Mechanisms. How cognitive therapy leads to symptom decrement is tentative. Theories suggest that changes in automatic thoughts (A. T. Beck, Rush, Shaw, & Emergy, 1979), beliefs, schemas, or attitudes (A. T. Beck et al., 1979; Teasdale & Fennell, 1982), attributional style (Ching & Dobson, 2010; DeRubeis et al., 1990), interpretation bias (Casey, Newcombe, & Oei, 2005; Clark et al., 1997; Wisco & Nolen-Hoeksema, 2010), threat appraisals (Jamieson, Nock, & Mendes, 2012), and awareness of cognitions (Forman, Herbert, Moitra, Yeomans, & Geller, 2007; Teasdale et al., 2001) are potential factors that explain symptom change following cognitive therapy. At best, all of these mediators are marginally supported by research as factors that explain symptom change following cognitive therapy, due to inherent methodological problems when testing these models and due to the absence of significant findings.

In the current study, cognitive therapy mediators of interest were: changes in automatic thoughts, stress appraisal, and awareness of cognitions. Each of these constructs has been suggested to be potential mediators of change in cognitive therapy for anxiety or depression.

Behavior Therapy Mechanisms. Much research has been devoted to identifying mechanisms of behavioral treatments (i.e., exposure) for anxiety disorders (Craske et al., 2008; Craske, Liao, Brown, & Vervliet, 2012). Basic clinical research and animal models have greatly added to this research base. However, nearly no studies have examined mechanisms of behavior therapies (e.g., behavioral activation, pleasant events scheduling) for depression (Dimidjian, Barrera, Martell, Munoz, & Lewinsohn, 2011). Two mechanisms hypothesized to be specific to behavior therapy are approach/avoidance tendencies (Roelofs et al., 2010; Woud, Becker, & Rinck, 2008) and self-efficacy (A. Bandura, 1983; Craske & Rachman, 1987; Williams, Dooseman, & Kleinfield, 1984). A third potential mediator is distress tolerance. Distress tolerance is the degree to which an individual can tolerate distress in the moment. Behavior therapy for disorders of negative affect often require the patient to engage in uncomfortable, stressful situations. Such situations most likely require utilization of distress tolerance to cope with the negative affect resulting from such exercises. Thus, the author hypothesized that with added engagement in these activities, distress tolerance would increase. The current study posited that these factors would be mediators of change specific to behavior therapy.

Non-Specific Mechanisms. Other mechanisms of interest in the current study that are non-specific to a certain treatment component and were of interest to the study are emotion regulation, homework compliance, and implicit associations. Cognitive strategies of cognitive therapy may be conceptualized as development of skills for emotion regulation, and behavioral exercises of behavior therapy may be conceptualized as practice of such skills; thus, the current study hypothesized that changes in emotion regulation would mediate changes in outcomes following both treatment components. In addition, homework compliance has been suggested to be a mediator of cognitive behavioral therapy (Westra, Dozios, & Marcus, 2007); it also was

examined as a non-specific mediator in the current study. Finally, although automatic or implicit associations has been suggested to be a mediator of cognitive therapy (Teachman, Marker, & Smith-Janik, 2008), behavior therapy is conceptualized to lead to changes in associative learning (Craske et al., 2008; Craske et al., 2012). Therefore, implicit associations may explain change in both of these components.

Mechanism Research - Limitations and Gaps. Many methodological flaws surface among tested mediation models, especially for cognitive therapy. An initial step in establishing treatment mediation is demonstrating that a therapy leads to greater change in outcomes than an alternative treatment (DeRubeis et al., 1990; Kazdin, 2007). Prior mediation studies often fail to meet this criterion (e.g., DeRubeis et al., 1990; Dozios et al., 2009), and research evidence is inconclusive as to whether cognitive therapy alone leads to symptom change.

Second, varying definitions of cognitive therapy result in generation of many potential mediators without strong conclusions as to what explains change. Third, many mediation studies fail to conduct repeated measurement to establish temporal precedence in their mediation models (e.g., Hoffart, 1995; Warmerdam, van Straten, Jongsma, Twisk, & Cuijpers, 2010) – a necessary step in mediation (Kazdin, 2007). For example, the direction of change between cognitions and symptoms has been poorly defined (DeRubeis et al., 1990; Johansson & Høglend, 2007). Further, most studies that have conducted strong repeated measurement to establish temporal precedence have done so with cognitive behavioral therapy rather than cognitive therapy alone (Hoffart, Sexton, Hedly, & Martinsen, 2008; Hofmann, 2004; Hofmann et al., 2007; Teachman et al., 2008).

Fourth, other methodological problems include failure to use other statistical mediation criteria defined by statisticians (Baron & Kenny, 1986; Kazdin, 2007), lack of a control

condition, and absence of intervention in the analysis (e.g., Hoffart, 1995; Kleim et al., 2013). In addition to the aforementioned methodological flaws, null results, weak effects, absence of replication, and unexamined effects often also explain why identification of mediators is limited.

In sum, the current study sought to address each of the following gaps in mechanism research for cognitive therapy: (1) no clear delineation of which component – cognitive or behavioral – leads to symptom change; (2) ambiguous definitions of these therapies; and (3) failure to conduct repeated measurement. No current statistical mediation methods have been developed for single-case designs; thus, all discussion of mediators were discussed as suggested mediation with the intention of informing larger-scale experimental group design studies that can establish statistical mediation.

Hypotheses

Hypotheses are organized by tested effects.

Overall Effects. Participants would demonstrate a decrease in symptoms, negative affect levels, and disability and an increase in positive affect levels during treatment compared to baseline as evidenced by a positive effect size.

Order Effects. Reductions in symptoms, negative affect levels, and disability and increases in positive affect levels would be greatest (effect size calculated from pre- to post-treatment) for individuals receiving the behavioral component first compared to individuals receiving the cognitive component first.

Incremental Effects. The behavior therapy component would demonstrate an incremental effect over the cognitive therapy component, whereas the reverse would not be found. That is, symptoms, negative affect levels, and disability would demonstrate additional reductions (and

positive affect levels would demonstrate additional increases) during the behavioral component following the cognitive component, whereas the reverse would not be true.

Mediators of Change. The following hypotheses are organized by specific mediation path.

Cognitive Therapy. Hypothesized mediators specific to cognitive therapy were awareness of cognitions, stress appraisal, and changes in frequency of automatic thoughts.

Behavior Therapy. Hypothesized mediators specific to behavior therapy were distress tolerance, approach/avoidance tendencies, emotional avoidance, treatment expectancy, and self-efficacy.

Non-specific. Hypothesized mediators non-specific to either cognitive or behavior therapy components were homework compliance, emotion regulation, and changes in automatic or implicit associations. Although changes in implicit associations have been suggested to be mediators for cognitive therapy in the literature, it was hypothesized that they mediate both cognitive therapy and behavior therapy. Behavior therapy requires engagement in exercises that likely restructure one's explicit and implicit cognitive associations, and thus they are conceptualized to be non-specific mediators.

Method

Study design

Participants. Participants were recruited from the UCLA Anxiety and Depression Research Clinic (ADRC). To be eligible, participants must have been 18 years or older, fluent in English, and have elevated levels of negative affect according to the Positive Affect and Negative Affect Scales (PANAS) (i.e., total negative affect score greater than 20) and Depression Anxiety Stress Scale (DASS-21) (i.e., total stress score greater than 10, total anxiety

score greater than six, or total depression score greater than 11). They also must have been willing to complete daily measures via an Internet website.

Ineligibility for the study was meeting criteria for psychotic disorder or bipolar disorder I or II according to the Mini International Neuropsychiatric Interview (MINI). Also, patients who endorsed having suicidal or non-suicidal self-injurious behavior in the last six months were excluded from participation.

Finally, if on prescribed medication, participants were required to be on a stable dose of psychopharmacological medication for at least one month prior to the start of the study. They also were strongly urged to wait until after therapy was over to change their medication if desired. Only one participant made a change to her medication prior to her fifth and ninth sessions. These changes are documented and taken into account when conducting visual inspection.

Twenty-three individuals seeking treatment for mental health problems inquired about the study and were screened over the phone for eligibility. Twelve did not meet inclusion/exclusion criteria. Two individuals did not attend the initial assessment and thus were dropped from the study. One individual dropped from the study following initial assessment because she reportedly found a treatment that required less assessment. Another individual completed the first seven sessions of treatment before dropping from therapy without providing a reason. Five participants were randomized to treatment and completed all treatment sessions. Four of the five participants completed follow-up assessment. One more participant was randomized to treatment, completed initial assessment, and completed the majority of treatment sessions (14/17 therapy sessions) before dropping from therapy due to chronic migraines that worsened and that resulted in biweekly trips to the hospital or her doctor. The five participants who completed

therapy and the participant who completed the majority of therapy were included in the current analyses (see Figure 1). Demographic and diagnostic information on the six participants can be found in Table 1.

Therapists. Therapists were the primary investigator and two other clinical psychology graduate students with 2-3 years of therapy experience. After a 1-day training led by the primary investigator and an expert in anxiety treatment, therapists were supervised weekly by this expert.

All therapists recorded videos of their sessions following consent by the client. To ensure adherence to the treatment protocols, two raters coded 15% of video recordings across participants, resulting in four coded recordings per therapist. The principal investigator randomly selected four videos from each of the three therapists to be coded for a total of twelve videos. To do so, she formulated a list of available videos for each therapist and selected four videos within each list without using a systematic random number generator.

Raters reported which skills were utilized in a given session and submitted these forms (see Appendix C) to the principal investigator. The principal investigator then counted the number of strategies in a given session that did not follow treatment protocol (e.g., session 6 of a patient who received cognitive first included a behavioral strategy; session 1 of psychoeducation contained a cognitive strategy). This number was then divided by the total number of strategies used in the session to determine a ratio score of the number of non-adherent strategies compared to the total number of strategies utilized in session (i.e., percentage of strategies that were non-adherent). Non-adherence ratios were then subtracted from 1 to generate an adherence ratio. In the current study, each therapist received the following range of adherence scores: therapist a .989, therapist b 1.000, and therapist c 1.000.

Both raters were graduate students with master's degrees in clinical psychology. They had been previously trained on the treatment protocol by both the principal investigator of the current study and her faculty advisor, and they had administered the current treatment with clients for another study the year prior. Thus, both raters were familiar with the treatment protocol; however, they were blind to study objectives and group assignment. Across two 90-minute sessions, the principal investigator trained both raters to code treatment videos using the coding sheet in Appendix C. After these training sessions, each rater coded the same twelve videos. Inter-rater reliability was 97.464%.

Procedure. All procedures were approved by the institutional review board of UCLA. Following written informed consent, eligible patients were asked to complete an initial assessment including self-report and behavioral measures and a diagnostic interview at UCLA. Patients then were asked to complete eight daily questions via a website for two weeks prior to starting therapy to establish a baseline and to ensure that clients would complete daily measures during therapy. Patients were informed that if they did not complete a minimum of 10/14 days of daily questions, they would not qualify to begin treatment. All participants who completed initial assessment also completed the minimum of baseline daily measures.

The primary investigator, who conducted initial assessment, then randomized each patient to receive either a cognitive component first or a behavioral component first followed by the remaining component. Based on this randomization, the patient was assigned to a therapist. Each therapist treated one patient from each group so that each therapist treated a total of two patients.

All participants, regardless of condition, received psychoeduation as their first session. Then, participants received eight sessions of either cognitive therapy or behavior therapy

followed by another eight sessions of the other treatment component. Thus, the total length of treatment was seventeen sessions. Patients were permitted to practice skills learned from the first nine sessions during the remaining eight sessions; however, the therapist did not assign or monitor this practice. Only homework related to the current module was assigned and monitored. Session durations were 50 minutes, and session frequency was biweekly. Participants completed daily and weekly measures throughout therapy, as well as during a mid-treatment assessment prior to Session 10 and a post-treatment assessment following Session 17 (Table 2). Finally, all participants were contacted one-month following post-treatment assessment to complete another battery of self-report and behavioral measures. Participants received $50 in cash for their completion of the follow-up assessment.

Treatment. Therapists were trained in standardized behavior and cognitive therapy principles from the Treatment for Affect Dimensions (TAD) protocols, which are currently being tested in a randomized clinical trial. These protocols – Positive Affect Treatment (PAT) and Negative Affect Treatment (NAT) – contain evidence-based principles for treating anxiety and depression. Behavioral strategies of PAT are most similar to pleasant events scheduling, and behavioral strategies of NAT are most similar to exposure therapy. Cognitive strategies of PAT include finding the silver lining, taking responsibility, and imagining the positive. The cognitive strategies in NAT are overestimation of odds, balancing responsibility, and decatastrophizing. In the current study, therapists were allowed to utilize principles from cognitive components of both TAD treatments for cognitive therapy; however, they could only utilize principles from the behavioral component of either NAT or PAT as predetermined by the primary investigator and faculty advisor following a case conceptualization. This was done to ensure that clients and therapists did not confuse these behavioral strategies as they are very similar but have different

objectives. Regardless of condition, the first session of psychoeducation contained education on mood cycles, rationale for cognitive and behavioral therapy, as well as a discussion around common thought patterns (e.g., catastrophizing, cognitive fusion) that clients may be experiencing.

Measures

Measures were administered daily, session-by-session and at pre-treatment, mid-treatment, post-treatment and at follow-up assessment (Table 2).

Participant Daily Measures. The following eight items were completed daily by the participant.

Participant daily measures assessing outcome.

Affect Levels. In order to obtain daily measures of negative affect levels, the following standard monitoring questions were asked: "On a scale of 0-10 (with 10 being the highest), what was your average level of anxiety or fear in the past 24 hrs?" "On a scale of 0-10 (with 10 being the highest), what was your average level of depression or sadness in the past 24 hrs?" "On a scale of 0-10 (with 10 being the highest), what was your average level of distress in the past 24 hrs?" "On a scale of 0-10 (with 10 being the highest), what was your average level of anger, guilt, or shame in the past 24 hrs?" "On a scale of 0-10 (with 10 being the highest), what was your average level of happiness, love, curiosity, interest, and gratitude in the past 24 hrs?"

Participant daily measures assessing mediators.

Awareness of Cognitions. Two items from the Philadelphia Mindfulness Scale (PHLMS; Cardaciotto, Herbert, Forman, Moitra, & Farrow, 2008) assessing awareness of cognitions were utilized and adapted for daily administration to participants. The first item assessed general awareness: "I am aware of what thoughts are passing through my mind". The second item

assessed awareness specific to thoughts related to mood: "I am aware of the thoughts I am having when my mood changes". These items had strong factor loadings (.48 and .56, respectively) on the awareness subscale of the PHLMS (Cardaciotto et al., 2008). Response options were (1) "not at all today", (2) "rarely today", (3) "sometimes today", (4) "often today", and (5) "very often today".

Treatment Expectancy. An item measuring participants' expectancy that they would improve from treatment was administered. Participants were asked, "Please rate how certain you are that you will improve from therapy," which will be rated on a 0-100 scale (0="will not at all"; 100= "highly certain will") (Bandura, 2006).

Participant Weekly Measures. The following measures were administered weekly. Given that therapists met with participants biweekly, half of the measures were administered on the first session of the week whereas the other half were administered on the second session of the week. Computerized tasks were administered during separate sessions. All of this was intended to reduce participant burden.

Participant weekly measures assessing outcome.

Depression Anxiety Stress Scale (DASS-21; Lovibond & Lovibond, 1995). As another measure capturing levels of negative affect, the DASS-21 was administered weekly. Participants rated 21 items related to past week depression, anxiety, and stress on a 0-3 Likert scale from 0="Did not apply to me at all" to 3="Applied to me very much, or most of time". The DASS-21 has been shown to have good to excellent reliability and is a valid measure of depression and anxiety (Antony, Bieling, Cox, Enns, & Swinson, 1998).

Positive Affect and Negative Affect Scales (PANAS; Watson, Clark, & Tellegen, 1988). In order to assess levels of negative affect throughout treatment, participants completed the

PANAS weekly. It is a 20-item scale, measuring levels of positive and negative affect. Participants were asked to indicate the extent to which ten positive emotions (e.g., interested, excited) and ten negative emotions (e.g., jittery, afraid) reflected how they felt over the past week. Items were rated on a 1-5 Likert scale with 1="very slightly or not at all" to 5="extremely". The measure has demonstrated high reliability and strong validity (Crawford & Henry, 2004).

Sheehan Disability Scale (SDS; Sheehan, 1983). To assess whether disability level changed throughout treatment and whether there was relative change in disability as a result of each treatment component, participants were administered the SDS weekly. The SDS is a 3-item measure that assesses the degree to which symptoms affect past week functioning in the work, leisure, and family domains. This measure has shown good to adequate reliability (including test-retest reliability, >.7) and validity (Leon, Shear, Porten, & Klerman, 1992).

Participant weekly measures assessing mediators.

Approach/Avoidance Task (AAT; Rinck & Becker, 2007). The AAT is a computerized task intended to measure one's implicit approach/avoidance tendencies. These tendencies have been conceptualized to be one's basic responses to the motive systems of emotional responding (Klein, Becker, & Rinck, 2011). A series of images eliciting positive and negative affect were presented individually in the center of a computer screen. These images were obtained from the International Affective Picture System (IAPS; Lang, Bradley, & Cuthbert, 1999) and were chosen based on their mean ratings of arousal and pleasure. For an image to be chosen as a positive or negative picture, its mean pleasure or arousal rating was required to fall between two and eight to ensure that negative images would elicit arousal without being too gruesome and

positive images would elicit pleasure without being too erotic. The mean pleasure and arousal ratings for the chosen set of images were 2.2-7.44 for pleasure and 2.66-6.46 for arousal.

The participant was instructed to push the “e” or “i" key as quickly as possible following image onset based on the background color of the picture frame – “e” for blue and “i" for green. Pressing the “e” key caused the image on the screen to reduce in size, and pushing the “i" key caused the image to increase in size, as if the keys had control over whether the image was moving closer or further away from the participant. Participants underwent 20 practice trials and 60 experimental trials. During practice trials, participants were given feedback if they push the incorrect key. 50% of experimental trials contained negative affect images, of which 50% had a blue border, and 50% of experimental trials contained positive affect images, of which 50% had a blue border. Thus, half the experimental trials were ‘compatible’ trials (i.e., negative images that reduced in size, positive images that increased in size) and the remaining half were ‘incompatible’ trials (i.e., positive images that reduced in size, negative images that increased in size). Order of experimental trials was randomized.

All trials with an incorrect response (3.25% of experimental trials) and all trials with response times below 150 ms (0% of experimental trials) or above 1500 ms (2.50% of all correct response trials with the majority – 57.14% – of these slow response times accounted for by patient C3; 1.23% of correct response trials accounted by the other five participants) were excluded from analyses, consistent with procedures of Krieglmeyer, Deutsch, De Houwer, De Raedt (2010). Mean response latencies were calculated for the four groups of trials: (1) negative images that reduced in size, (2) negative images that increased in size, (3) positive images that reduced in size, and (4) positive images that increased in size.

Acceptance and Action Questionnaire (AAQ-II; Bond et al., 2011). The AAQ-II is a measure of psychological flexibility, which is defined as the ability to accept and be present with difficult internal and external experiences as well as engage in behavior consistent with one's values. It is a 10-item self-report measure that asks participants to rate how true each statement is for them on a Likert scale from 1="Never true" to 7="Always true." Higher scores indicate greater emotional avoidance and less psychological flexibility. The AAQ-II was adapted in the current study to instruct participants to rate how accurate these statements have been for them in the past week. This measure has been shown to be a valid and reliable measure with good internal consistency (α=.84) and test-retest reliability (.79-.81) (Bond et al., 2011).

Implicit Associations Test: Core Beliefs. The Implicit Association Test (IAT; A. G.. Greenwald, McGhee, & Schwartz, 1998) is a computerized, timed measure of implicit associations between pairs of constructs. That is, it tests whether certain automatic associations (e.g., flowers and pretty, insects and ugly) are stronger than other associations (e.g., flowers and ugly, insects and pretty). The current study utilized this paradigm to measure individuals' implicit associations between core-beliefs and themselves.

Within a trial, two target words and two attribute words appeared at the top corners of the screen with attribute words appearing below the target words, and a stimulus – a descriptor word – appeared in the middle of the screen (Figure 2). Participants were instructed to hit the "e" key if the stimulus was a descriptor of either the target or attribute word on the left of the screen, and they were instructed to hit the "i" key if the stimulus was a descriptor of either the target or attribute word on the right of the screen. Instructions indicated that participants should go as quickly as possible without making mistakes. If a mistake was made, an "X" appeared below the stimulus word and the participant had to enter the correct answer before moving on to the next

trial. A trial ended when the client hit the correct key, and reaction times (ms) were recorded. Intervals between trials were 250 ms. All of the aforementioned procedures were consistent with recommendations of Nosek et al. (2007).

The current study had three target word pairs: (1) "Failure" / "Success", (2) "Unlovable" / "Lovable", and (3) "Helpless" / "Able", and the attribute word pair across all trial blocks was "Me" / "Not Me". Nosek et al. (2007) recommended that for each target pair, there should be seven response blocks: (a) 20 practice trials with only target pairs on the screen, (b) 20 practice trials with only attribute pairs on the screen, (c) 20 trials with both target and attribute words on the screen, (d) 40 trials identical to block "c", (e) 20 practice trials with only target pairs on the screen but target words switch sides of the screen from block "a", (f) 20 trials with both target and attribute words on the screen but attribute words switch sides of the screen from block "c", and (g) 40 trials identical to block "f". I eliminated block "e" and reduced the number of trials within each response block to: (a) 10, (b) 10, (c) 5, (d) 20, (f) 5, and (g) 20 to reduce participant burden. In addition to the three different trial target word pairs, an additional practice target word pair (i.e., "insect" / "flower"; attribute words: "good" / "bad") was presented at the start of the task. Thus, the total amount of trials across all blocks in the current study was 280 (compared to 6800 trials if the current study complied with recommendations of Nosek et al., 2007). Participants completed this measure during pre-treatment, mid-treatment, post-treatment and follow-up assessments, as well as once per week, in addition to other computerized and self-report measures. Therefore, to reduce participant burden, a reduced number of trials was administered.

"D" scores were calculated based on recommendations of Nosek et al. (2007). Practice response blocks (i.e., "a" and "b") were excluded from analyses. Within a target pair, if a

participant had more than 10% of trials with latencies less than 300 ms, the target pair was excluded from analysis. Also, any trials with response latencies greater than 10,000 ms were eliminated from the response block. No participants had trials with latencies less than 300 ms or greater than 10,000 ms and thus no trials were excluded. Mean response times across trials of each remaining block - "c", "d", "f", and "g" - were calculated. Difference scores of "c" and "f" and of "d" and "g" were then computed. The author then calculated the standard deviation of reaction times of blocks "c" and "f" and the standard deviation of reaction times of blocks "d" and "g". Finally, difference scores were divided by their standard deviation, and these two values were then averaged. Again, all of these calculations are consistent with recommendations of Nosek et al. (2007).

Positive D scores indicated that the participant had a stronger association between the positive construct and him/herself (i.e., success/me, lovable/me, able/me), and negative D scores indicated that the participant had a stronger association between negative construct and him/herself (i.e., failure/me, unlovable/me, helpless/me). Thus, increases in D scores across treatment are expected. The original IAT has demonstrated good internal consistency, test-retest reliability (ranging from 0-40 days between test and retest), and validity (Nosek, Greenwald, & Banaji, 2007). Although practice test-effects have been demonstrated with the IAT (Nosek et al., 2007), the author included a control condition (i.e., insects/flowers, good/bad) to examine its pattern of change over time in comparison to the other blocks.

General Self-Efficacy Scale (GSES; Schwarzer & Jerusalem, 1995). The GSES is a 10-item self-report measure of perceived self-efficacy around problem solving, coping, and goal achievement. Example items were: "I can always manage to solve difficulty problems if I try hard enough," "I am confident that I could deal efficiently with unexpected events," and "If I am

in trouble, I can usually think of a solution." Items were rated on a 4-point Likert scale from 1="Not at all true" to 4="Exactly true." The scale has demonstrated high reliability (α=.76-.90) and strong validity (Schwarzer & Born, 1997).

Stress Appraisal Measure (SAM; Peacock & Wong, 1990). The SAM is a 28-item measure of stress/threat appraisal. Items were rated on 1-5 Likert scale (1="not at all" – 5="extremely") as a measure of participant's perception of an identified problem in his/her life. Examples included "Is this going to have a positive impact on me?", "How eager am I to tackle this problem?", and "Is this a totally hopeless situation?" This measure has seven subscales: threat, challenge, centrality, self-control, other control, uncontrollability, and stressfulness. Theoretically, only the threat subscale measures negative appraisal of all situations, and thus it was the only subscale utilized in visual analyses. The overall measure has demonstrated good reliability and validity (Peacock & Wong, 1990).

The Computerized Mirror Tracing Persistence Task (MTPT-C; Strong et al., 2003). The MTPT-C is a behavioral measure of distress tolerance. Modified from the original paper and pencil version (Quinn, Brandon, & Copeland, 1996), the MTPT-C requires the participant to trace the outline of various shapes on the computer screen using a computer mouse. Participants were presented with three consecutive shapes and were asked to move the mouse so that the red dot on the computer screen traced the outline of each shape on the computer screen. S/he was informed that the mouse and dot on the screen move in the opposite direction. Further, each time s/he incorrectly outlines the shape or stops for a duration of more than 2 seconds, s/he was required to start again. On the third figure, the participant had the option of quitting the task by pressing a key on the keyboard. The time (sec) that it takes for the participant to click this key was the measure of persistence or distress tolerance.

Participant measures of covariates.

Changes in Therapy/Medication Form. To ensure that clients' medications remained stable or that changes in medication or outside therapy was captured, a form was administered weekly. Participants indicated whether there had been any changes to their medication regimen in the past week, and if so, what they were.

Participant Pre-Treatment, Mid-Treatment, Post-Treatment, and Follow-up Measures. This battery of measures included weekly measures in addition to the assessments listed below.

Participant pre-treatment, mid-treatment, post-treatment, and follow-up measures assessing mediators.

Automatic Thoughts. The Automatic Thoughts Questionnaire Revised (ATQ-R; Hollon & Kendall, 1980; Kendall, Howard, & Hays, 1989) and the Anxiety Self-Statements Questionnaire (ASSQ; Kendall & Hollon, 1989) was administered to assess automatic thoughts. The questionnaires have 30-32 items that measure self-statements around depression, inability to cope, anxiety, and positive affect (Safren et al., 2000). Items were rated on a 5-point Likert scale from 1="not at all" to 5="all the time" or "very much". Each measure has demonstrated good psychometric properties (Kendall & Hollon, 1989; Kendall et al., 1989).

Difficulties in Emotion Regulation Scale (DERS; Gratz & Roemer, 2004). The DERS is a 36-item self-report measure of emotion regulation. Items were rated on a 1-5 Likert scale from 1="Almost never" to 5="Almost always". It has demonstrated great reliability and is a valid measure of emotion regulation capabilities (Gratz & Roemer, 2004).

Participant pre-treatment, mid-treatment, post-treatment, and follow-up measures assessing covariates.

Demographic Questionnaire. A demographic questionnaire was administered at initial assessment. It included age, gender, race, English fluency, education level, marital status, prior treatment history, presence of medical conditions, and medication regimen.

Mini International Neuropsychiatric Interview – Version 7.0.0 (MINI; Sheehan et al., 1998). The MINI is brief structured diagnostic interview that assesses presence of DSM-5 disorders. The measure has demonstrated comparable reliability and validity statistics to other diagnostic interviews (e.g., SCID-P, CIDI) despite its shorter administration time (Lecrubier et al., 1997; Sheehan et al., 1998; Sheehan et al., 1997). It was administered at initial assessment to assess presence of psychological disorders.

Therapist Session Measures.

Homework compliance. A form measuring homework compliance was completed by therapists following each session. Therapists were to report whether the client "fully completed", "partially completed", or "not at all" completed homework.

Data analysis

Overall Effects. Scores of each measure were graphed for every participant to visually inspect trends across treatment: (1) pre-, mid-, post-treatment, and follow-up (PMPF) data, and (2) daily data (Appendix Figures A1-A6).

Overall effect sizes were examined by calculating an effect size of treatment compared to baseline using the DHPS macro developed by Shadish et al. (2014) in SPSS v23.0. The DHPS macro calculates Hedges' g and its associated variance for single-case experimental designs. The statistic assumes no trend and normal distribution of outcome. Histograms of outcome data were generated in SPSS v23.0 to identify forms of distributions (Appendix Figure B1). All outcome

data, with the exclusion of daily distress, were normally distributed. Thus, distress was excluded from all effect size calculations.

The DHPS macro also assumes no trend. Although calculators that model trend are advantageous, most effect size calculators for single-case designs assume no trend or require the statistician to specify the form of that trend, which often is unknown. Further, unlike other single-case effect size calculators (Moeyaert, Ferron, Beretvas, & Van den Noortgate, 2014; Rindskopf, 2014; Swaminathan, Rogers, & Horner, 2014), the DHPS uses a comparable effect size statistic as those reported in the literature for group design studies, allowing for comparison of effect sizes across single-case and group design studies (Shadish, 2014). It also accounts for the number of cases in the calculation, number of time points within a case, ratio between case variance and total variance, and lag-one autocorrelation (Shadish, 2014).

PMPF and session-by-session data were merged into a single dataset (henceforth, session-by-session data). In the current study, there were multiple outcomes of session-by-session and daily data. Multiple outcomes of the same comparison is a form of dependency; therefore, an aggregate effect size was calculated.

Prior to aggregation, outliers of effect sizes were examined with a Grubbs Test using the Outliers package in R. Potential outliers were noted, but they were not removed given the very few effect sizes aggregated. A fixed effects weighted average was calculated to aggregate effect sizes across outcomes. A fixed effects model was conducted, rather than a random effects model, because the methods of each single-case experimental design were the same and sample size per effect size did not differ. Further, sample size was too small to run a powered random effects model.

The weighted average effect was calculated according to the following formula:

$$\overline{T_\bullet} = \frac{\sum_{i=1}^{k} w_i T_i}{\sum_{i=1}^{k} w_i}$$

, where $\overline{T_\bullet}$ is the weighted average effect size, T is the effect size for each outcome, w is the weight of each effect size, and k is the number of subjects.

The formula for conditional variance was:

$$v_\bullet = \frac{1}{\sum_{i=1}^{k} (\frac{1}{v_i})}$$

, where v is the variance associated with each effect size.

The Q test was conducted to determine homogeneity of effects. Note that due to the small number of effect sizes, the power of Q is low. The formula for Q was:

$$Q = \sum_{i=1}^{k} [\frac{(T_i - \overline{T_\bullet})^2}{v_i}]$$

To conduct the fixed effects model, the Metafor package in R was run. Both individual and aggregate effect sizes were reported, because the current study was interested in comparing effect sizes across outcomes. Further, the low power for aggregation using the fixed effects model encouraged reporting of individual effect sizes.

Influence diagnostics then were run on the fixed effects model using the Metafor package in R. Any effect sizes that were suggested to be influential were discussed and removed, and the model was re-run without the influential effect size.

Order Effects. Scores of each measure were graphed for every participant to visually inspect trends within groups: (1) PMPF data, and (2) daily data (Appendix Figures A1-A6).

Order effects were examined by comparing effect sizes of treatment outcomes (e.g., levels of anxiety, depression, and stress; level of disability; levels of positive and negative affect) among patients who were randomized to receive the behavioral component first compared to the cognitive component first. Using the DHPS macro developed by Shadish et al. (2014), effect sizes of overall effects within each group (i.e., behavioral first [BF], cognitive first [CF]) were calculated. Fixed effects weighted averages were calculated in the same format as overall effects.

To determine whether effect sizes differed between the BF and CF groups and between session-by-session and daily data, a fixed effects model was conducted with group (0=BF, 1=CF) and frequency of data collection (0=session-by-session, 1=daily) as predictors.

Incremental Effects. Scores of each measure were graphed for every participant to visually inspect trends within groups: (1) PMPF data, and (2) daily data (Appendix Figures A1-A6).

Incremental effects were examined by: (1) calculating the effect size of outcomes of the first component compared to baseline, and then (2) calculating the effect size of outcomes of the second component compared the first component, within each group, using the DHPS macro (2014) in SPSS v23.0. Prior to aggregation, outliers of effect sizes were examined independently for timing of incremental effect (i.e., from baseline to first component, from first component to second component). Individual and aggregate effect sizes were reported. Fixed effects weighted averages were calculated separately for each group using the Metafor package in R.

To determine whether effect sizes were dependent on group status (BF vs CF), on frequency of outcome assessment (session-by-session vs daily), and on timing of incremental effect (first component compared to baseline vs second component compared to first session), a fixed effects model was conducted with group (0=BF, 1=CF), frequency of data collection

(0=session-by-session, 1=daily), and timing of incremental effect (0=first component compared to baseline, 1=second component compared to first session) as predictors. Influence diagnostics were conducted using the Metafor program in R. Any influential effect sizes were removed, and the fixed effects model was re-run to compare outcomes with and without the effect size.

Mediators. Given that no formal statistical mediation analyses for single-case designs exist, the current study could not assess statistical or causal mediation. However, data could be inspected visually to examine relations of variables across treatment as an initial step in identifying temporal precedence and patterns of variability; thus, *potential* mediation can be suggested. To assess presence of potential mediation, session-by-session scores of each mediator variable were graphed adjacent to the outcome variable (i.e., DASS-Total, which is the total number of anxiety, stress, and depressive symptoms). Trends were examined for presence of potential mediation. Suggested mediation was based on: (1) the mediator demonstrating some variability and trend across treatment, and (2) the mediator demonstrating a similar pattern of scores as that of the outcome measure, or preferably, (3) the mediator demonstrating a similar pattern of scores as that of the outcome *and* this change seemed to occur prior to change in outcome.

Missing data

One participant (patient C3) completed 14 out of 17 sessions. Analyses that included post-assessment outcome scores utilized this participant's outcome scores from her 13th and 14th sessions. All other participants completed every session measure and larger assessments. Two participants – one from each group – did not complete follow-up assessments. Effect size calculations require a minimum of three participants per group; therefore, no effect size calculations were conducted for follow-up assessments. During participant B1's and C3's initial

assessments, the AAT could not be administered due to a glitch in the program. No compensatory measures were taken, because visual inspection of this measure at baseline was not conducted.

The percent of daily data that was not missing across participants was 77.91% (range 35.80% – 100%). Note, however, that patient B1 had completed only 35.80% of daily questions; thus, the estimate of completed daily data across participants would be higher if this participant were excluded or considered an outlier. These missing data were handled through listwise deletion in effect size estimates and were not included in visual inspection.

Results

Overall effects

Visual Inspection of PMPF Data for Overall Effects. Visual inspection of trends across treatment and follow-up suggest that most patients (n=4) generally improved across treatment (Appendix Figure A1). Two participants (C2 and C3) did not seem to demonstrate improvement across most outcomes. Trends of patient improvement did not seem to differ between groups (Appendix Figure A1).

Visual Inspection of Daily Data for Overall Effects. Visual inspection of trends across daily data suggest that no improvements were made from baseline to post-treatment (Appendix Figures A2-A6), and trends of patient improvement did not seem to differ between groups (Appendix Figures A2-A6). Slopes and variances of daily data within each phase across treatment are presented in Table 3.

Overall Effect Sizes. Overall effect size estimates for session-by-session data were positive, indicating that patients improved as a result of therapy. The largest and smallest effect sizes corresponded to the PANAS negative scale (G=3.95, VarG=.567) and PANAS positive

scale (G=.354, VarG=.131), suggesting that patients exhibited the largest effect in reductions in negative emotion severity and smallest effect in the intensity of positive emotion growth. Effect sizes of DASS-T and SDS-T were .723 (VarG=.107) and .436 (VarG=.072), respectively.

Overall effect size estimates for daily data were positive, indicating that patients generally improved as a result of therapy. Effect sizes for daily outcomes were as follows: G=.254 (VarG=.019) for anxiety levels, G=.168 (VarG=.016) for depression levels, G=.025 (VarG=.017) for anger, guilt, and shame levels, and G=.111 (VarG=.008) for positive emotion levels.

Examination of outliers identified the PANAS negative effect size of 3.95 to be an outlier (G=2.441, U=.028, p=.000). The aggregate overall effect of treatment was .186 (SE=.055, p=.000, CI=.079-.294). Effect sizes were not homogenous Q(df=7)=31.238, p=.000. Influence diagnostics (Appendix Figure B2) suggest that the PANAS negative scale, daily anger, guilt, and shame, and daily positive emotions were three influential effect sizes. The PANAS negative effect size was identified as influential by the rstudent plot (i.e., a plot of externally standardized residuals) and the QE.del plot (i.e., plot of chi square test for homogeneity if effect size were omitted). Daily positive emotions was identified as influential by the rstudent plot (i.e., a plot of externally standardized residuals), cook.d plot (i.e., number of standard deviations the set of predicted values changes when the effect size is left out) and the hat plot and weight plots where daily positive emotions had the largest influence and weights. Finally, daily anger, guilt, and shame was identified as influential by the rstudent (i.e., a plot of externally standardized residuals) and QE.del (i.e., plot of chi square test for homogeneity if effect size were omitted) plots. Given that none of these effect sizes demonstrated considerable influence across most of these diagnostics, none were excluded from aggregation.

Test of Predictors in the Fixed Effects Model. Heterogeneity may have been due to frequency of data collection (i.e., session-by-session versus daily data). Therefore, frequency of data collection was added as a predictor in the fixed effects model. Although frequency of data collection was a significant predictor (Table 6), tests of residual heterogeneity still were significant Q(df=6)=22.092, p=.001. Order of components may be accounting for some of the remaining heterogeneity.

Order effects

Visual Inspection of PMPF Data for Order Effects. No order effects were identified within groups for: (1) symptoms of anxiety, depression, and stress; (2) levels of negative affect, (3) level of disability in work, social life, and family life; or, (4) levels of positive affect (Appendix Figure A1).

Visual Inspection of Daily Data for Order Effects. No order effects were identified within groups for changes in negative affect (anxiety, depression, anger, distress, guilt, shame) or positive affect (happiness, curiosity, love, interest, gratitude) (Appendix Figures A2-A6).

Order Effect Sizes. Effect sizes between groups differed by outcome (Table 4). Larger effect sizes were present in the BF group for the DASS-T, PANAS negative, and SDS-T. On the contrary, a larger effect size of the PANAS positive was evident in the CF group. The BF group had double and triple the magnitude of effects for the DASS-T, PANAS negative, and SDS-T, respectively, and the CF group had a six-fold increase in magnitude of improvement on the PANAS positive scale (Table 4).

Effect sizes between groups also differed for daily data, but these differences were much smaller than differences between group effect sizes for session-by-session data. All other trends were in favor of the BF group.

Aggregate effects were as follows. The BF aggregate effect size was .141 (SE=.094, p=.133, CI=-.043-.326). Effect sizes were not homogenous Q(df=7)=23.942, p=.001. Influence diagnostics identified the PANAS negative and daily anger, guilt, and shame as influential effect sizes (Appendix Figure B3). The PANAS negative effect size was identified as influential by the rstudent plot (i.e., a plot of externally standardized residuals) and the QE.del plot (i.e., plot of chi square test for homogeneity if effect size were omitted), whereas daily anger, guilt, and shame was identified as influential by the cook.d plot (i.e., number of standard deviations the set of predicted values changes when the effect size is left out) and QE.del plot (i.e., plot of chi square test for homogeneity if effect size were omitted). Daily positive affect had the highest weight and influence according to the hat and weight plots. Given that none of these effect sizes demonstrated considerable influence across most of these diagnostics, none were excluded from aggregation. The CF aggregate effect size was .165 (SE=.085, p=.051, CI=-.001-.332). Effect sizes were homogenous Q(df=7)=7.579, p=0.371.

Test of Predictors in the Fixed Effects Model. To test whether group status and frequency of data collection were significant predictors, a fixed effects model was run. Although frequency of administration remained a significant predictor, group status (i.e., BF vs CF group) was not a significant predictor (Table 7); test of residual heterogeneity was significant QE(df=13)=26.888, p=.013, suggesting that more factors likely account for the unexplained variance.

These combined results suggest that although visual inspection did not detect any differences between groups, analysis of individual effect sizes demonstrates trends of larger effects for the majority of measures – especially in reducing negative outcomes - when the behavioral component preceded the cognitive component. These trends were not confirmed by

aggregation; however, with greater power, group status may have emerged as a significant predictor.

Incremental effects

Visual Inspection of PMPF Data for Incremental Effects. No incremental effects were identified within groups for: (1) symptoms of anxiety, depression, and stress; (2) levels of negative affect, (3) level of disability in work, social life, and family life; or, (4) levels of positive affect (Appendix Figure A1).

Visual Inspection of Daily Data for Incremental Effects. No incremental effects were identified within groups for changes in negative affect (anxiety, depression, anger, distress, guilt, shame) or positive affect (happiness, curiosity, love, interest, gratitude) (Appendix Figures A2-A6).

Incremental Effect Sizes of First Component Compared to Baseline. The incremental effects of each component can be found in Table 5. For session-by-session data, incremental effects of the behavioral component compared to baseline were more than double to triple the size of incremental effects of the cognitive component compared to baseline in reducing negative outcomes. The incremental effect of the cognitive component compared to baseline was greater than the incremental effect of the behavioral component compared to baseline in enhancing positive affect by a factor of six.

For data collected daily, no clear pattern of results emerged for incremental effects of the behavioral component compared to baseline and of the cognitive component compared to baseline across outcomes and groups (Table 5).

Aggregate effects were as follows. The BF aggregate incremental effect size of the first component compared to baseline was .075 (SE=.102, p=.462, CI=-.124-.274). Effect sizes were

not homogenous Q(df=7)=17.569, p=.014. Influence diagnostics identified the PANAS negative and daily anger, guilt, and shame as influential effect sizes (Appendix Figure B3). The PANAS negative effect size was identified as influential by the rstudent plot (i.e., a plot of externally standardized residuals) and the QE.del plot (i.e., plot of chi square test for homogeneity if effect size were omitted), whereas daily anger, guilt, and shame was identified as influential by the cook.d plot (i.e., number of standard deviations the set of predicted values changes when the effect size is left out) and QE.del plot (i.e., plot of chi square test for homogeneity if effect size were omitted) (Appendix Figure B4). Given that neither of these effect sizes demonstrated considerable influence across most of these diagnostics, neither was excluded from aggregation. The CF aggregate incremental effect size of the first component compared to baseline was .053 (SE=.072, p=.458, CI=--.088-.194). Effect sizes were homogenous Q(df=7)=4.602, p=.708.

Incremental Effect Sizes of Second Component Compared to First Component. For session-by-session data, there were greater incremental effects of the cognitive component following a behavioral component for the DASS-T, PANAS negative, and SDS-T outcomes, but there was a greater incremental effect of the behavioral component following a cognitive component for the PANAS positive outcome (Table 5). Effects sizes more than doubled between groups.

For daily data, effects sizes did not reflect any consistent pattern (Table 5).

Aggregate effects were as follows. The BF aggregate incremental effect size of the second component compared to first component (i.e., incremental effect of the cognitive component compared to behavioral component) was .219 (SE=.089, p=.014, CI=.044-.394). Effect sizes were homogenous Q(df=7)=6.463, p=.487. The CF aggregate incremental effect size of the second component compared to first component (i.e., incremental effect of the

behavioral component compared to cognitive component) was .199 (SE=.095, p=.037, CI=.012-.385). Effect sizes were homogenous Q(df=7)=4.058, p=.773.

Test of Predictors in the Fixed Effects Model. None of the predictors (frequency of data collection, group status, timing of incremental effect) were significant in the model (Table 8). Test of residual heterogeneity was not significant QE(df=28)=29.667, p=.379.

These combined results suggest that although visual inspection did not detect any differences between groups, analysis of individual effect sizes suggest trends for larger incremental effects in reducing negative outcomes for the cognitive component compared to behavioral component and for the behavioral component compared to baseline. These trends were confirmed by aggregation.

Mediators

Cognitive Mediators. Awareness of cognitions was hypothesized to be a cognitive mediator. Unfortunately, there was nearly no variability in daily ratings, suggesting that this measure of awareness did not mediate outcomes (Appendix Figures A7-A8). Stress appraisal of threat and frequency of maladaptive thoughts also were hypothesized to be cognitive mediators. Overall, there appeared to be minimal change from pre- to post-treatment in degree of stress appraisal, but there was a decline in overall maladaptive thoughts across treatment regardless of component. Nevertheless, both of these factors appear to be potential mediators only during the cognitive component (Appendix Figures A9-A10), suggesting that both of these factors are potential component-specific mediators of cognitive therapy. Client B3's report of taking medication at sessions five and nine did not appear to affect these results.

Behavioral Mediators. Behavioral observation revealed that client C3 did not attempt to complete the distress tolerance task at any time point. Thus, her data were coded as 0 sec for

every session. Although hypothesized as a behavior therapy specific mediator, changes in distress tolerance were not associated with changes in symptoms throughout treatment according to visual analysis, suggesting that this factor is not a behavior therapy or non-specific mediating factor (Appendix Figure A11). Similarly, changes in approach-avoidance tendencies did not appear to relate to symptom changes across treatment (Appendix Figure A12). Indeed, for both of these measures, there was little variability across treatment.

Nearly all patients demonstrated reductions in emotional avoidance across treatment. Although it was hypothesized that avoidance tendencies would be a potential behavior therapy mediator, trends suggest a strong correlation between changes in emotional avoidance and changes in symptoms across group status and treatment components. Therefore, self-reported emotional avoidance may be a non-specific mediator of treatment change (Appendix Figure A14). Likewise, trends suggest that treatment expectancy (compared to Appendix Figure A5) and general self-efficacy – hypothesized to be behavioral component specific mediators – appear to be non-specific mediators (Appendix Figures A15-A16). General self-efficacy demonstrated growth from pre- to post-treatment for half of the patients (n=3), and treatment expectancy appeared to demonstrate slight improvement from pre- to post-treatment across most participants (n=4). Client B3's report of changes in medication at sessions five and nine did not appear to have influence on these relations.

Non-specific Mediators. As hypothesized, trends demonstrated that regardless of group status or treatment component, increased homework completion was associated with greater reduction in symptoms (Appendix Figure A13); thus, homework completion may be a non-specific mediator of treatment change. Client B3's report of medication change at sessions five

and nine did not seem to decrease client's homework compliance, and it did not seem to have an effect on the overall relation between these two variables.

Emotion regulation was hypothesized to be a non-specific mediator. Visual analysis demonstrated overall reductions in use of maladaptive emotion regulation strategies from pre- to post-treatment. However, change in symptoms appeared to emerge prior to change in emotion regulation (e.g., B1, C1, C3) (Appendix Figure A17). Therefore, while symptoms and emotion regulation may be related, changes in emotion regulation did not appear to be a potential mediator.

Also, although predicted to be a non-specific mediator, implicit associations did not seem to demonstrate any understandable trend across time. The "control/practice" condition was expected to become slightly more positive across time due to practice effects, but even the D scores of this subtest were scattered in an unidentifiable pattern. Clearly, this measure of implicit associations did not relate to outcome and is not suggested to be a potential mediator (Appendix Figure A18).

Discussion

The current study examined the incremental and order effects of behavior and cognitive therapy components and potential treatment mediators at a nuanced level using a series of single-case experimental designs and frequent repeated measurement of multiple constructs in an effort to understand how to maximize efficiency of cognitive behavioral therapies for anxiety and depressive disorders. Order effects, incremental effects, and potential component-specific and treatment mediators were identified.

The majority (n=4) of patients improved from therapy. The overall effect size for all participants ranged from g=.354-3.95 depending on which session-by-session outcome was

measured. These effect sizes were comparable to those in the literature for cognitive therapy, behavior therapy, and cognitive behavioral therapy compared to control. Weighted effect size estimates reported in Norton and Price's (2007) meta-analysis, which examined the efficacy of cognitive behavioral therapy, cognitive therapy, and behavior therapy for anxiety, were .72-2.23. Weighted effect sizes of cognitive behavioral therapy, cognitive therapy, and behavior therapy for depression ranged from .41-.99 in the meta-analysis conducted by Cuijpers et al. (2013) with a mean effect size of .53 after correcting for publication bias. Participants of the current study met criteria for anxiety and/or depressive disorders. The upper and lower bounds of the effect sizes reported in the literature fall within the range of individual effect sizes of session-by-session data reported here.

Note, however, that overall effects of daily outcomes were much lower (g=.026-.254). These small effect sizes may be due to the type of assessment or frequency of measurement. Indeed, frequency of data collection was a significant predictor of variability in effect sizes. Each of these outcomes was measured with a single item and was not previously validated for its validity or sensitivity to change. Further, patients were asked to complete these items daily. Over time, due to patient fatigue, patients may have had less motivation to respond honestly or accurately.

In terms of order effects, patients who received the behavioral component first – compared to cognitive component first – demonstrated superior outcomes through reduction in symptoms and negative emotions, whereas patients who received the cognitive component first – compared to behavior component first – demonstrated greater increases in positive emotions across treatment. Incremental effects from the addition of the cognitive component following the behavior component were evident in terms of reducing negative outcomes. In contrast, a greater

incremental effect from the addition of a behavioral component was observed in terms of increasing positive affect. Incremental and order effects both suggest that most improvement in reducing negative outcomes results from a behavioral component preceding a cognitive component and that greatest improvement in increasing positive outcomes results from the cognitive component preceding a behavioral component.

Basic science from Pavlovian fear conditioning and extinction suggest that the greatest learning during exposure therapy develops when expectancy of the CS-US pairing is maximally violated (Craske et al., 2012). Given that the objective of many cognitive strategies is to diminish this expectancy (e.g., reducing overestimation, decatastrophizing), cognitive therapies that precede behavior therapy theoretically would reduce the efficacy of exposure-based therapies. Pleasant events scheduling – a behavioral treatment utilized for one patient during behavior therapy of the current study – can be construed as a form of exposure therapy because patients are forced to expose themselves to situations that they appraise to be too difficult or uncomfortable. Therefore, results from the current study corroborate this theory and are the first to demonstrate order effects. Depending on outcome, effect sizes of the behavioral first group were double or triple the size of effects of the cognitive first group in reducing negative outcomes (see Table 4). On the contrary, only the cognitive first group demonstrated improvements in positive outcomes (g=.132-.657), whereas the behavioral first group demonstrated slight decreases in positive outcomes (g=-.007 - -.090).

Both incremental and order effects confirm that reductions in negative outcomes occur when the behavioral component precedes the cognitive component and that enhanced positive outcomes occur with the reverse order. It may be that these effects are capturing the same phenomenon. Perhaps order effects are accounted for by the incremental effect of the cognitive

component following the behavioral component for negative outcomes, and perhaps they are accounted for by the incremental effect of the behavioral component following the cognitive component for positive outcomes. Another question is whether the incremental effect of the cognitive component following a behavioral component is due to the behavioral component being less effective than the cognitive component in reducing negative outcomes or is due to an interaction of the two components resulting in enhanced outcomes when implemented in that order. A similar question can be posited for the incremental effect of behavior therapy following cognitive therapy in enhancing positive outcomes.

Incremental effect sizes of first component compared to baseline suggest that much larger incremental effects emerge from the behavioral component in reducing negative outcomes for session-by-session data, whereas much larger incremental effects result from the cognitive component in enhancing positive outcomes for session-by-session data. These trends do not hold for daily data, for which no clear pattern across outcomes and components emerged. This pattern of results suggest that incremental effects of the cognitive component compared to behavioral component in reducing negative outcomes are not due to greater efficacy of the cognitive component. Likewise, incremental effects of the behavioral component compared to cognitive component in enhancing positive outcomes are not due to greater efficacy of the behavioral component. Rather, it is likely that the combination and order of these components has an effect on outcome.

Most group design studies comparing cognitive therapy to behavior therapy in head to head trials suggest that both treatments are equally effective in reducing negative outcomes or that behavioral therapy is more effective in reducing negative outcomes (Dimidjian et al., 2006; Norton & Price, 2007; Olatunji et al., 2013), and that the combination of the two are the most

effective (Norton & Price, 2007). These studies further support the notion that the pattern of incremental effects in the current study may be due to the benefits received from the combination of these two components.

Another question remaining is why each order was associated with a different pattern of results. That is, why does cognitive therapy preceding behavior therapy increase positive outcomes and behavior therapy preceding cognitive therapy decrease negative outcomes? Behavior therapy preceding cognitive therapy may decrease negative outcomes through the expectancy mechanism described earlier; behavior therapy that precedes cognitive therapy facilitates heightened expectancy, which results in a greater decrement of symptoms than would be present had the converse order been implemented. Cognitive therapy preceding behavior therapy may facilitate enhanced positive affect via greater therapeutic alliance and an absence of anticipatory anxiety associated with behavior therapy. This would leave room for more positive emotions, such as relaxed, calm, excited, hopeful, optimistic, and secure. Group design studies that use more advanced statistical methodology should begin to tease apart what are the mechanisms leading to these effects.

Nonetheless, both groups demonstrated incremental effects over their counterparts, suggesting that each component of cognitive behavioral therapy has a role in eliciting change. Surprisingly, however, there was a greater incremental effect for the cognitive component compared to the behavioral component (g=1.091 for DASS-T; g=.774 PANAS negative; g=.971 for SDS-T) than there was for the behavioral component compared to the cognitive component (g=.377 for DASS-T; g=.423 for PANAS negative; g=.264 for SDS-T) despite research suggesting the contrary. One meta-analysis suggested no differences in overall outcomes between these treatments (e.g., Norton & Price, 2007), which provides evidence against presence

of incremental effects. Also, results from component analysis and other meta-analytic studies suggest that cognitive strategies demonstrate no incremental effect over behavioral strategies (Ching & Dobson, 2010; Jacobson et al., 1996; Longmore & Worrell, 2007; Raes et al., 2011). Further, for some populations, behavioral strategies demonstrate much stronger effects (Dimidjian et al., 2006; Olatunji et al., 2013). The unexpected results in the current study may be due to the nature of our analysis and type of cognitive therapy. Many prior studies have examined differences in outcomes between therapies but do not statistically compute and compare effect sizes and do not examine these effects with pure cognitive therapy (absence of behavioral strategies) (Ching & Dobson, 2010; Jacobson et al., 1996; Longmore & Worrell, 2007; Raes et al., 2011). This combination of factors may have resulted in this effect surfacing here but was hidden in previous studies. Another possibility, of course, is that the effect is a result of type I error due to an outlier present in our small sample size (n=3 per group).

Mediators

The current study aimed to identify potential mediators of each component and of cognitive behavioral therapy, more generally. Very few studies examined mediation of these components in a single study or properly defined the treatment components to ensure that they did not overlap (DeRubeis et al., 1990; Dozios et al., 2009). The current study builds upon prior work by examining potential mediation of clearly-defined cognitive and behavioral strategies for anxiety and depression. Given the use of the single case design, no formal establishment of treatment mediation or identification of mechanisms could be made, because no statistical methods have been developed for these purposes. However, due to the feasibility of frequent repeated measurement of multiple constructs in single-case experimental designs, examining which constructs may explain change in outcomes through visual analysis can be beneficial in

providing knowledge about how to construct future randomized controlled trials examining mediation. Thus, the current study sought to identify suggested mediators that demonstrated similar change patterns to the outcome across therapy.

Factors that emerged as potential treatment mediators were homework compliance, emotional avoidance, general self-efficacy, and treatment expectancy. Potential component-specific mediators were stress appraisal and automatic thoughts.

Homework Compliance. Consistent with hypotheses, homework compliance – as assessed by therapists – was identified as a potential non-specific treatment mediator. Visual analysis demonstrated that as homework completion increased throughout treatment, total number of symptoms declined for all patients, which is consistent with prior work that has demonstrated a link between homework compliance and CBT outcomes for depression (Addis & Jacobson, 2000; Burns & Spangler, 2000; Coon & Thompson, 2003; Neimeyer & Feixas, 1990) and anxiety (Edelman & Chambless, 1993; LeBeau, Davies, Culver, & Craske, 2013; Leung & Heimberg, 1996; Westra et al., 2007). Although the current study did not conduct a fine-tuned assessment of homework compliance, results and prior research suggest a dose-response relationship between symptom improvement and homework compliance across treatment (Edelman & Chambless, 1993) and that relations are not bidirectional but rather that homework compliance predicts treatment outcome (Burns & Spangler, 2000). Practice may not only enhance self-efficacy, which in turn might affect outcomes, but homework practice may also lead to learning and development of a behavioral repertoire of what to do in certain situations to cope.

Research that continues to conduct detailed assessment and analyses could expose potential ceiling effects of homework compliance on outcome, such that after a certain amount of

homework, change in outcome is no longer evident. It is unknown how frequent and how long homework practice should be conducted for each type of treatment strategy for optimal results. It may be that too much homework practice leads to confusion or a reduction in treatment self-efficacy, because when there is continued practice without continued improvement, patients may no longer associate behavior change with treatment improvement, resulting in a subsequent plateau or decrement in symptom improvement.

Emotional Avoidance. Despite predictions that emotional avoidance would be a component-specific mediator, visual inspection demonstrated clear relations between emotional avoidance and symptom change across components and groups, suggesting that emotional avoidance may be a non-specific mediator of change. Emotional avoidance, also known as experiential avoidance, is the avoidance of internal – mental, emotional, and physical – experiences (Hayes, Strosahl, & Wilson, 1999). Prior research has demonstrated clear relations between emotional avoidance and symptom severity and symptom development (Compare, Zarbo, Shonin, Van Gordon, & Marconi, 2014; Naifeh, Tull, & Gratz, 2012; Tull & Gratz, 2008). Strong relations also have been demonstrated between mindfulness-based interventions (e.g., mindfulness-based stress reduction, acceptance and commitment therapy [ACT]) and treatment outcomes (Berking, Neacsiu, Comtois, & Linehan, 2009; Forman et al., 2007; Weinrib, 2011; Zettle, 2003).

The few studies that have examined the relationship between emotional avoidance and outcomes of ACT and CBT have found stronger relations with ACT than CBT or non-significant relations between CBT and emotional avoidance (Arch, Eifert, et al., 2012; Forman et al., 2007; Lappalainen et al., 2007; Wolitzky-Taylor, Arch, Rosenfield, & Craske, 2012). Although formal mediation was not established through assessment of temporal precedence (Kazdin, 2007) in the

following studies, two studies found that emotional avoidance statistically mediated the relation between treatment and outcome (Bond & Bunce, 2000; Forman et al., 2007), bolstering the theory that this factor has a mediating role in treatment change across cognitive and behavioral components of CBT.

Theoretical explanations for the potential mediating effect of emotional avoidance on the relation between behavior therapy and symptom improvement is apparent. Exposure and pleasant events scheduling (i.e., strategies of behavioral therapy) require the patient to deliberately approach external experiences, which often are avoided due to anticipation of unpleasant feelings (e.g., depression, anxiety, stress). Thus, repeated engagement in these activities likely will lead to reductions in emotional avoidance (Bouton, 2004).

The potential mediating role of emotional avoidance in the relation between cognitive therapy and outcomes is surprising. No strategies in the cognitive therapy component directly targeted changing acceptance or avoidance of internal experiences; however, change in emotional avoidance during this component – regardless of group status – was evident. Emotional avoidance may influence symptom change in cognitive therapy through the same mechanism as exposure in behavioral therapy (Boyd & Levis, 1983). That is, cognitive strategies require discussion of internal experiences, which is a form of exposure to these unwanted experiences. Future work should test these possible mechanisms with more varied and detailed assessment.

Given that symptoms and emotional avoidance were both measured using self-report, shared method variance may account for some of their covariation. On the other hand, item inspection suggests that these measures are assessing different constructs such that the

covariation in report of symptoms and report of emotional avoidance are unlikely to be fully explained by method variance.

Implicit associations may explain the link between emotional avoidance and change of symptoms during cognitive therapy in that changes in behavior (i.e., approach behavior) leads to new learning which forms new implicit associations between constructs; however, in the current study, there was no change in implicit associations across treatment, suggesting that it would not be a mediating link. The absence of an implicit association mediation in the current study may be due to the specific assessment used in the current study. In order to reduce participant burden, patients completed three blocks of IAT pairs – (1) failure/success and me/not me; (2) unlovable/lovable and me/not me; and (3) helpless/able and me/not me. It may be that these specific associations did not capture change across treatment. Therefore, future studies would benefit from testing other associations or from developing an idiographic IAT word pair. An idiographic IAT would contain word pairs that uniquely capture implicit associations for that individual. For example, an individual who has social anxiety may associate "party" with "danger" and "home" with "safe", whereas an individual without concerns of judgment might not hold the same associations. Thus, if more specific implicit word pairs were used in the current study, implicit associations possibly could have explained the link between emotional avoidance and outcome in the current study.

Emotion Regulation. Although an association between emotion regulation and outcome emerged, visual inspection of temporal precedence suggested that emotion regulation was not a potential mediator. One study found that changes in self-reported emotion regulation mediated the effect of cognitive behavioral therapy, compared to treatment as usual, on frequency of deliberate self-harm at nine-month follow-up (Slee, Spinhoven, Garnefski, & Arensman, 2008).

No other studies examining formal mediation of emotion regulation on treatment outcomes were found; however, research examining the effects of emotion regulation on the development of psychopathology and on treatment outcome suggests a potential mediating role of this variable.

A clear link has been established between emotion regulation deficits and psychopathology (Aldao, Nolen-Hoeksema, & Schweizer, 2010). Research also has examined the efficacy of emotion regulation skills training as a component of cognitive and behavioral based therapies. Dialectical behavior therapy – a cognitive behavioral skills-based treatment that teaches emotion regulation skills – has been found to be effective in treating patients with borderline personality disorder, history of self-harm, post-traumatic stress disorder, substance dependence, and treatment-resistant depression (Harley, Susan, Safren, Jacobo, & Fava, 2008; Harned, Korslund, Foa, & Linehan, 2012; Linehan et al., 2006; Linehan et al., 2002). Berking et al. (2008) examined emotion regulation skills training as a supplemental strategy to a cognitive behavioral based treatment for inpatients. They found that the training led to change in emotion regulation skills from pre- to post-treatment and that the training plus a cognitive behavioral based treatment led to enhanced outcomes compared to the treatment alone. This finding was replicated again in a later study among inpatients with major depressive disorder (Berking, Ebert, Cuijpers, & Hofmann, 2013). In another study, an affect skills training for PTSD led to better remission rates for PTSD than did a supportive therapy plus exposure-based treatment (Cloitre et al., 2010). Results from a case study has suggested efficacy of emotion regulation therapy for generalized anxiety disorder (Mennin, 2004). Finally, emotion regulation skills training for children with depression has shown promising results (Kovacs et al., 2006), and researchers have pushed for the development of supplemental emotion regulation training for CBT for child anxiety (Hannesdottir & Ollendick, 2007).

Although the potential mediating role of emotion regulation in treatments for anxiety and depression is understudied, absence of a relation between emotion regulation and outcome in the current study is somewhat surprising given the aforementioned findings. It is especially surprising in light of the apparent relation between emotional avoidance and outcome, because emotional avoidance can be construed as a type of emotion regulation strategy, which is any effort taken to influence what, when, and how emotions are had (Gross, 1998). In fact, other similar forms of emotion regulation strategies – passive coping and suppression – are related to emotional avoidance and often mediate the relation between these emotion regulation strategies and symptom severity (Fledderus, Bohlmeijer, & Pieterse, 2010; Kashdan, Barrios, Forsyth, & Steger, 2006). In the current study, a self-report measure of emotion regulation was administered at pre-, mid-, and post-treatment and at follow-up. It could be that this measure was not sensitive enough to change or was not administered frequently enough. Future studies that include frequent behavioral measurement of emotion regulation may help to tease apart these relations.

Self-Efficacy and Treatment Expectancy. Both general self-efficacy and treatment expectancy demonstrated patterns of results across time that were similar to outcomes, suggesting that self-efficacy and treatment expectancy may be other potential mediators of treatment effects. Bandura (1982) defined self-efficacy as belief in one's own ability to complete an action and distinguished this from outcome expectancy, which is the belief that one's behavior or actions will lead to intended change (Bandura, 1982; Bandura, 1977). General self-efficacy was characterized in the current study as judgment of one's ability to cope with negative events (Bandura, 1977). Trends of a positive relation between general self-efficacy and symptom improvement is not surprising given that aims of both the behavioral and cognitive

components were to increase ability to cope with negative external and internal events. In behavior therapy, patients practiced coping through exposure or pleasant events scheduling, and in cognitive therapy, patients were given concrete cognitive skills to cope with such events. Bandura theorized that predictability and controllability were two factors that influenced self-efficacy and that there are two forms of controllability – behavioral and cognitive (Bandura, 1982). Behavioral controllability is the belief in one's ability to modify or control events to prevent them from occurring, and cognitive controllability is the belief in one's ability to manage negative events if they surface (Bandura, 1982). Both of these forms of controllability nicely map on to behavioral and cognitive components and potentially explain their relations. Additional assessment of change in controllability across time would further delineate what factors may influence outcome.

Brown et al. (2014) defined treatment self-efficacy as one's belief that s/he will engage in and complete therapy, and outcome expectancy as belief that one will improve from therapy. Self-efficacy and outcome expectancy have been found to be related (e.g., Brown et al., 2014) but independent constructs (Maddux, Sherer, & Rogers, 1982). Like self-efficacy, outcome expectancy predicts outcomes in evidence-based psychotherapies (Borkovec & Costello, 1993; Chambless, Tran, & Glass, 1997; Pricea, Andersona, Henricha, & Olasov Rothbaumb, 2008; Safren, Heimberg, & Juster, 1997) and medication-based therapies (Sotsky et al., 1991; Stewart-Williams & Podd, 2004) for anxiety and depression. Brown et al. (2014) examined bidirectional relations between both of these constructs and treatment outcome. They found that while both constructs predicted treatment outcome, outcome did not predict change in each of these constructs. While the current study cannot corroborate these results with statistics, similar to Brown et al. (2014), visual analysis suggests that self-efficacy does predict treatment outcome.

Implications from current study results are that general self-efficacy and treatment outcome expectancy are both associated with symptom improvement. Although visual analysis could not clearly establish temporal precedence, change in general self-efficacy appeared to precede change in outcome, whereas temporal precedence could not be identified with treatment expectancy. Thus, although both factors may be potential mediators of treatment outcome, general self-efficacy has more evidence in support of this hypothesis.

Stress Appraisal and Frequency of Automatic Thinking. Stress appraisal and frequency of automatic thinking were expected to explain change during cognitive therapy, because much of cognitive therapy for anxiety and depression is explicitly dedicated to the restructuring of automatic thoughts through reappraisal. Stress appraisal and frequency of maladaptive automatic thinking, as assessed in the current study, did appear to be cognitive-specific mediators; however, presence of temporal precedence was not clear from visual analysis alone, and future studies should conduct formal mediation analysis to further understand these relations. Beck (2011) hypothesized that cognitive therapy works through changing thoughts, but the exact mechanisms of this treatment have not been well studied (DeRubeis et al., 1990).

There has been much debate in the field over whether cognitive change explains symptom change during cognitive therapy. Much of the research on this is muddled, unfortunately, due to examination of mediation with cognitive therapy that often includes behavioral strategies. Nonetheless, while some studies suggest that change from cognitive therapy is not mediated by change in cognitions (Fava, Bless, Otto, Pava, & Rosenbaum, 1994; Safran et al., 1987), others have found evidence in support of cognitive change mediating or accounting for treatment change (e.g., Garratt, Ingram, Rand, & Sawalani, 2007). The current

results suggest that hypothesized mechanisms of early theorists and treatment developers were accurate and are consistent with prior research.

Similar item content across measures may explain some of the variability accounting for the associations between outcome and stress appraisal and between outcome and automatic thinking. An item inspection was conducted across measures, and it was determined that the content of items across measures was distinct, as evidenced by prior studies demonstrating their discriminant validity with symptom measures of anxiety and depression (Burgess & Haaga, 1994; Safran et al., 1987); thus, it is not likely that similar item content across measures explained associations among outcome, stress appraisal, and automatic thinking. Further, stronger relations among these factors only were found during the cognitive component. If there was similarity in item content across measures, then relations among factors should not differ by component.

Other Potential Mediating Factors. No other constructs (implicit associations, distress tolerance, approach/avoidance tendencies, awareness of cognitions) were potential mediators. Hypothesized behavioral mediators that were not corroborated to be potential mediators by visual analysis in the current study were distress tolerance and automatic approach/avoidance tendencies. Aside from the possibility that these factors do not mediate treatment change, patient burden could be another reason why effects are not present. Patients were asked to complete these measures weekly prior to session in addition to completion of self-report questionnaires. Thus, patient fatigue may have increased, and motivation may have decreased.

Another possibility is that the validity of these measures may have influenced results. For example, unlike the original MTCT, in the current study participants were not provided with rewards for their performance, which may have affected motivation to perform well on the task.

Behavioral observations indicated that client C3 made no attempt to try the task during any session. Further, the task was administered so frequently (i.e., once a week), compared to in other prior studies, that its validity with such frequent repeated measurement may be in question. Repeated measurement may have led to practice effects. Changes across treatment may not be due to the intervention itself but rather the participant's enhanced ability to respond more quickly with repeated practice. Such effects have been examined in multiple studies (A. G. Greenwald & Nosek, 2001; A. G. Greenwald, Nosek, & Banaji, 2003; Teachman & Woody, 2003).

For the AAT, there is no gold standard to measure approach/avoidance tendencies; therefore, the current study developed its own version of the measure that included features from prior AATs but formulated its own content. The current version had never been tested for its psychometric properties, which may have affected results. Also, like the MTCT, the AAT in the current study was administered weekly, which is more frequent than any known study to administer versions of this paradigm.

Awareness of cognitions was hypothesized to be a cognitive mediator, given that awareness is a key component of mindfulness and monitoring, both of which are treatment strategies that influence treatment outcomes and improvement in psychological well-being (Baer, 2009; Febbraro & Clum, 1998; Kazdin, 1974; Keng, Smoski, & Robins, 2011; Kopp, 1988; Ong, Shapiro, & Manber, 2008; Taylor, 1985). However, awareness of cognitions did not appear to be a factor that influenced outcome in the current study. Visual analysis demonstrates nearly no variability in this measure across treatment. Lack of variability across time could be due to poor construct validity resulting in participants having difficulty understanding what each item was intended to measure, due to the Likert scale not providing a wide enough array of options to allow for variability, or due to the treatment not leading to factor change. The two items were

taken from a full measure – the PHLMS, which demonstrated good psychometric properties (Cardaciotto et al., 2008); however, the individual items as used in the current study were never tested for their validity or reliability as independent measures of awareness of cognitions.

If future studies determine that psychometric properties of these items are sound, then the question becomes why there was no variability in the current study across all participants or between behavioral and cognitive treatment components. Although this treatment did not specifically target becoming aware of one's emotions and thoughts, these direct techniques were not prohibited, and many treatment skills indirectly trained participants in how to become aware of their internal experiences (e.g., exposure, pleasant events scheduling, imagining the positive). Examination of this construct across treatments comparing the current study's broadly defined cognitive behavioral therapy to mindfulness-focused treatments may help to identify if directly targeting increasing awareness is necessary for variability in this measure to emerge.

Limitations. In addition to aforementioned limitations, a few other limitations are worth noting. First, there are limits on what implications can drawn from single-case experimental designs given that their sample sizes are n of one. Even meta-analytic techniques in the current study utilized only three single-case designs per group. Results that emerged might have resulted from type I error or conversely results that did not emerge might have resulted from type II error. Thus, this type of study design functions best as pilot studies intended to inform large-scale group design studies. Note, however, that Hedges' g, which was the effect size calculated in the current study, does correct for small sample size bias. Further, the effect size calculator, developed by Shadish et al. (2014), takes into account the number of cases, number of time points within a case, ratio between case variance and total variance, and lag-one autocorrelation.

The number of measures that were administered and the frequency of measurement increased type I error rate. Future single-case experimental designs that reduce the number of measures to those that were found to explain change in the current study would diminish some of the measure overload found in the current study.

Effect size calculations with baseline assessments only included one to fourteen time points depending on the outcome measured, whereas comparison components contained anywhere from four to fifty time points. The unequal number of time points across phases during effect size calculation may have biased results and should be interpreted with caution. Unfortunately, due to limited resources and in an effort to reduce patient burden, baselines were limited to 10-14 days. Future single-case designs should measure longer baselines to reduce potential bias in effect size calculations.

Despite effect size calculations identifying order and incremental effects, visual analysis did not corroborate these findings. While this may diminish the integrity of these results, most likely, the reason for inconsistency across types of analyses is due to the sensitivity of these analyses. It may be that effect size calculations were able to identify effects unidentifiable to the human eye.

Visual inspection in the current study could identify potential mediators but it could not identify formal mediation due to lack of statistical methods developed for this purpose. Thus, future studies should continue to examine formal mediation using group design studies and meeting criteria of Kazdin (2007).

The current study's broad definitions of treatment components may have influenced outcomes. In the current study, the author was curious about mediators and change of broadly-defined behavioral and cognitive components. That means that although therapists were given

parameters on what content they could cover in session, the skills administered could vary across patients and therapists. Therefore, some of these same factors that do not seem to relate to outcomes of broadly-defined components may emerge as potential mediators if examined with narrowly-defined components. Future studies should continue to examine mediation with both broadly-defined and narrowly-defined therapies.

Implications. These limitations notwithstanding, the implications of results of the current study provide information on characteristics of treatment development often overlooked by large randomized clinical trials. This study explored how to arrange commonly implemented strategies that build cognitive behavioral therapy – the most supported evidence-based treatment for anxiety and depression. Results suggest that behavior therapy strategies likely should precede cognitive strategies for decreasing negative outcomes, whereas cognitive therapy strategies seemingly should precede behavioral strategies for increasing positive outcomes. Future single-case experimental designs should further delineate order of strategies within a component (e.g., should decatastrophizing precede overestimation?) and the minimum number of sessions needed for optimal improvement. Larger scale studies can then examine whether there are factors that moderate these order effects including type of treatment, type of symptom profile or level of severity, baseline history and demographic characteristics of the patient, and experience of therapist.

Other implications of this study are identification of potential mediators – factors that may explain change in cognitive behavioral therapy for anxiety and depression. Homework compliance, emotional avoidance, general self-efficacy, and treatment expectancy were identified as factors that potentially explained change across treatment regardless of which treatment component was administered. Also, stress appraisal and frequency of maladaptive

automatic thinking appeared to explain change during cognitive therapy. Future group-design studies that can implement formal tests of mediation should examine these factors as potential mechanisms. Even more exciting would be the development of formal mediation analyses for single-case designs.

Summary. The current study examined change in outcomes of six patients receiving cognitive behavioral therapy through detailed analysis, the results of which were presence of order and incremental effects of both cognitive therapy and behavior therapy with the optimal order being behavioral therapy preceding cognitive therapy and identification of potential treatment mediators and component-specific mediation. All of these results emerged from a small-scale study with only three therapists, six participants, and eleven months of data collection. The utility of single-case experimental designs in informing large-scale group design studies can greatly advance the field.

Table 1. Baseline demographic and clinical characteristics of participants

Characteristic	Total (n=6)	Behavioral First (n=3)	Cognitive First (n=3)
Age in years, *M (SD)*	29.50 (12.05)	24.33 (8.39)	34.67 (14.57)
Gender			
Female	n=4	n=2	n=2
Ethnicity/Race			
White/Caucasian	n=2	n=1	n=1
Black/African-American	--	--	--
Hispanic/Latino	n=1	n=1	--
Asian/Pacific Islander	n=3	n=1	n=2
Native American	--	--	--
Other	--	--	--
Marital status			
Single	n=5	n=3	n=2
Partner	--	--	--
Married	n=1	--	n=1
Divorced	--	--	--
Separated	--	--	--
Widowed	--	--	--
Education in years, *M (SD)*	15.17 (3.19)	13.00 (3.00)	17.33 (1.53)

Currently employed	n=3	n=2	n=1
Income			
<25,000	n=5	n=3	n=2
25-50,000	--	--	--
51-75,0000	n=1	--	n=1
76-100,000	--	--	--
>100,000	--	--	--
On medication	n=2	n=2	--
Diagnoses			
Major depressive disorder	n=3	n=2	n=1
Panic disorder	n=5	n=2	n=3
Agoraphobia	n=2	n=1	n=1
Social anxiety disorder	n=3	n=1	n=2
Obsessive compulsive disorder	n=2	--	n=2
Post-traumatic stress disorder	n=1	--	n=1
Generalized anxiety disorder	n=3	n=1	n=2

Table 2. Participant measures

Measure	Administration frequency	Number of Items	Administration duration
Total for daily measures:		7	3 min
Daily negative affect levels	daily	4	~2 min
Awareness of cognitive patterns	daily	2	~1 min
Treatment expectancy	daily	1	<1min
Total for 1st session weekly measures:		36	29 min
(beginning of sessions 1, 3, 5, 7, 10, 13, 15, 17)			
DASS-21	weekly (1st session)	21	~7 min
SDS	weekly (1st session)	3	~1 min
GSES	weekly (1st session)	10	~4 min
IAT	weekly (1st session)	--	~15 min
Changes in therapy/medication	weekly (1st session)	2	~1 min
Total for 2nd session weekly measures:		58	35 min
(beginning of sessions 1, 3, 5, 7, 10, 13, 15, 17)			
PANAS	weekly (2nd session)	20	~5 min
AAQ-II	weekly (2nd session)	10	~5 min
SAM	weekly (2nd session)	28	~10 min
AAT	weekly (2nd session)	--	~10 min
MTCT	weekly (2nd session)	--	~10 min

Total for pre-, mid-, post-, follow-up measures:		203	127 min
(baseline assessment, beginning of session 10, end of session 17, 1-month following post)			
ATQ/ASSQ	Pre, Mid, Post, FU	62	~25 min
DERS	Pre, Mid, Post, FU	36	~12 min
Demographic Questionnaire	Pre, Mid, Post, FU	11	~4 min
	+ weekly measures total	109	56 min

Note: Pre=pre-treatment, mid=mid-treatment, post=post-treatment, FU=follow-up, DASS-21=Depression Anxiety and Stress Scale, SDS=Sheehan Disability Scale, GSES=General Self-Efficacy Scale, IAT=Implicit Association Test, PANAS=Positive Affect Negative Affect, AAQ-II=Acceptance and Action Questionnaire, SAM=Stress Appraisal Measure, AAT=Approach/Avoidance Task, ATQ/ASSQ=Automatic Thoughts Questionnaire / Anxious Self-Statements Questionnaire, DERS=Difficulties in Emotion Regulation Questionnaire, MTPT-C=The Computerized Mirror Tracing Persistence Task

Table 3. Slopes and variances of daily outcome data within each group and phase

Outcome	Patient ID	First Component	Baseline Slope (R^2)	Behavioral Slope (R^2)	Cognitive Slope (R^2)
What was your	B3	Behavioral	0.201 (0.115)	-0.089 (0.193)	0.031 (0.013)
average level	B1	Behavioral	0.146 (0.304)	0.067 (0.258)	-0.105 (0.245)
of ANXIETY	B2	Behavioral	-0.045 (0.066)	0.007 (0.017)	0.030 (0.167)
or fear in past		Average Slope	0.101	-0.005	-0.015
24 hours? (0-	C2	Cognitive	0.005 (0.001)	-0.005 (0.001)	-0.028 (0.047)
10)	C1	Cognitive	0.351 (0.385)	-0.029 (0.074)	-0.039 (0.215)
	C3	Cognitive	0.249 (.156)	0.095 (0.351)	0.057 (0.104)
		Average Slope	0.202	0.020	-0.003
What was your	B3	Behavioral	0.183 (0.088)	-0.112 (0.240)	-0.046 (0.110)
average level	B1	Behavioral	0.145 (0.150)	0.049 (0.110)	0.089 (0.253)
of	B2	Behavioral	0.090 (0.116)	0.001 (0.000)	-0.027 (0.039)
DEPRESSION		Average Slope	0.139	-0.021	0.005
or sadness in	C2	Cognitive	-0.032 (0.030)	0.010 (0.005)	-0.030 (0.054)
past 24 hours?	C1	Cognitive	0.309 (0.143)	-0.058 (0.254)	0.032 (0.089)
(0-10)	C3	Cognitive	0.448 (0.406)	-0.075 (0.195)	-0.065 (0.135)
		Average Slope	0.242	-0.041	-0.011
What was your	B3	Behavioral	0.164 (0.079)	-0.007 (0.001)	-0.131 (0.185)
average level	B1	Behavioral	0.014 (0.001)	-0.007 (0.007)	0.052 (0.096)
of ANGER,	B2	Behavioral	-0.036 (0.033)	0.002 (0.001)	-0.034 (0.109)

GUILT, or		Average Slope	0.047	-0.004	-0.034
SHAME in	C2	Cognitive	-0.042 (0.056)	-0.014 (0.012)	0.017 (0.021)
past 24 hours?	C1	Cognitive	0.199 (0.151)	-0.058 (0.254)	0.015 (0.027)
(0-10)	C3	Cognitive	0.155 (0.250)	0.004 (0.000)	-0.034 (0.044)
		Average Slope	0.104	-0.023	-0.002
What was your	B3	Behavioral	0.282 (0.465)	-0.141 (0.356)	0.000 (0.000)
average level	B1	Behavioral	0.041 (0.023)	0.008 (0.003)	0.048 (0.081)
of DISTRESS	B2	Behavioral	-0.033 (0.026)	-0.010 (0.028)	0.057 (0.185)
in past 24		Average Slope	0.097	-0.048	0.035
hours?	C2	Cognitive	-0.032 (0.023)	-0.003 (0.000)	0.032 (0.051)
(0-10)	C1	Cognitive	0.199 (0.151)	-0.058 (0.254)	0.015 (0.027)
	C3	Cognitive	0.166 (0.049)	-0.110 (0.289)	-0.040 (0.032)
		Average Slope	0.111	-0.057	0.002
What was your	B3	Behavioral	0.164 (0.321)	0.022 (0.067)	0.014 (0.004)
average level	B1	Behavioral	0.023 (0.004)	-0.073 (0.156)	0.090 (0.445)
of happiness,	B2	Behavioral	-0.038 (0.038)	-0.028 (0.227)	0.025 (0.084)
love, curiosity,		Average Slope	0.050	-0.026	0.043
interest, and	C2	Cognitive	-0.007 (0.001)	-0.003 (0.007)	0.027 (0.032)
gratitude in	C1	Cognitive	-0.169 (0.160)	0.006 (0.004)	0.010 (0.020)
past 24 hours?	C3	Cognitive	-0.031 (0.020)	-0.017 (0.076)	-0.006 (0.005)
(0-10)		Average Slope	-0.064	-0.005	0.010

Table 4. Order effect sizes within each group

	Behavioral First		Cognitive First	
	G	VarG	G	VarG
DASS-T	0.967	0.351	0.449	0.198
PANAS N	7.006	2.615	2.516	0.931
PANAS P	-0.007	0.147	0.657	0.363
SDS-T	0.661	0.284	0.173	0.119
Daily anxiety	0.281	0.051	0.252	0.090
Daily depression	0.117	0.049	0.153	0.036
Daily anger, guilt, shame	-0.211	0.051	0.127	0.037
Daily positive emotions	0.113	0.025	0.076	0.018
Aggregate	0.141	0.049	0.165	0.085

Note: G = Hedges' g, VarG = Variance of G, DASS-T = Depression Anxiety and Stress Scale total score, PANAS N = Positive Affect Negative Affect negative subscale, PANAS P = Positive Affect Negative Affect positive subscale, SDS-T = Sheehan Disability Scale total score

Table 5. Incremental effect sizes within each group

	Incremental effect of BT compared to baseline		Incremental effect of CT compared to baseline		Incremental effect of CT compared to BT		Incremental effect of BT compared to CT	
	G	VarG	G	VarG	G	VarG	G	VarG
DASS-T	0.595	0.280	0.255	0.147	1.091	0.364	0.377	0.181
PANAS N	7.234	4.190	1.790	0.870	0.774	0.277	0.423	0.168
PANAS P	0.011	0.223	0.619	0.448	-0.022	0.057	0.390	0.186
SDS-T	0.274	0.154	0.022	0.091	0.971	0.412	0.264	0.180
Daily anxiety	0.174	0.043	0.068	0.069	0.364	0.080	0.259	0.057
Daily depression	-0.061	0.059	0.029	0.022	0.276	0.060	0.407	0.049
Daily anger, guilt, shame	-0.343	0.055	0.019	0.028	0.231	0.041	0.216	0.069
Daily positive emotions	0.201	0.042	0.025	0.013	0.109	0.022	-0.061	0.028
Aggregate	0.075	0.102	0.053	0.072	0.219	0.089	0.199	0.095

Note: BT=behavior therapy component, CT=cognitive therapy component, G=Hedges' g, VarG=Variance of G, DASS-T=Depression Anxiety and Stress Scale total score, PANAS N=Positive Affect Negative Affect negative subscale, PANAS P=Positive Affect Negative Affect positive subscale, SDS-T=Sheehan Disability Scale total score

Table 6. Fixed effects model examining overall effect of treatment compared to baseline with frequency of data collection as a predictor

	Estimate	SE	p	CI
Intercept	0.689	0.175	0.000	0.346 - 1.032
Frequency of collection	-0.558	0.184	0.002	-0.919 - -0.196

Table 7. Fixed effects model examining overall effect of treatment compared to baseline with group status and frequency of data collection as predictors

	Estimate	SE	p	CI
Intercept	0.505	0.194	0.009	0.125-0.084
Group status	0.022	0.127	0.862	-0.226-0.270
Frequency of collection	-0.413	0.192	0.032	-0.790--0.036

Table 8. Fixed effects model examining incremental effects with group status, frequency of data collection, and timing of incremental effect as predictors

	Estimate	SE	p	CI
Intercept	0.253	0.133	0.056	-0.007-0.513
Group status	-0.016	0.090	0.862	-0.192-0.161
Frequency of collection	-0.208	0.119	0.082	-0.442-0.026
Timing of incremental effect	0.129	0.090	0.151	-0.047-0.305

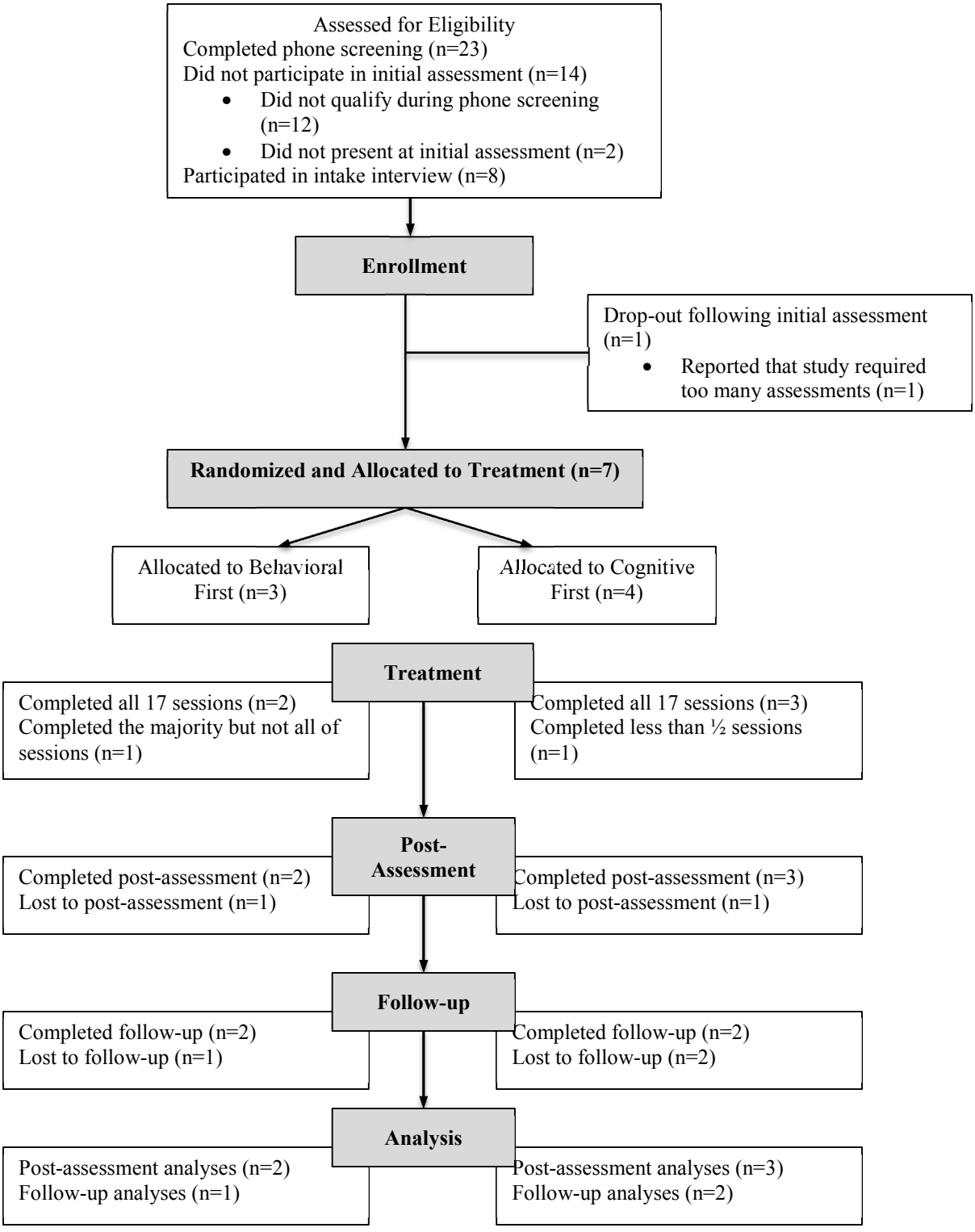


Figure 1. Patient flow through the study

Figure 2. Implicit Association Test example of a trial

Appendix A

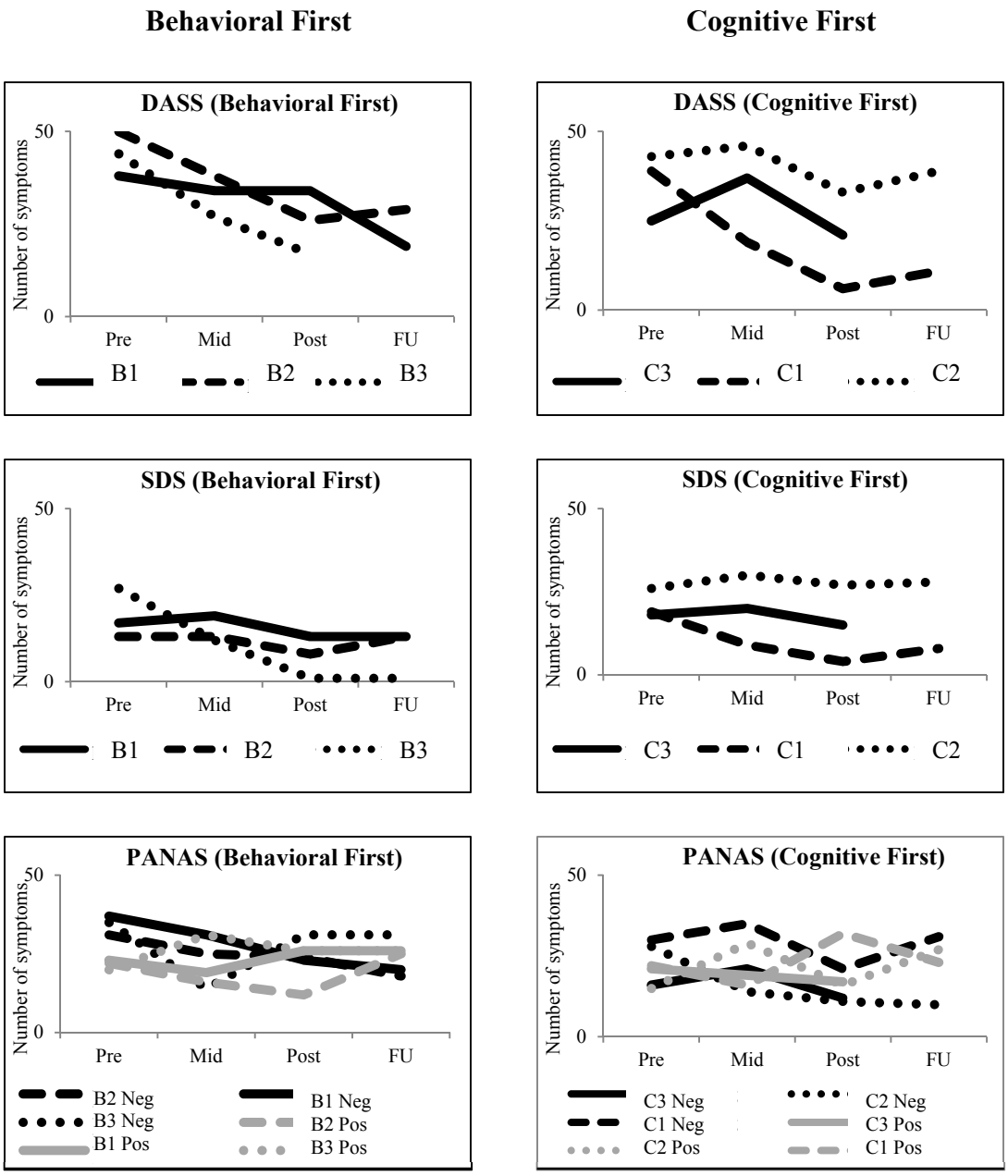


Figure A1. Graphs of outcomes at pre-, mid-, post-treatment and one-month follow-up assessment. B1, B2, B3 = Codes for patients who received behavioral first, C1, C2, C3 = Codes for patients who received cognitive first, DASS = Depression Anxiety Stress Scale Total, SDS = Sheehan Disability Scale, PANAS = Positive Affect Negative Affect Scale, Neg = Negative, Pos = Positive

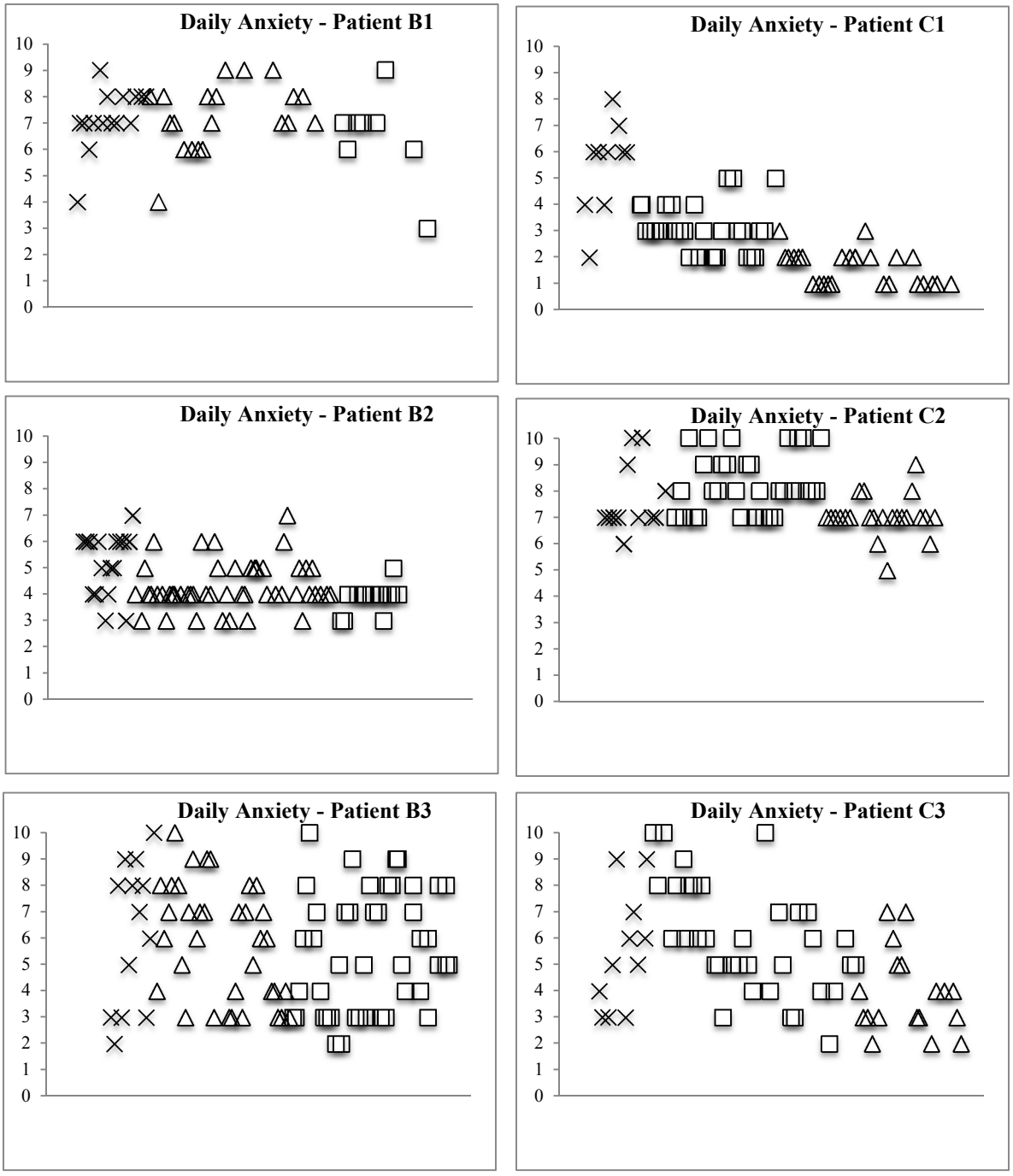


Figure A2. Graphs of daily assessment outcome of anxiety distributed by group. = baseline data points; Δ = behavioral component data points, □ = cognitive component data points

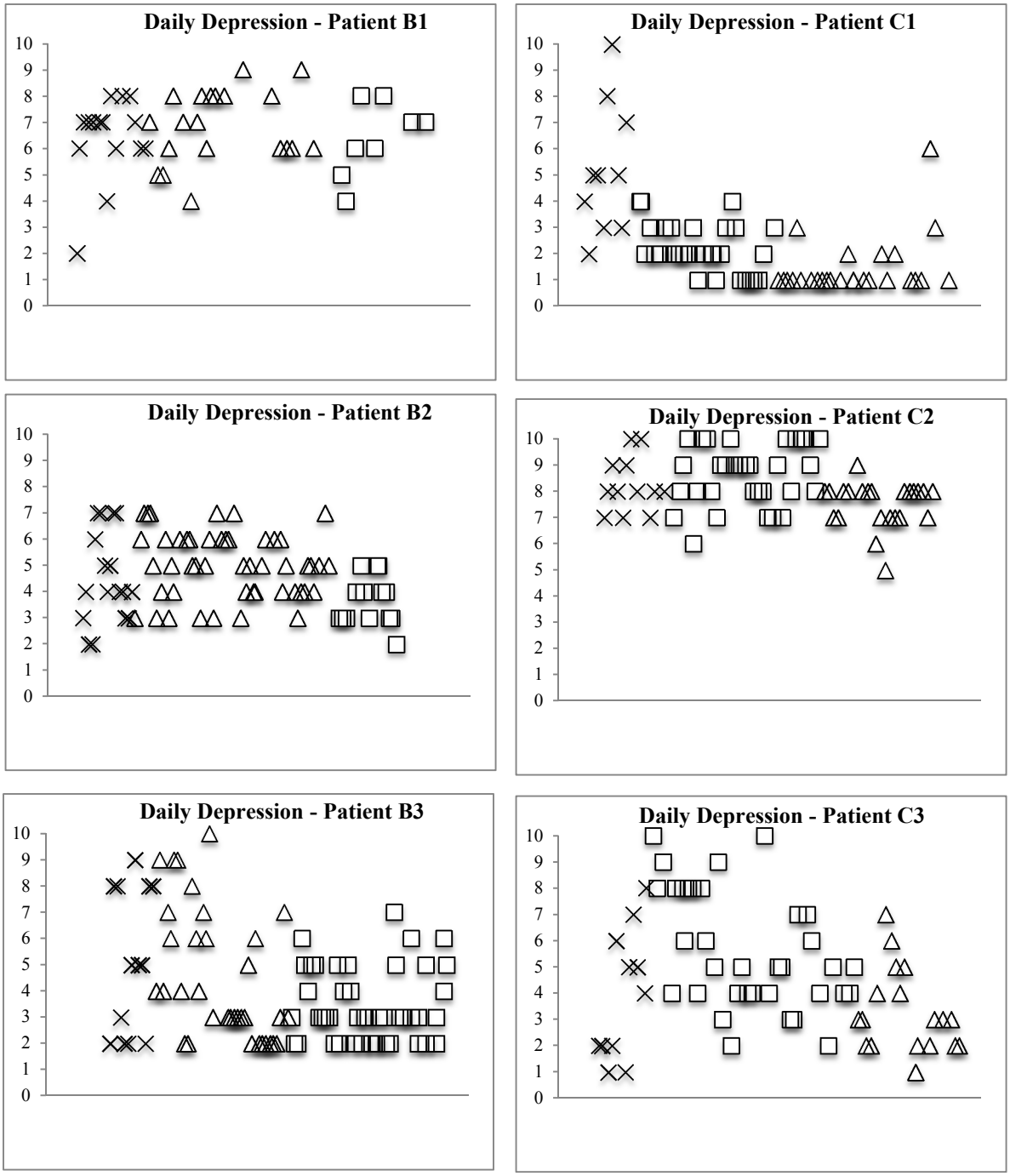


Figure A3. Graphs of daily assessment outcome of depression distributed by group. □ = baseline data points; Δ = behavioral component data points, □ = cognitive component data points

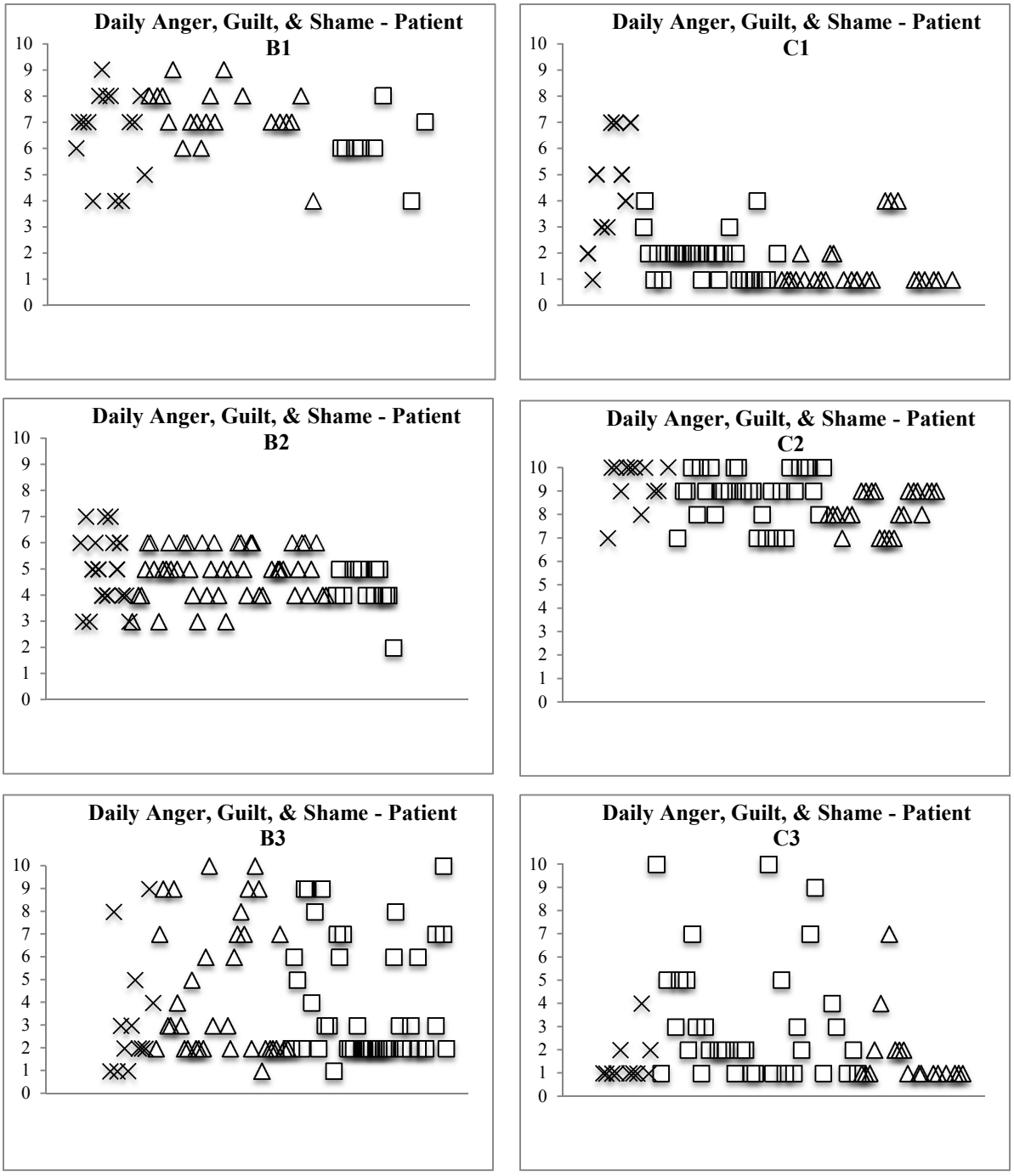


Figure A4. Graphs of daily assessment outcome of anger, guilt, and shame distributed by group. □ = baseline data points; Δ = behavioral component data points, □ = cognitive component data points

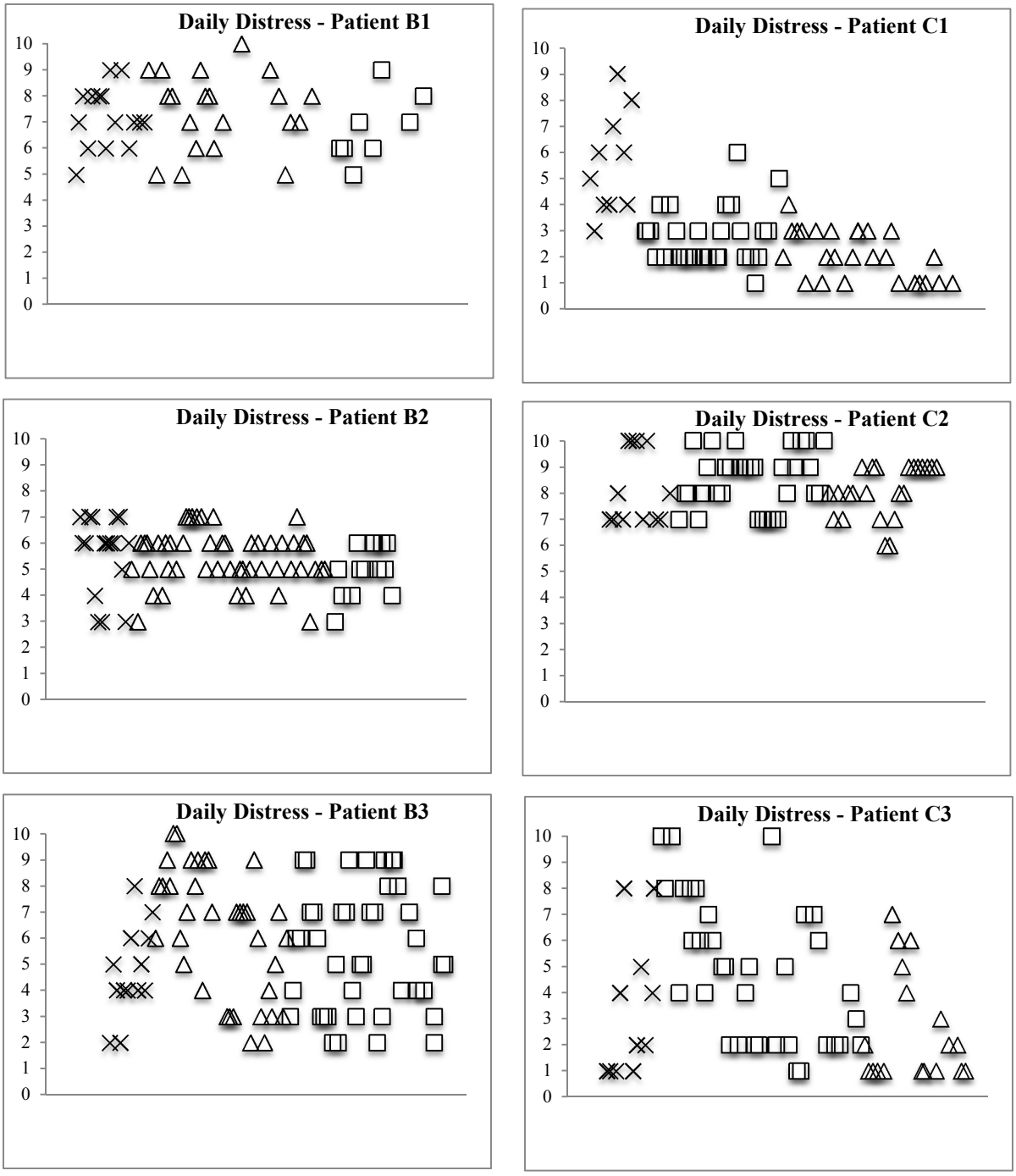


Figure A5. Graphs of daily assessment outcome of distress distributed by group. □ = baseline data points; Δ = behavioral component data points, □ = cognitive component data points

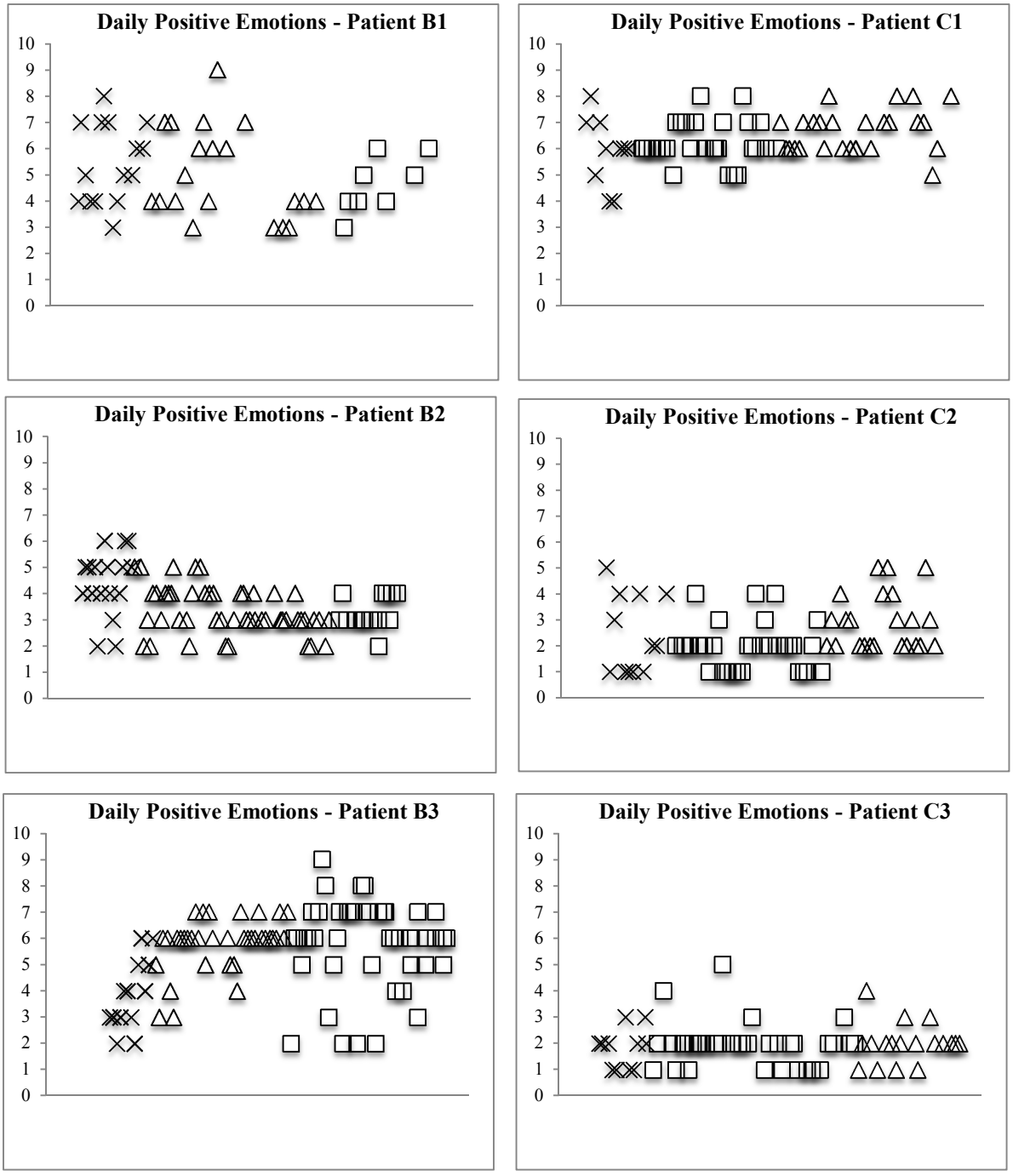


Figure A6. Graphs of daily assessment outcome of positive emotions distributed by group. = baseline data points; Δ = behavioral component data points, □ = cognitive component data points

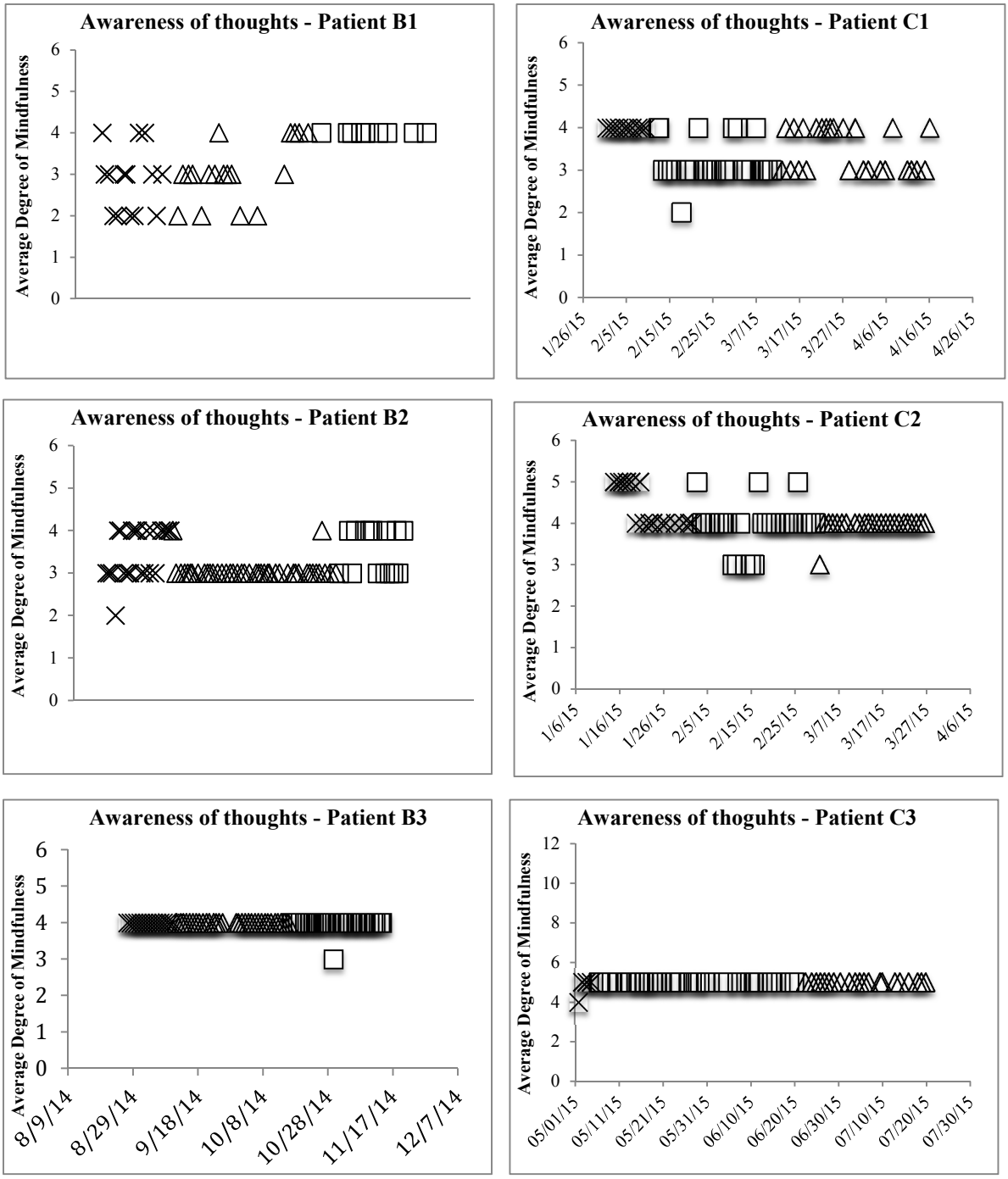


Figure A7. Graphs of daily assessment mediator of awareness of thoughts distributed by group. = baseline data points; Δ = behavioral component data points, □ = cognitive component data points

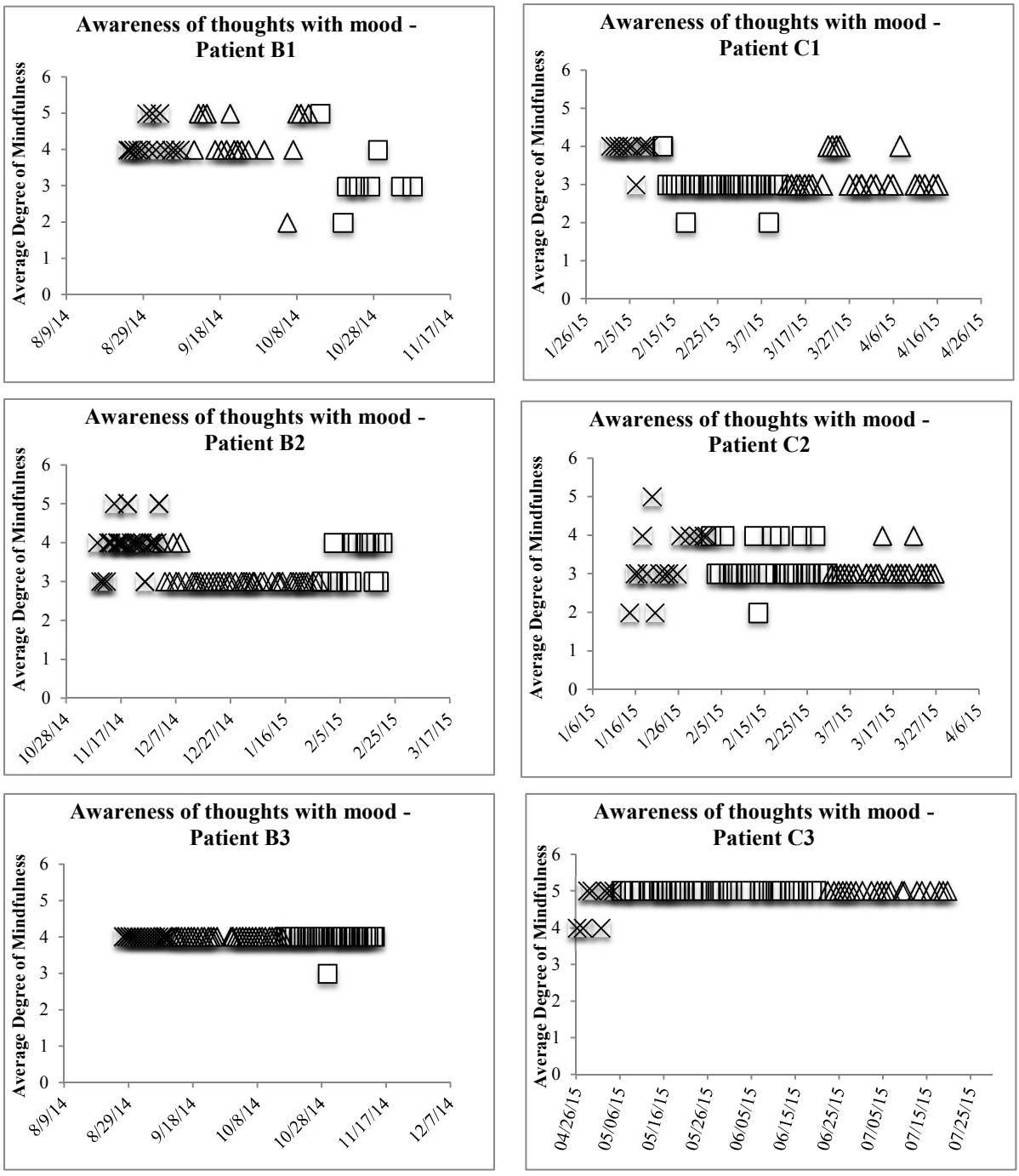


Figure A8. Graphs of daily assessment mediator of awareness of thoughts with changes in mood distributed by group. = baseline data points; Δ = behavioral component data points, □ = cognitive component data points

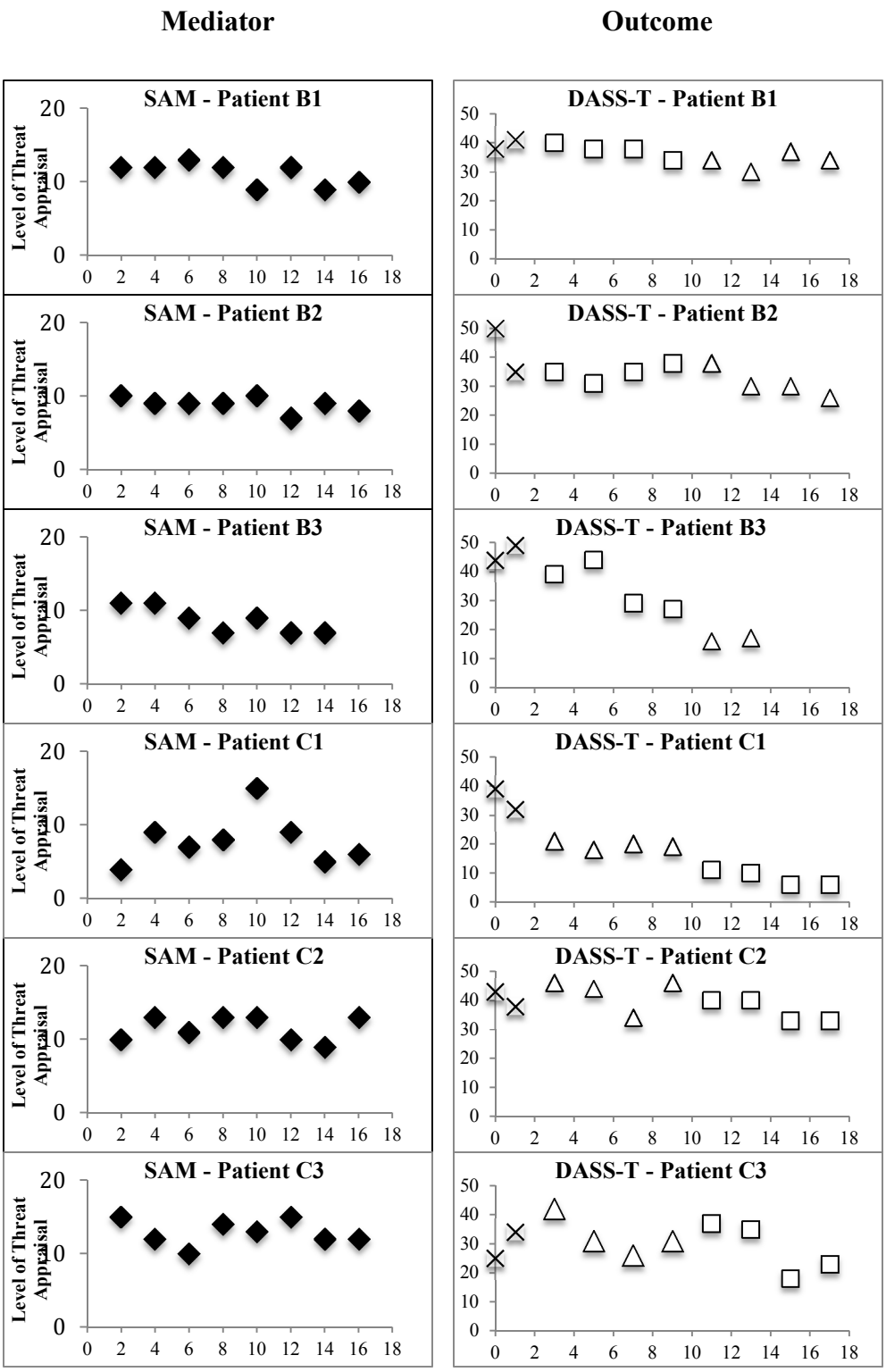


Figure A9. Graphs of session-by-session mediator of stress appraisal and outcome of depression, anxiety and stress. ◆ = mediator data point; = baseline data points; Δ = cognitive component data points, □ = behavioral component data points

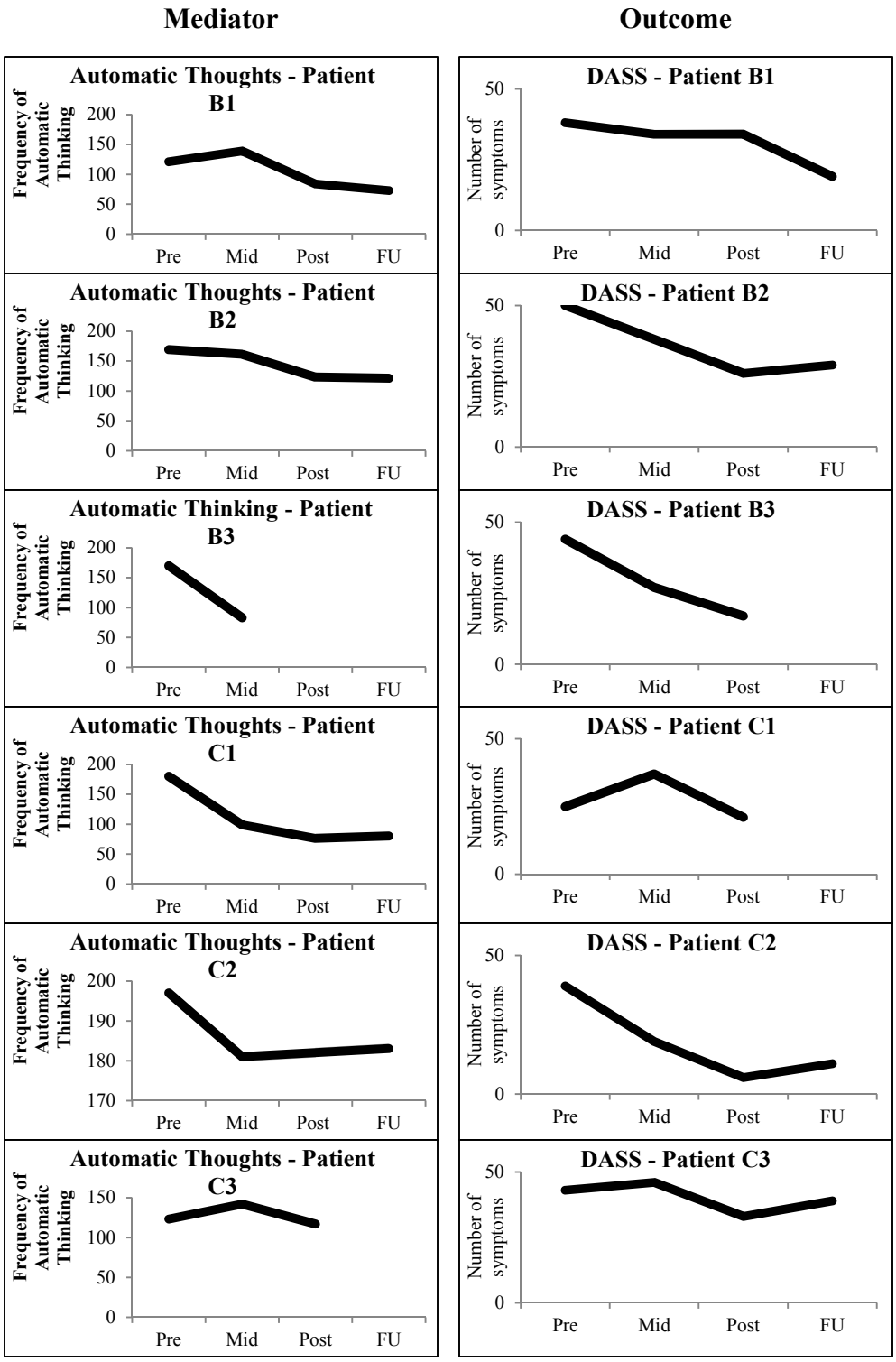


Figure A10. Graphs of session-by-session mediator of automatic thoughts and outcome of depression, anxiety and stress

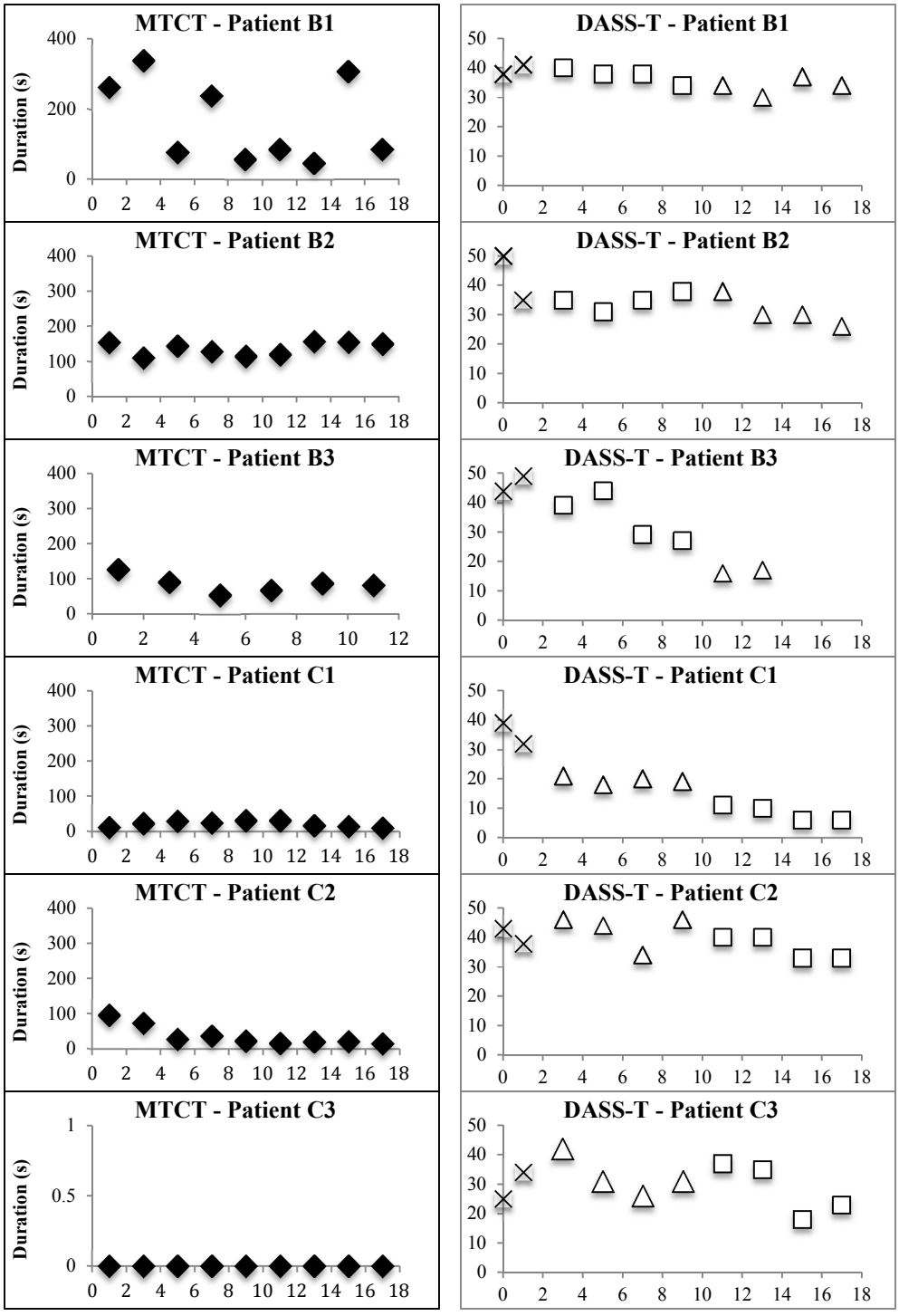


Figure A11. Graphs of session-by-session mediator of distress tolerance and outcome of depression, anxiety and stress. ◆ = mediator data point; ▯ = baseline data points; Δ = cognitive component data points, □ = behavioral component data points

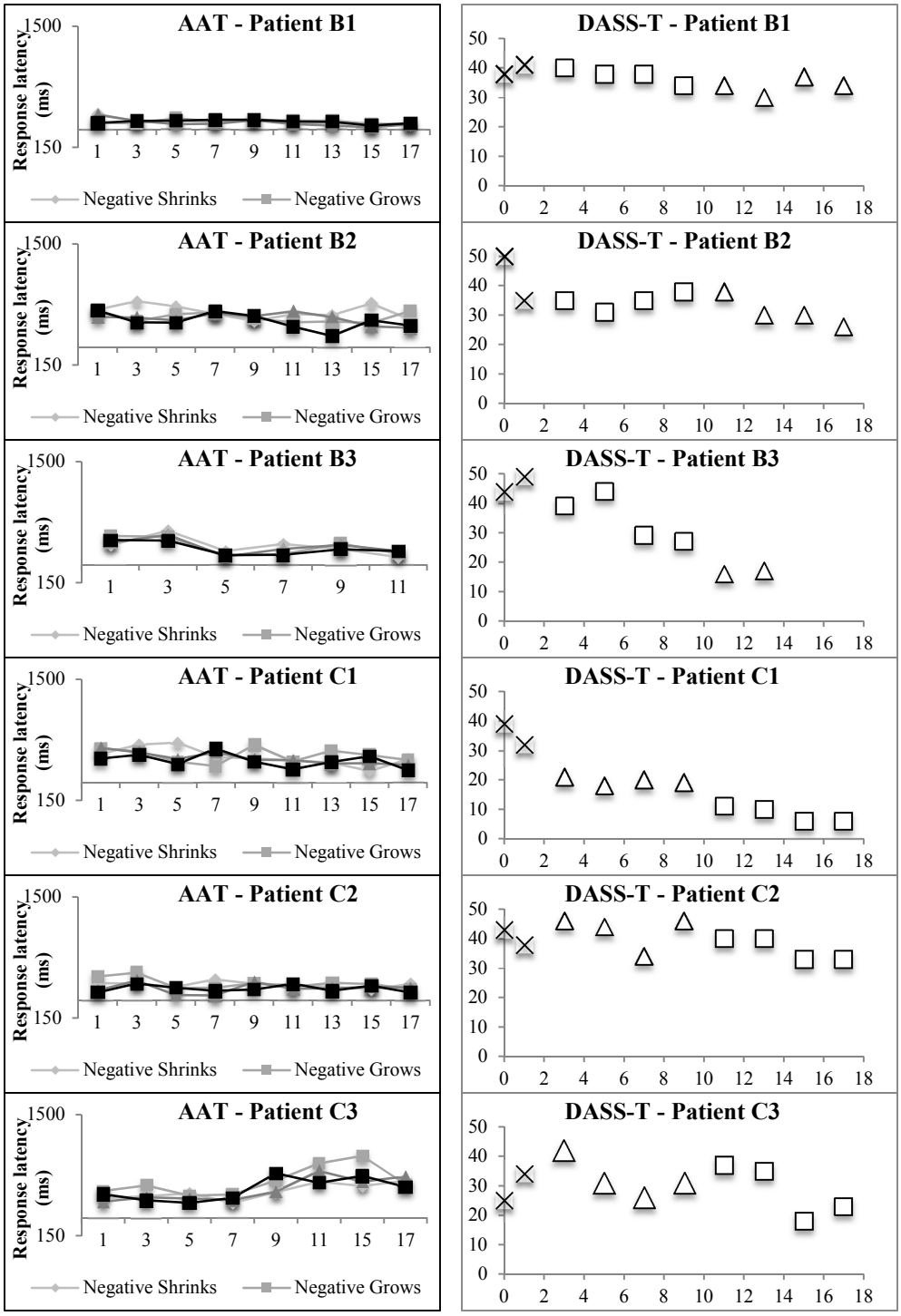


Figure A12. Graphs of session-by-session mediator of avoidance tendencies and outcome of depression, anxiety and stress. ◆= mediator data point; □ = baseline data points; Δ = cognitive component data points, □ = behavioral component data points

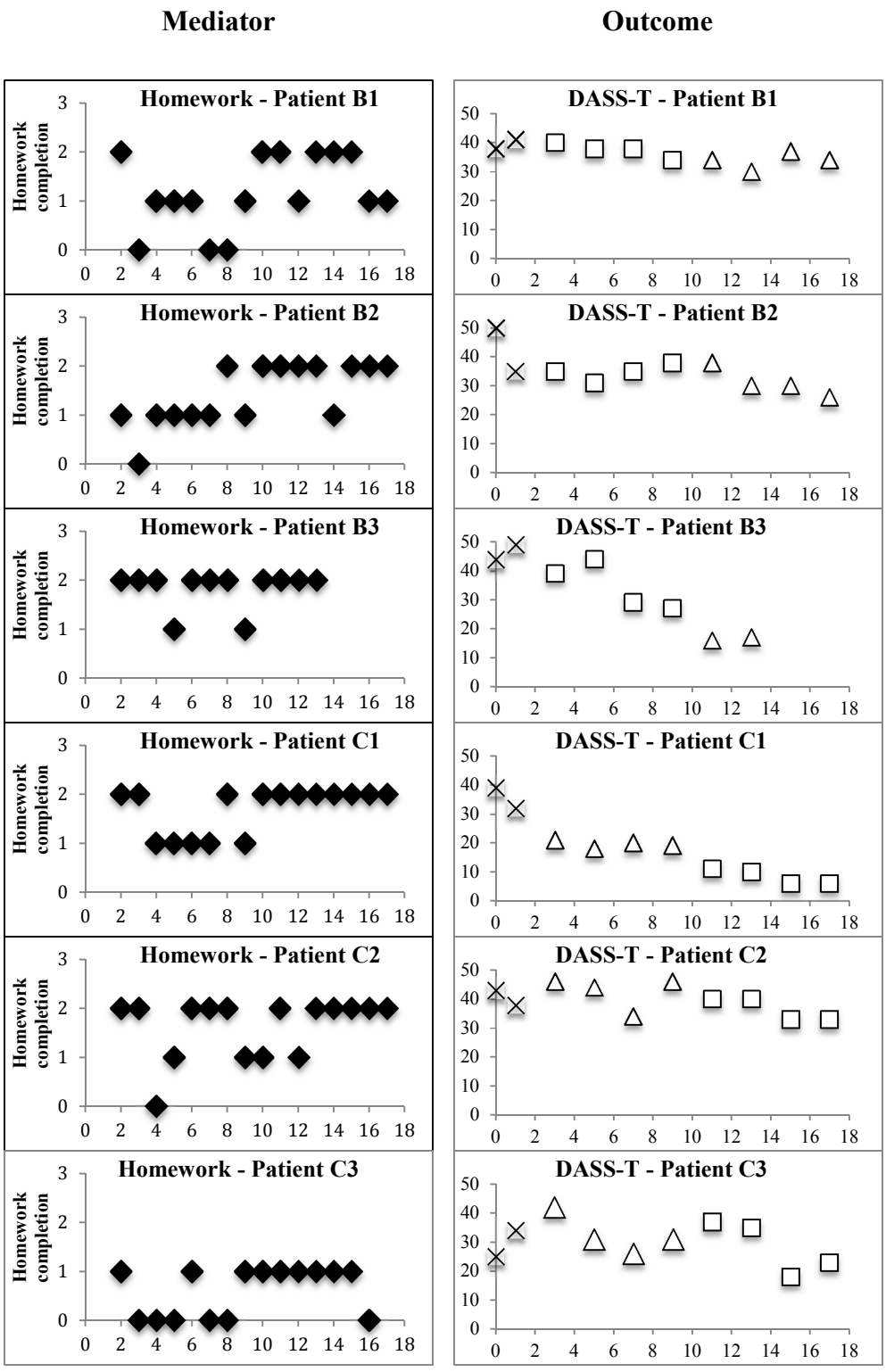


Figure A13. Graphs of session-by-session mediator of homework compliance and outcome of depression, anxiety and stress. ◆ = mediator data point; ▯ = baseline data points; Δ = cognitive component data points, □ = behavioral component data points

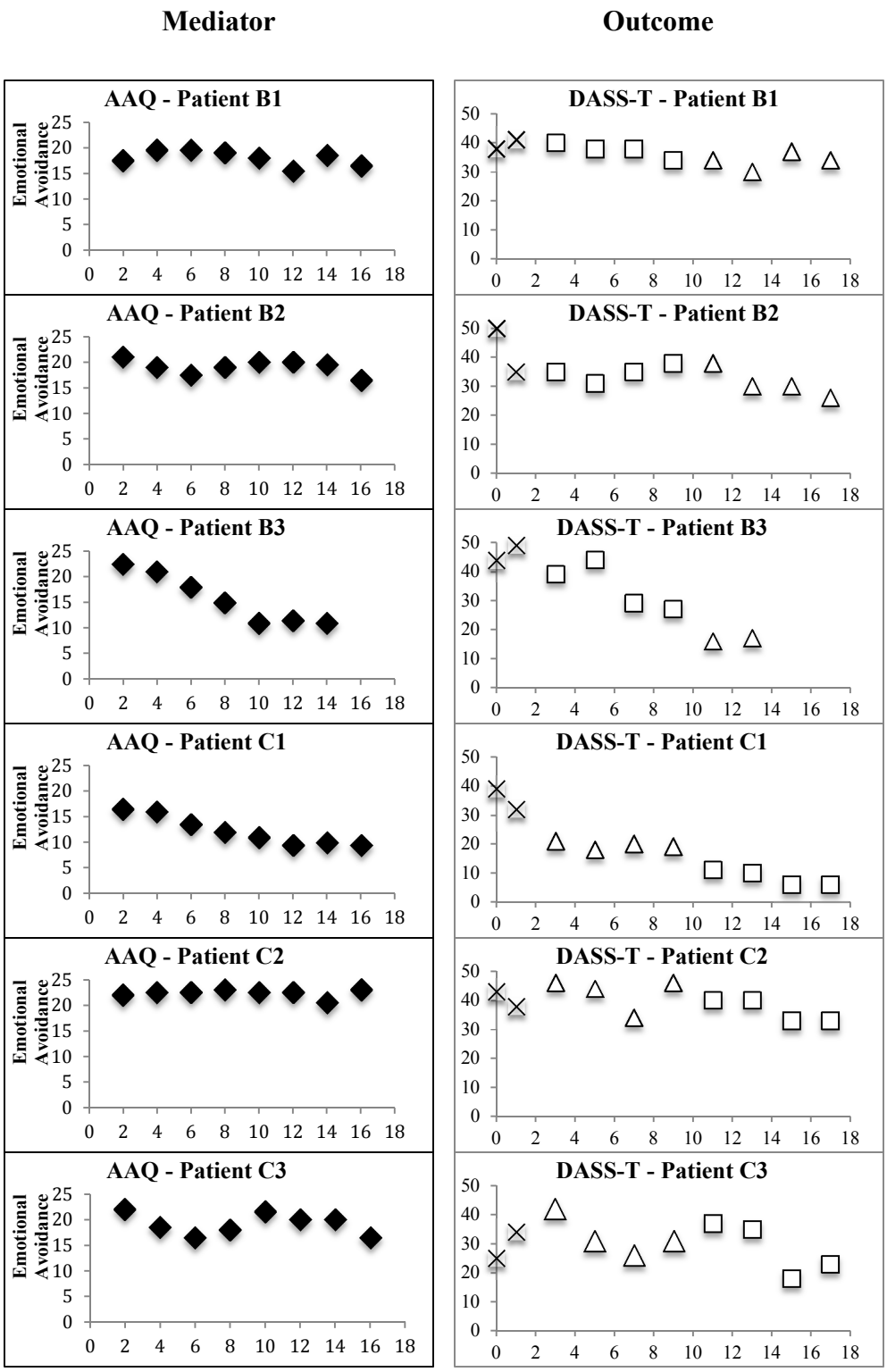


Figure A14. Graphs of session-by-session mediator of experiential avoidance and outcome of depression, anxiety and stress. ◆ = mediator data point; ▯ = baseline data points; Δ = cognitive component data points, □ = behavioral component data points

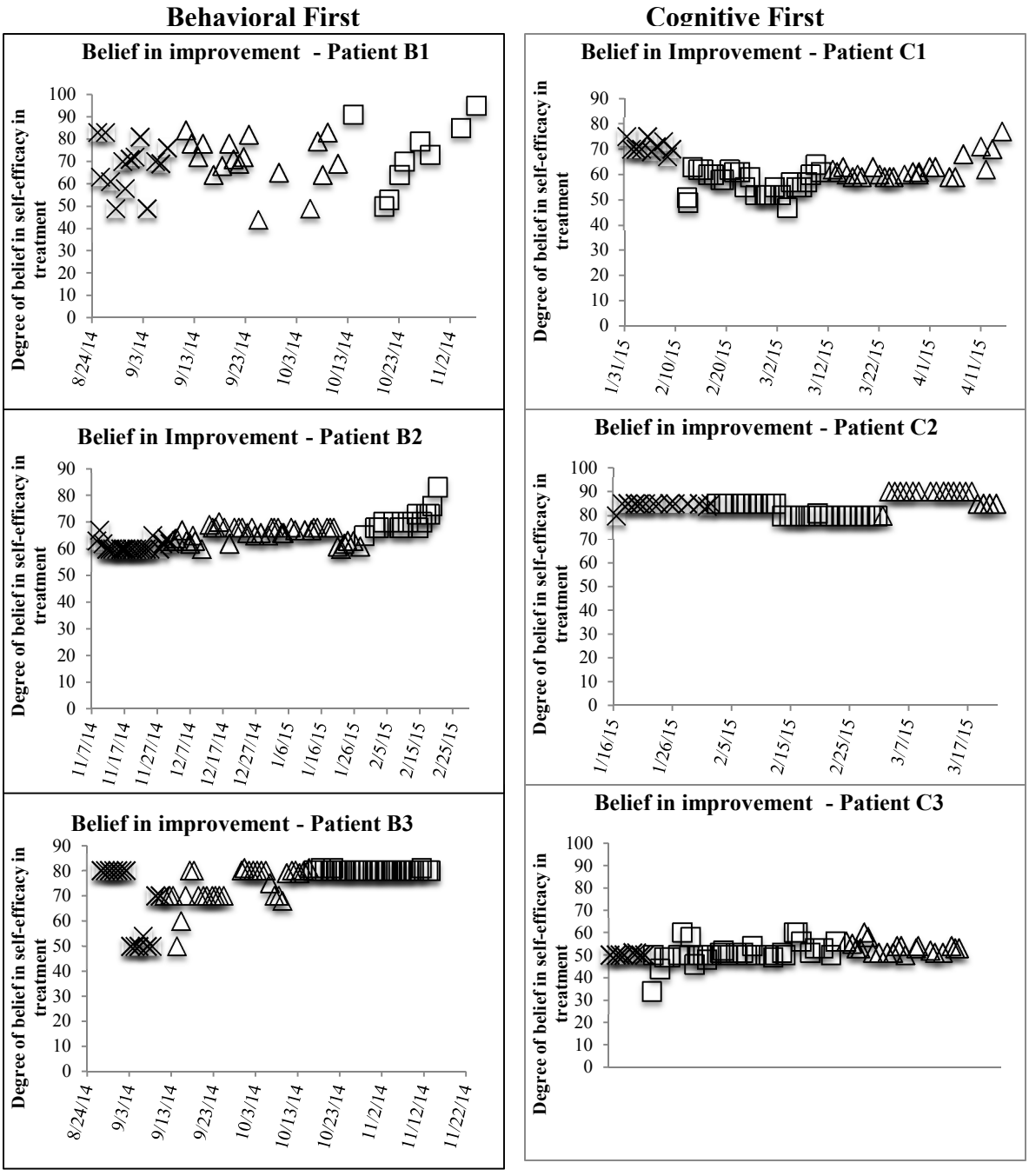


Figure A15. Graphs of daily mediator of belief in improvement in therapy and outcome of depression, anxiety and stress. = baseline data points; Δ = cognitive component data points, □ = behavioral component data points

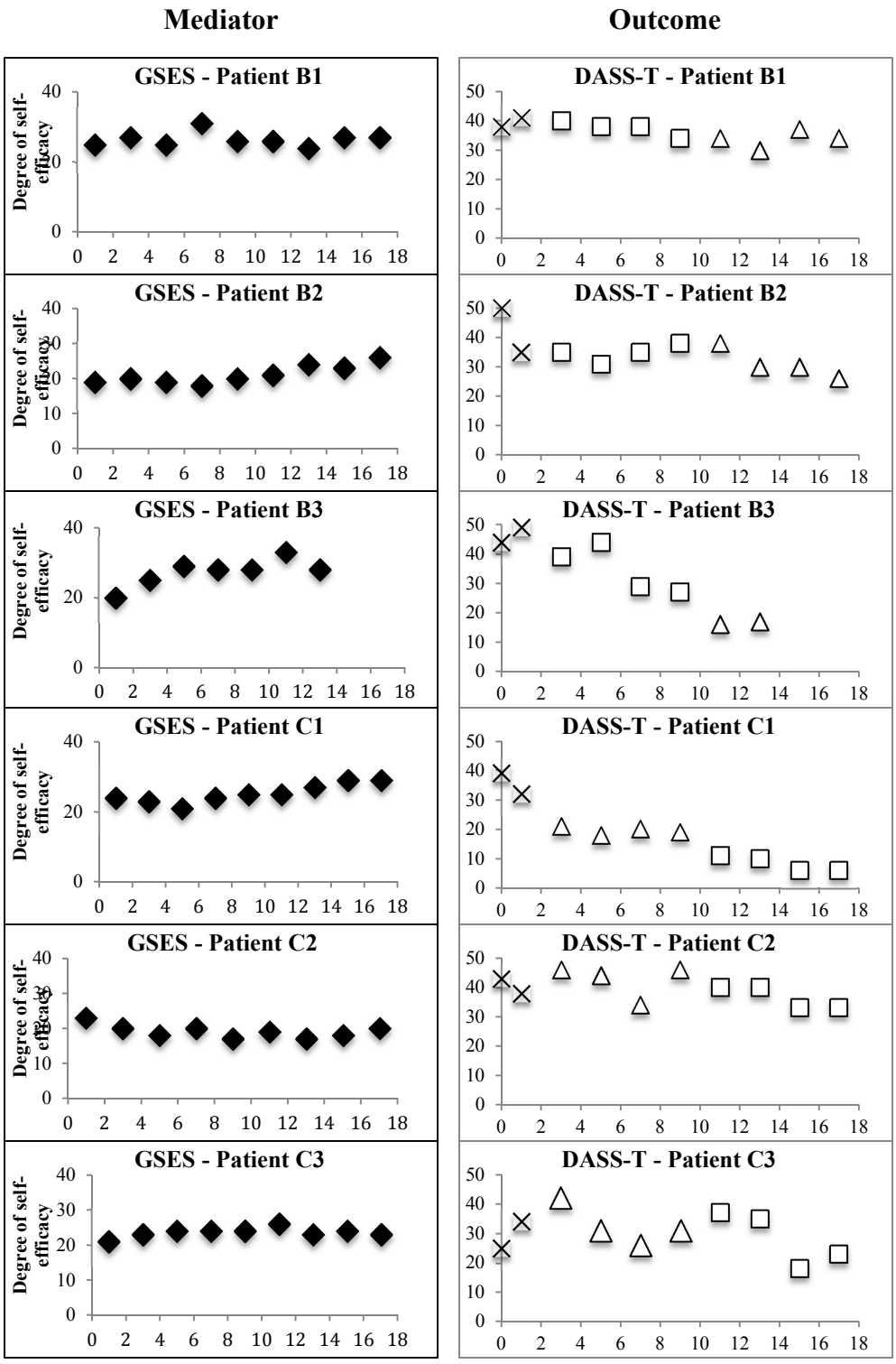


Figure A16. Graphs of session-by-session mediator of general self-efficacy and outcome of depression, anxiety and stress. ◆ = mediator data point; ▯ = baseline data points; Δ = behavioral component data points, □ = cognitive component data points

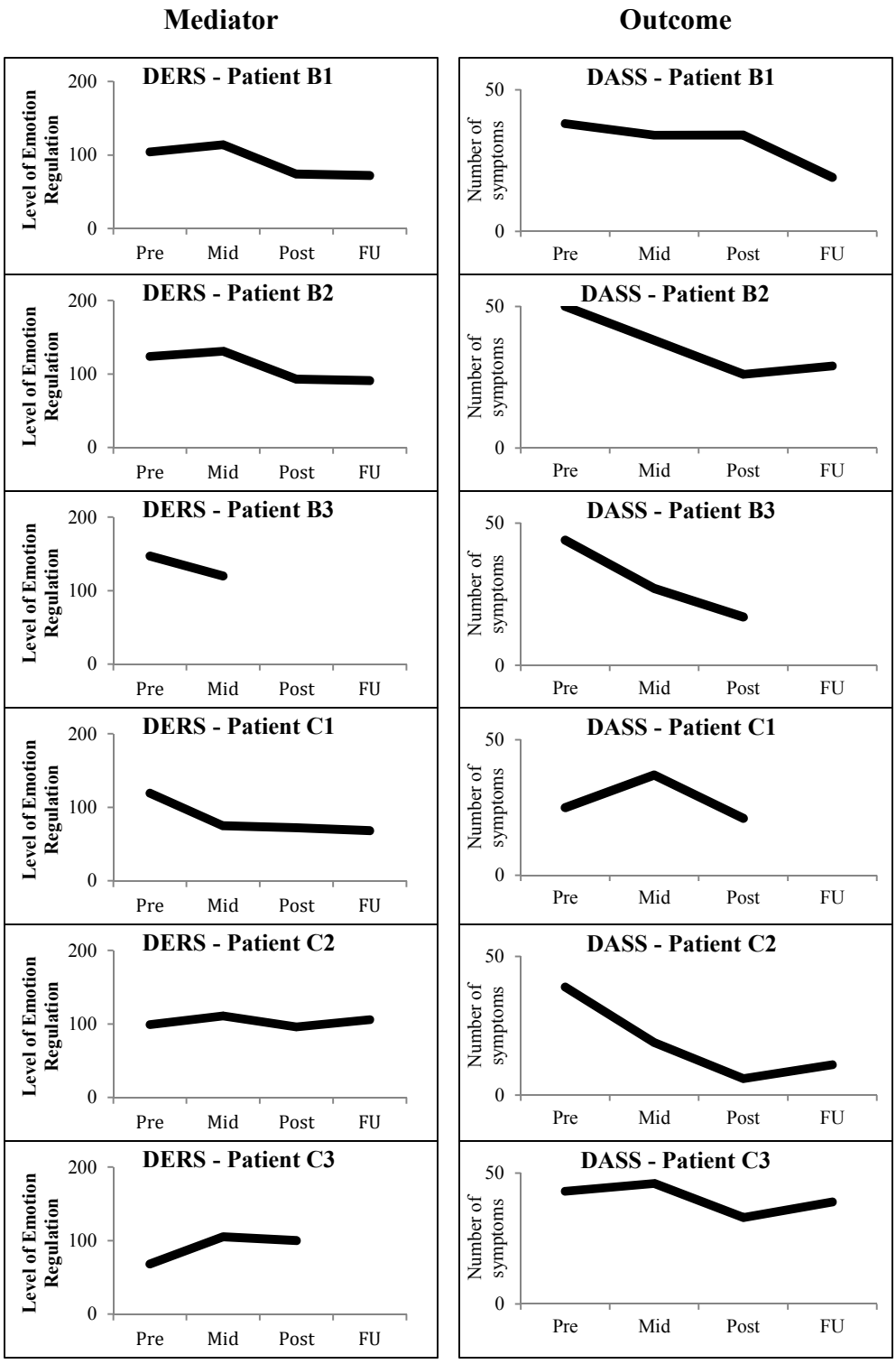


Figure A17. Graphs of mediator of distress tolerance and outcome of depression, anxiety, and stress scores at pre-treatment, mid-treatment, post-treatment, and follow-up assessment distributed by group

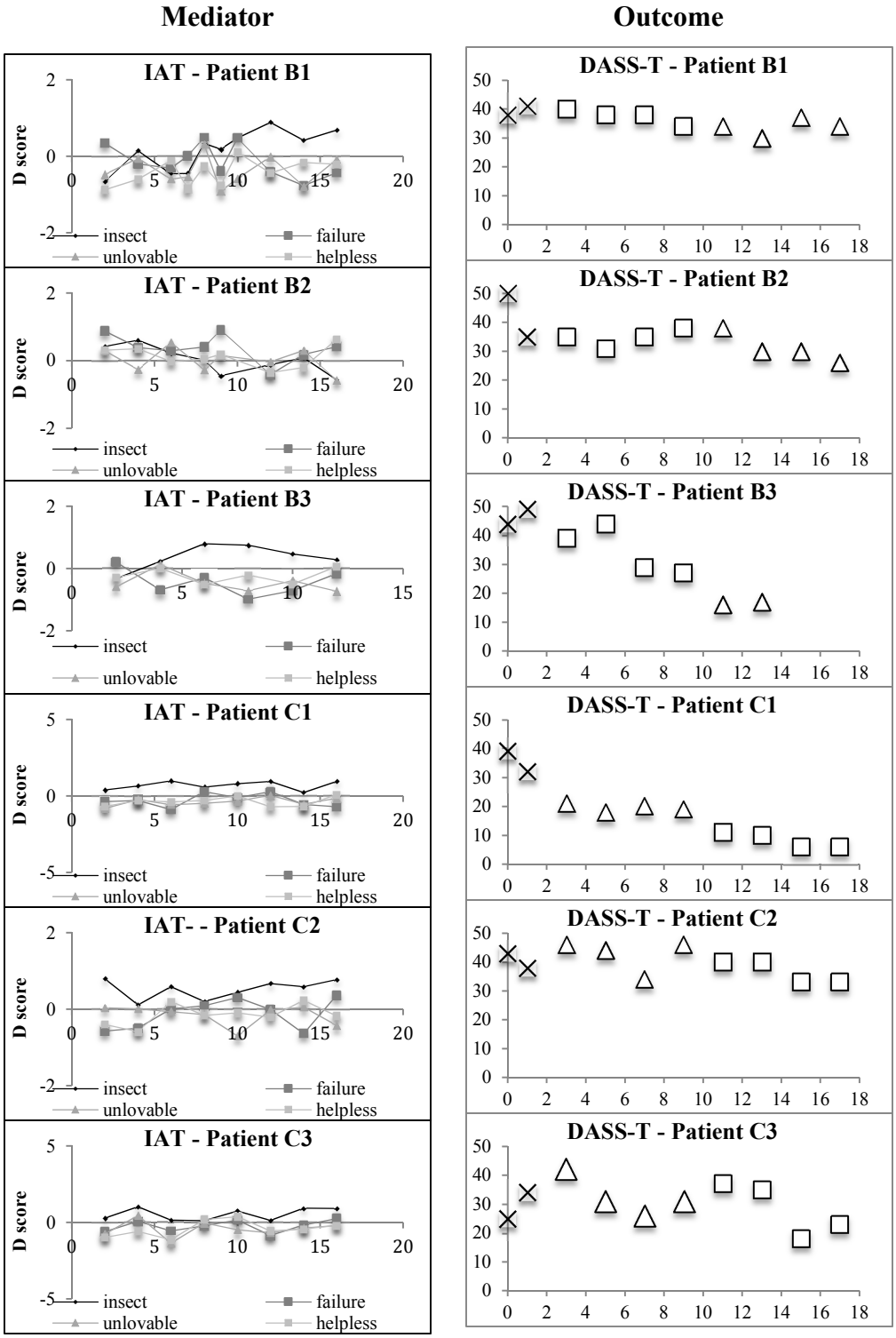
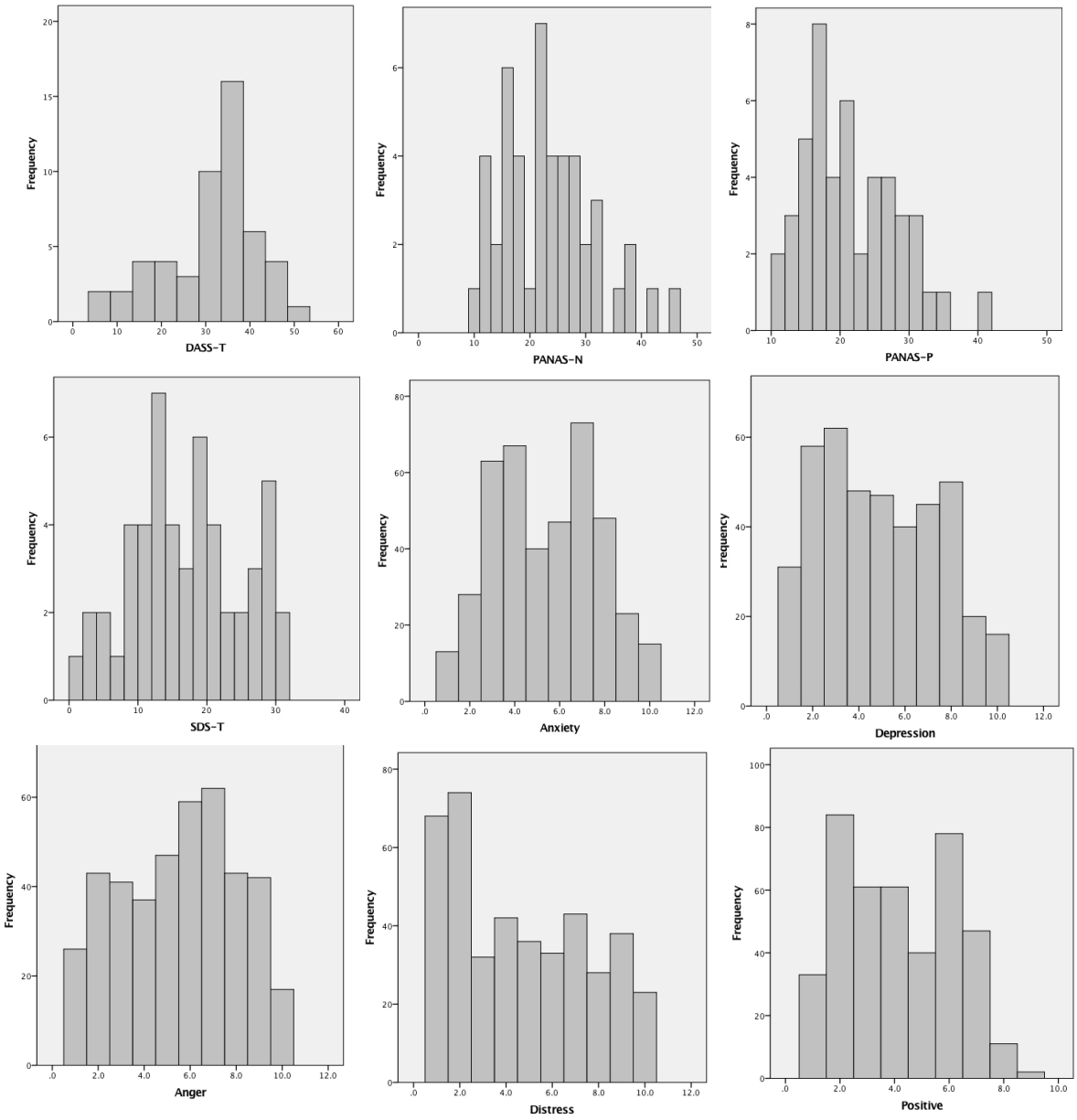
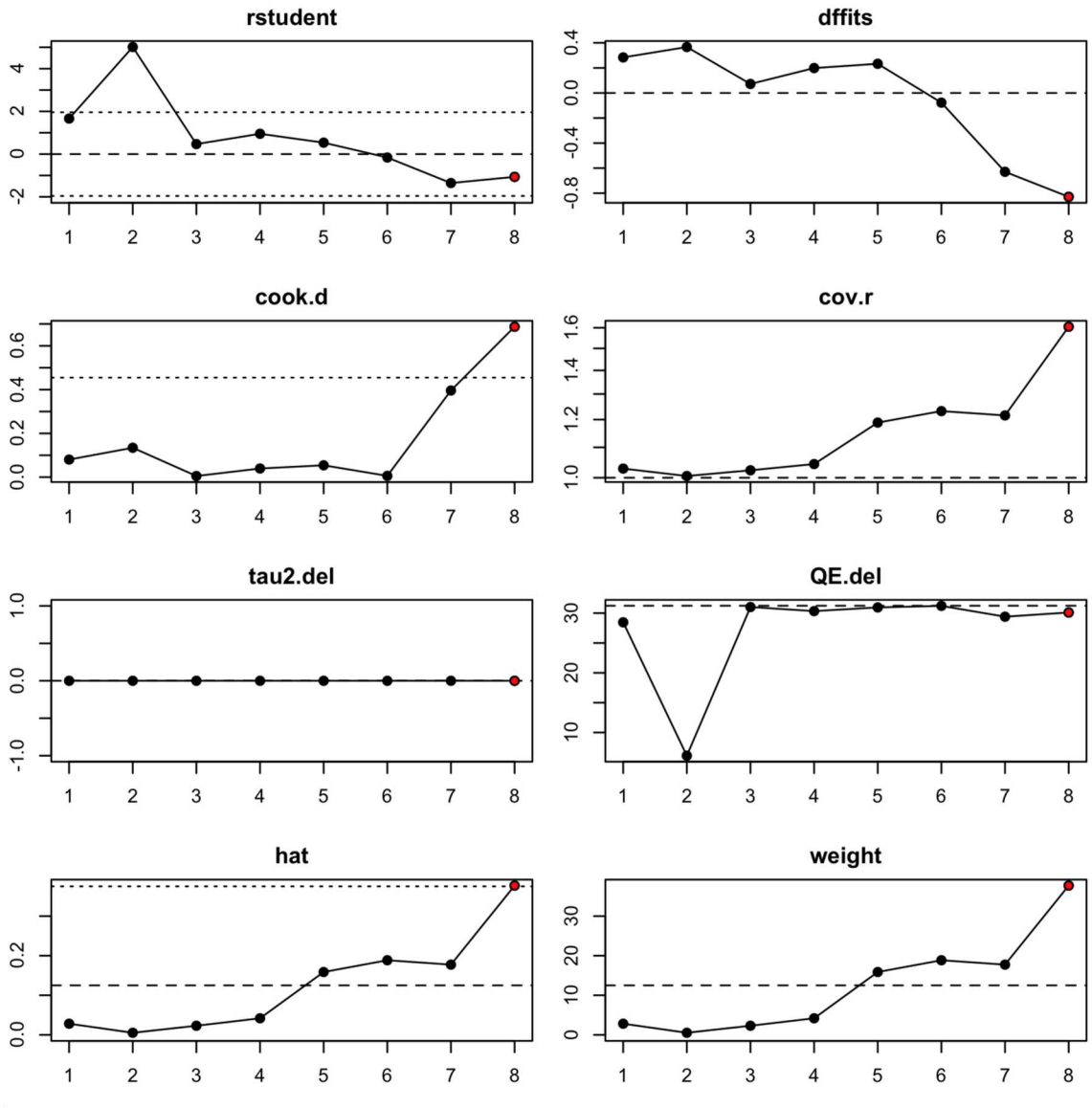


Figure A18. Graphs of session-by-session mediator of implicit associations and outcome of depression, anxiety and stress. ◆ = mediator data point; □ = baseline data points; Δ = behavioral component data points, □ = cognitive component data points

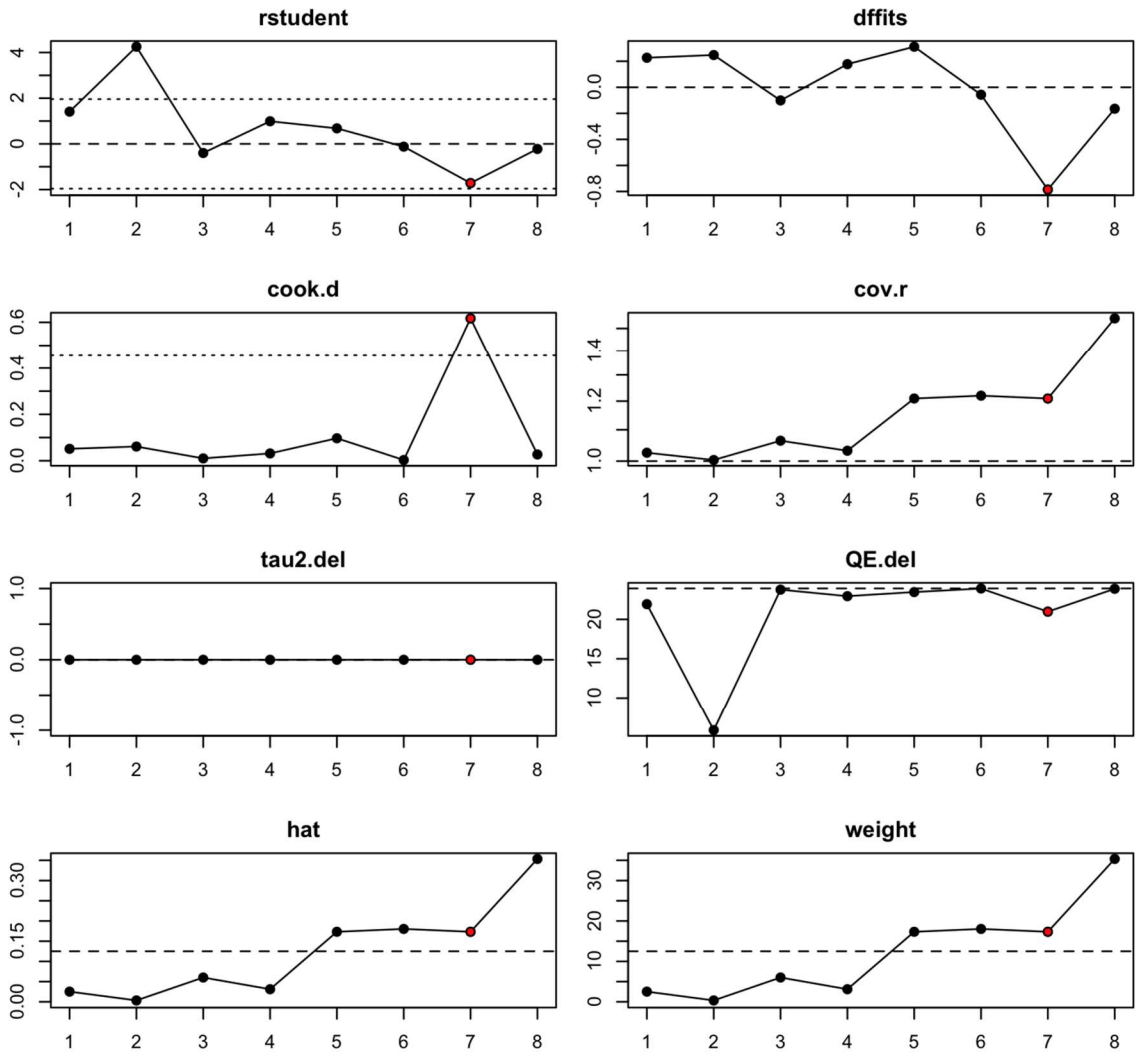
Appendix B



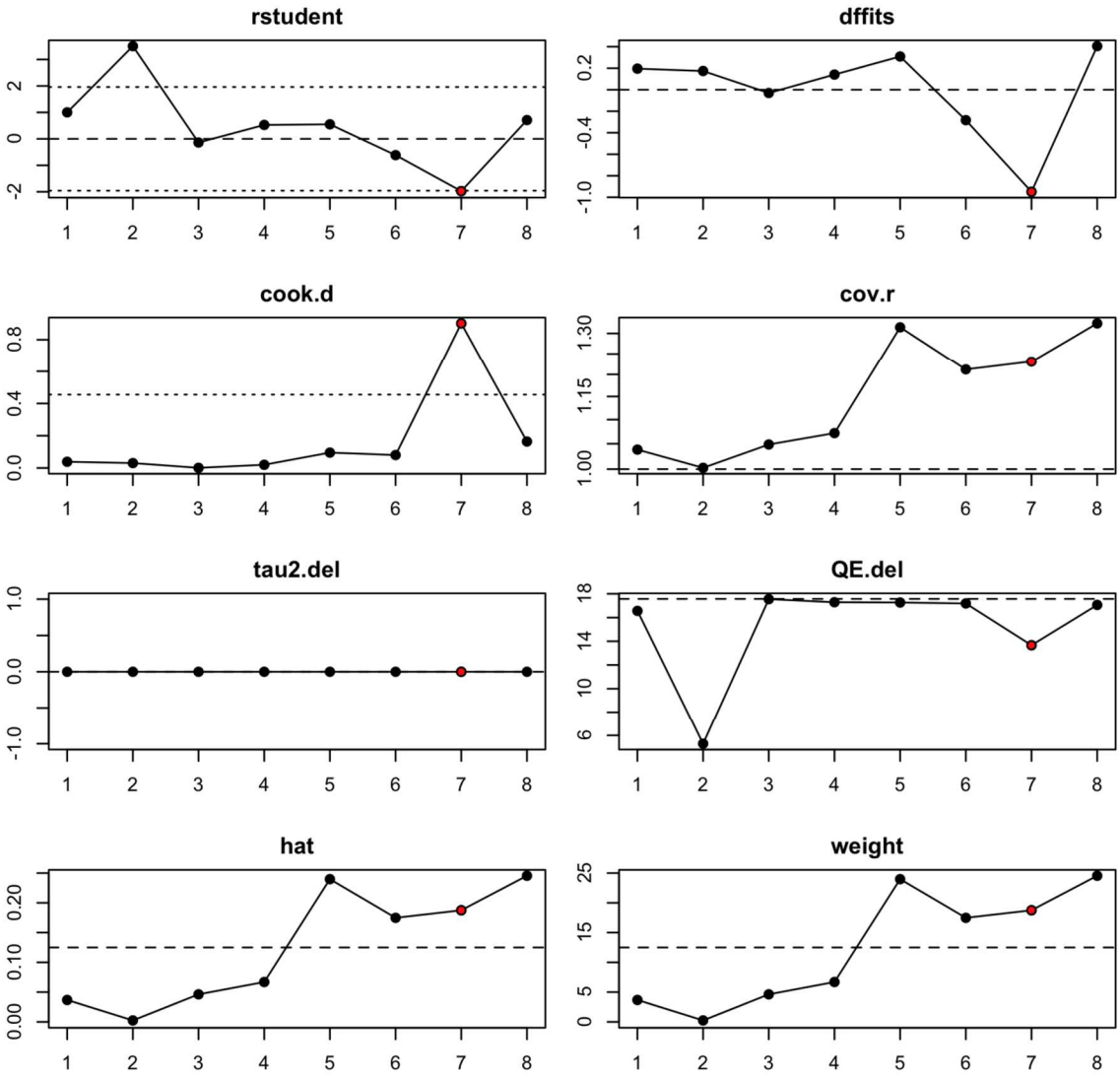
Appendix Figure B1. Histograms of outcome variables, starting from top left: DASS-T, PANAS-Negative, PANAS-Positive, SDS-T, daily anxiety, daily depression, daily anger, guilt and shame, daily distress, daily positive emotions



Appendix Figure B2. Influence diagnostics of aggregation of overall effects. Note, 1=DASS-T effect size, 2=PANAS negative effect size, 3=PANAS positive effect size, 4=SDS-T, 5=daily anxiety, 6=daily depression, 7=daily anger, guilt, and shame, 8=daily positive emotions.



Appendix Figure B3. Influence diagnostics of aggregation of effect of treatment compared to baseline within the behavioral group. Note, 1=DASS-T effect size, 2=PANAS negative effect size, 3=PANAS positive effect size, 4=SDS-T effect size, 5=daily anxiety effect size, 6=daily depression effect size, 7=daily anger, guilt, and shame effect size, 8=daily positive emotions effect size.



Appendix Figure B4. Influence diagnostics of aggregation of incremental effect of first component compared to baseline within the behavioral group. Note, 1=DASS-T effect size, 2=PANAS negative effect size, 3=PANAS positive effect size, 4=SDS-T effect size, 5=daily anxiety effect size, 6=daily depression effect size, 7=daily anger, guilt, and shame effect size, 8=daily positive emotions effect size.

Appendix C

Form C1. Treatment session coding sheet

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
Psychoeducation Mood is composed of emotions (This factor refers to the general idea that emotions or feelings comprise mood. It does not refer to the parts of mood, which is a variable to code below.)																	
There is positive and negative mood (Also acceptable is "there are positive and negative emotions".)																	
Tendencies to generally experience varying degrees of mood (This refers to the general idea that some people have too much negative mood or too little positive mood.)																	
Effects of low positive mood (Effects of low positive mood include fatigue, irritability, anhedonia, etc.)																	
Effective of high negative mood (Effects of high negative mood include rapid heartbeat, sweating, etc.)																	
Parts of mood (The three parts of mood are behaviors, sensations, and thoughts. This is different from the first coding variable in psychoeducation, which talks about what is the definition of mood).																	
Mood cycle (Each part of mood affects other parts of mood, all of which affect mood more generally. This factor can also include upward and downward mood cycles).																	
Treatment rationale (This factor need only address that there are two general parts of this treatment – one that focuses on changing one's behavior and the other that focuses on working with one's thoughts in an effort to increase one's overall positive mood or decrease one's overall negative mood)																	
Cognitive Focus of the module (i.e., to influence our mood by working with our thoughts through concrete strategies or skills learned in session)																	
Review the importance of practice																	

(For this factor to be checked, it must include more than just a mention of the word "practice". It must at minimum clearly state that practice is important and/or explain why it is important)																		
The effect of restructuring thoughts (This factor simply refers to the general idea that by working with our thoughts we can increase our negative mood. Often examples of how interpretations of events affect our mood and behavior are used here. It differs from the first coding variable in Cognitive by simply expanding on it.)																		
Overestimate the likelihood of negative event Introduce *overestimate the likelihood of negative event* (For this factor to be checked, it must include more than a sentence indicating that we are going to work on decreasing this tendency. It must explain *how* the client is going to decrease this tendency by explaining how to use the skill. It may be done with or without use of the workbook.)																		
Highlight in-session *overestimate the likelihood of negative event* (This factor is checked only when the client or therapist notices that the client has just engaged in use of this skill while in session at any point during session or while at home as client reviews homework in session.)																		
Practice in-session for *overestimate the likelihood of negative event* (This can be done formally through use of handout or informally orally.)																		
Use handout for in-session *overestimate the likelihood of negative event* (This box can be checked when client or therapist is clearly using the workbook to work through this exercise. Client need not necessary write, but they must be following step by step instructions written in the workbook. For this box to be checked the coding variable "Practice in-session" must also be checked.)																		
Assign *overestimate the likelihood of negative event* for																		

homework (This box can be checked when therapist assigns any homework related to this skill.)																		
Blowing things out of proportion / catastrophizing Introduce *Blowing things out of proportion / catastrophizing* (For this factor to be checked, it must include more than a sentence indicating that we are going to work on decreasing this tendency. It must explain *how* the client is going to decrease this tendency by explaining how to use the skill. It may be done with or without use of the workbook.)																		
Highlight in-session *Blowing things out of proportion / catastrophizing* (This factor is checked only when the client or therapist notices that the client has just engaged in use of this skill while in session at any point during session or while at home as client reviews homework in session.)																		
Practice in-session for *Blowing things out of proportion / catastrophizing* (This can be done formally through use of handout or informally orally.)																		
Use handout for in-session *Blowing things out of proportion / catastrophizing* (This box can be checked when client or therapist is clearly using the workbook to work through this exercise. Client need not necessary write, but they must be following step by step instructions written in the workbook. For this box to be checked the coding variable "Practice in-session" must also be checked.)																		
Assign *Blowing things out of proportion / catastrophizing* for homework (This box can be checked when therapist assigns any homework related to this skill.)																		
Balancing Responsibility Introduce *Balancing Responsibility* (For this factor to be checked, it must include more than a sentence indicating that we are going to work on decreasing this tendency. It must explain *how* the client is going to decrease this tendency by explaining how to																		

use the skill. It may be done with or without use of the workbook.)																		
Highlight in-session *Balancing Responsibility* (This factor is checked only when the client or therapist notices that the client has just engaged in use of this skill while in session at any point during session or while at home as client reviews homework in session.)																		
Practice in-session for *Balancing Responsibility* (This can be done formally through use of handout or informally orally.)																		
Use handout for in-session *Balancing Responsibility* (This box can be checked when client or therapist is clearly using the workbook to work through this exercise. Client need not necessary write, but they must be following step by step instructions written in the workbook. For this box to be checked the coding variable "Practice in-session" must also be checked.)																		
Assign *Balancing Responsibility* for homework (This box can be checked when therapist assigns any homework related to this skill.)																		
Three types of tendencies to ignore the positive (anticipation, ownership, recognition) (The therapist need only briefly and generally introduce these tendencies, but she does not need necessarily introduce the skills associated with these tendencies. Further, if only two or three of these tendencies are introduced, then do not check this box.)																		
Introduce the idea of balanced thinking (This factor refers to the idea that we are not trying to encourage only positive thinking but rather thinking that is both positive and negative and more based in evidence.)																		
Silver Lining Introduce *Silver Lining* (For this factor to be checked, it must include more than a sentence indicating that we are going to work on increasing this tendency. It must explain *how* the client is going to increase this tendency by explaining how to use the skill. It may be done with or without use of the																		

workbook.)																		
Highlight in-session *Silver Lining* (This factor is checked only when the client or therapist notices that the client has just engaged in use of this skill while in session at any point during session or while at home as client reviews homework in session.)																		
Practice in-session for *Silver Lining* (This can be done formally through use of handout or informally orally.)																		
Use handout for in-session *Silver Lining* (This box can be checked when client or therapist is clearly using the workbook to work through this exercise. Client need not necessary write, but they must be following step by step instructions written in the workbook. For this box to be checked the coding variable "Practice in-session" must also be checked.)																		
Assign *Silver Lining* for homework (This box can be checked when therapist assigns any homework related to this skill.)																		
Taking Ownership Introduce *Taking Ownership* (For this factor to be checked, it must include more than a sentence indicating that we are going to work on increasing this tendency. It must explain *how* the client is going to increase this tendency by explaining how to use the skill. It may be done with or without use of the workbook.)																		
Highlight in-session *Taking Ownership* (This factor is checked only when the client or therapist notices that the client has just engaged in use of this skill while in session at any point during session or while at home as client reviews homework in session.)																		
Practice in-session for *Taking Ownership* (This can be done formally through use of handout or informally orally.)																		
Use handout for in-session *Taking Ownership* (This box can be checked when client or therapist is clearly using the workbook to work through this exercise. Client need not necessary write, but they must be																		

following step by step instructions written in the workbook. For this box to be checked the coding variable "Practice in-session" must also be checked.)																		
Assign *Taking Ownership* for homework (This box can be checked when therapist assigns any homework related to this skill.)																		
Imagining the Positive Introduce *Imagining the Positive* (For this factor to be checked, it must include more than a sentence indicating that we are going to work on increasing this tendency. It must explain *how* the client is going to increase this tendency by explaining how to use the skill. It may be done with or without use of the workbook.)																		
Highlight in-session *Imagining the Positive* (This factor is checked only when the client or therapist notices that the client has just engaged in use of this skill while in session at any point during session or while at home as client reviews homework in session.)																		
Practice in-session for *Imagining the Positive* (This can be done formally through use of handout or informally orally.)																		
Use handout for in-session *Imagining the Positive* (This box can be checked when client or therapist is clearly using the workbook to work through this exercise. Client need not necessary write, but they must be following step by step instructions written in the workbook. For this box to be checked the coding variable "Practice in-session" must also be checked.)																		
Assign *Imagining the Positive* for homework (This box can be checked when therapist assigns any homework related to this skill.)																		
Review homework (This box is checked any time the therapist asks about the homework from the prior session.)																		
Behavioral Exposure What is avoidance, why do we do it, why is it problematic (This factor refers the evolutionary history of avoidance																		

and why it generates or maintains anxiety)																	
What is a safety signal (This factor can be checked if the therapist defines this construct for the client. It is any factor that reduces anxiety or increases the feeling of safety in an anxiety-provoking situation. The phrase "safety behavior" need not be said but the idea must be discussed for this coding variable to be checked)																	
Development of list of situations avoided because of unpleasant feelings (This is essentially the hierarchy but it need not be called that and it need not be ordered in any order of difficulty. As long as there is at least 2 situations listed/discussed, then this box can be checked.)																	
Identify optimal exposure (This factor does *not* refer to *any* exposure. Rather it specifically refers to the situation that is the absolute most frightening or uncomfortable situation a person can encounter. The client need not ever engage in this exposure during or after treatment.)																	
Define in-session exposure (This factor refers to the exposure that the client plans to engage in in session. It is completing the handout formally by writing or informally verbally by answering questions about expectancy/predicted outcome, terms of exposure, etc. It is NOT actually practicing it in session.)																	
Practice in-session exposure, completing form before and after (This factor is checked when client engages in in-session exposure and answers questions from the workbook formally by writing it out or informally by orally answering these questions with therapist. These questions include, what was your peak distress, did your predicted outcome come true, etc.)																	
Assign exposure homework (This box can be checked when therapist assigns any homework related to this skill.)																	
Review exposure homework (This box is checked any time the therapist asks about the exposure homework from the prior session.)																	

Ask client what learned from homework (If at any point during session, therapist asks client what s/he learned during therapy sessions or between sessions, check this box.)																	
Pleasant activities Focus of module (i.e., to influence our mood by working with our behaviors)																	
Assign monitoring of behaviors and mood (Ask client to complete monitoring form)																	
Review monitoring and what learned (When client completes behavior monitoring form over the prior week, ask client what s/he wrote and what s/he learned. Does not need to formally ask these questions but the answers must somehow be generated throughout sessions for this box to be checked.)																	
Connect behaviors to mood (Reviews the connection between behaviors in mood. i.e., that behavior influences mood and we can increase our mood by engaging positive behavior)																	
Development of list of pleasant activities (This factor is checked if at least 2 or more pleasant activities are written or discussed)																	
Discuss barriers to completion (Any type of discussion around steps needed to complete activities or trouble shooting around problems to complete them or discussion of obstacles that hinder completion of activities)																	
Assign activity homework (This box can be checked when therapist assigns any homework related to this skill.)																	
Review homework through imaginal exercise of recounting experience (This box is checked any time the therapist asks about the pleasant activities homework from the prior session.)																	
Client engages in (or therapist encourages) speak slowly (Only check if this occurs during homework review)																	
Client engages in (or therapist encourages) speak in first person																	

(Only check if this occurs during homework review)																	
Client engages in (or therapist encourages) share thoughts (Only check if this occurs during homework review)																	
Client engages in (or therapist encourages) share emotions (Only check if this occurs during homework review)																	
Client engages in (or therapist encourages) share physical sensations (Only check if this occurs during homework review)																	
Assess how vivid imaginal exercise was for client (Only check if this occurs during homework review)																	
Assess what client learned (If at any point during session, therapist asks client what s/he learned during therapy sessions or between sessions, check this box.)																	
Relapse Prevention (Check this box if there is any discussion on what to expect after treatment is over)																	

References

Addis, M. E., & Jacobson, N. S. (2000). A closer look at the treatment rationale and homework compliance in cognitive-behavioral therapy for depression. *Cognitive Therapy and Research, 24*, 313-326.

Aldao, A., Nolen-Hoeksema, S., & Schweizer, S. (2010). Emotion-regulation strategies across psychopathology: A meta-analytic review. *Clinical Psychology Review, 30*, 217–237.

American Psychological Association. (1995). *Template for developing guidelines: Interventions for mental disorders and psychosocial aspects of physical disorders*. Washington, DC: Author.

Antony, M.M., Bieling, P.J., Cox, B.J., Enns, M.W., & Swinson, R.P. (1998). Psychometric properties of the 42-item and 21-item versions of the Depression Anxiety Stress Scales (DASS) in clinical groups and a community sample. *Psychological Assessment, 10*, 176-181.

APA Presidential Task Force on Evidence-Based Practice. (2006). Evidence-based practice in psychology. *American Psychologist, 61*, 271-285.

Arch, J. J., Eifert, G. H., Davies, C., Plumb Vilardaga, J. C., Rose, R. D., & Craske, M. G. (2012). Randomized clinical trial of cognitive behavioral therapy (CBT) versus acceptance and commitment therapy (ACT) for mixed anxiety disorders. *Journal of Consulting and Clinical Psychology, 80*, 750-765.

Arch, J. J., Wolitzky-Taylor, K., Eifert, G. H., & Craske, M. G. (2012). Longitudinal treatment mediation of traditional cognitive behavioral therapy and acceptance

and commitment therapy for anxiety disorders. *Behavior Research and Therapy, 50*, 469-478.

Baer, R. A. (2009). Self-focused attention and mechanisms of change in mindfulness-based treatment. *Cognitive Behaviour Therapy, 38*, 15-20.

Bandura, A. (1982). Self-efficacy mechanism in human agency. *American Psychologist, 37*, 122-147.

Bandura, A. (1977). *Social Learning Theory*. Englewood Cliffs, NJ: Prentice-Hall

Bandura, A. (1983). Self-efficacy determinants of anticipated fears and calamities. *Journal of Personality and Social Psychology, 45*, 464–469.

Barlow, D. H., Fairchione, T. J., Fairholme, C. P., Ellard, K. K., Boisseau, C. L., Allen, L. B., & Ehrenreich May, J. T. (2010). *Unified Protocol for Treatment of Emotional Disorders* (1st ed.). New York: Oxford University Press.

Barlow, D. H., Nock, M. K., & Herzen, M. (2009). *Single case experimental designs: Strategies for studying behavior change* (3rd ed.). Boston: Pearson Education, Inc.

Baron, R. M., & Kenny, D. A. (1986). The moderator-mediator variable distinction in social psychological research: Conceptual, strategic, and statistical considerations. *Journal of Personality and Social Psychology, 51*, 1173-1182.

Beck, A. T., Rush, A. J., Shaw, B. F., & Emergy, G. (1979). *Cognitive therapy of depression*. New York: The Guilford Press.

Beck, J. S. (2011). *Cognitive Behavior Therapy* (2nd ed.). New York: The Guilford Press.

Berking, M., Ebert, D., Cuijpers, P., & Hofmann, S. G. (2013). Emotion regulation skills training enhances the efficacy of inpatient cognitive behavioral therapy for major

depressive disorder: A randomized controlled trial. *Psychotherapy and Psychosomatics, 82*, 234-245.

Berking, M., Neacsiu, A., Comtois, K. A., & Linehan, M. M. (2009). The impact of experiential avoidance on the reduction of depression in treatment for borderline personality disorder. *Behaviour Research and Therapy, 47*, 663–670.

Berking, M., Wupperman, P., Reichardt, A., Pejic, T., Dippel, A., & Znoj, H. (2008). Emotion-regulation skills as a treatment target in psychotherapy. *Behaviour Research and Therapy, 46*, 1230–1237.

Bond, F. W., & Bunce, D. (2000). Mediators of change in emotion-focused and problem-focused worksite stress management interventions. *Journal of Occupational Health Psychology, 5*, 156-163.

Bond, F. W., Hayes, S. C., Baer, R. A., Carpenter, K. M., Guenole, N., Orcutt, H. K., . . . Zettle, R. D. (2011). Preliminary psychometric properties of the Acceptance and Action Questionnaire–II: A revised measure of psychological inflexibility and experiential avoidance. *Behavior Therapy, 42*, 676-688.

Borkovec, T. D., & Costello, E. (1993). Efficacy of applied relaxation and cognitive-behavioral therapy in the treatment of generalized anxiety disorder. *Journal of Consulting and Clinical Psychology, 61*, 611-619.

Bouton, M. E. (2004). Context and behavioral processes in extinction. *Learning & Memory, 11*, 485-494.

Boyd, T. L., & Levis, D. J. (1983). Exposure is a necessary condition for fear-reduction: A reply to De Silva and Rachman. *Behavior Research and Therapy, 21*, 143-149.

Brown, L., Wiley, J. F., Wolitzky-Taylor, K., Roy-Byrne, P., Sherbourne, C., Stein, M. B., . . . Craske, M. G. (2014). Changes in self-efficacy and outcome expectancy as predictors of anxiety outcomes from the CALM study. *Depression and Anxiety, 31*, 678-689.

Burgess, E., & Haaga, D. A. F. (1994). The Positive Automatic Thoughts Questionnaire (ATQ-P) and the Automatic Thought Questionnaire - Revised (ATQ-RP): Equivalent measures of positive thinking. *Cognitive Therapy and Research, 18*, 15-23.

Burns, D., & Spangler, D. L. (2000). Does psychotherapy homework lead to improvements in depression in cognitive–behavioral therapy or does improvement lead to increased homework compliance? *Journal of Consulting and Clinical Psychology, 68*, 46-56.

Cardaciotto, L., Herbert, J. D., Forman, E. M., Moitra, E., & Farrow, V. (2008). The assessment of present-moment awareness and acceptance: The Philadelphia Mindfulness Scale. *Assessment, 15*, 204-223.

Casey, L. M., Newcombe, P. A., & Oei, T. P. S. (2005). Cognitive mediation of panic severity: The role of catastrophic misinterpretation of bodily sensations and panic self-efficacy. *Cognitive Therapy and Research, 29*, 187–200.

Chambless, D. L., Sanderson, W. C., Shoham, V., Bennett Johnson, S., Pope, K. S., Crits-Christoph, P., et al. (1996). An update on empirically validated therapies. *The Clinical Psychologist, 49*, 5-18.

Chambless, D. L., Tran, G. Q., & Glass, C. R. (1997). Predictors of response to cognitive-behavioral group therapy for social phobia. *Journal of Anxiety Disorders, 11*, 221–240.

Ching, L. E., & Dobson, K. S. (2010). An investigation of extreme responding as a mediator of cognitive therapy for depression. *Behaviour Research and Therapy, 48*, 266-274.

Chorpita, B. F., Bernstein, A., & Daleiden, E. L. (2011). Empirically guided coordination of multiple evidence-based treatments: An illustration of relevance mapping in children's mental health services. *Journal of Consulting and Clinical Psychology, 79*, 470-480.

Chorpita, B. F., & Daleiden, E. L. (2009). Mapping evidence-based treatments for children and adolescents: Application of the distillation and matching model to 615 treatments from 322 randomized trials. *Journal of Consulting and Clinical Psychology, 77*, 566-579.

Clark, D. M., Salkovskis, P. M., Lars-Göran, Ö., Breitholtz, E., Koehler, K. A., Westling, B. E., . . . Gelder, M. (1997). Misinterpretation of body sensations in panic disorder. *Journal of Consulting and Clinical Psychology 65*, 1203–1213.

Cloitre, M., Stovall-McClough, C., Nooner, K., Zorbas, P., Cherry, S., Jackson, C. L., . . . Petkova, E. (2010). Treatment for PTSD related to childhood abuse: A randomized controlled trial. *The American Journal of Psychiatry, 167*, 915-924.

Compare, A., Zarbo, C., Shonin, E., Van Gordon, W., & Marconi, C. (2014). Emotional regulation and depression: A potential mediator between heart and mind. *Cardiovascular Psychiatry and Neurology*, *2014,* 324374.

Coon, D. W., & Thompson, L. W. (2003). The relationship between homework compliance and treatment outcomes among older adult outpatients with mild-to-moderate depression. *The American Journal of Geriatric Psychiatry, 11*, 53–61.

Craske, M. G., Kircanski, K., Zelikowsky, M., Mystkowski, J., Chowdhury, N., & Baker, A. (2008). Optimizing inhibitory learning during exposure therapy. *Behaviour Research and Therapy, 46*, 5-27.

Craske, M. G., Liao, B., Brown, L., & Vervliet, B. (2012). Role of inhibition in exposure therapy. *Journal of Experimental Psychopathology, 3*, 322-345.

Craske, M. G., & Rachman, S. J. (1987). Return of fear: Perceived skill and heart-rate responsivity. *British Journal of Clinical Psychology and Psychotherapy, 26*, 187–199.

Crawford, J. R., & Henry, J. D. (2004). The Positive and Negative Affect Schedule (PANAS): Construct validity, measurement properties and normative data in a large non-clinical sample. *British Journal of Clinical Psychology, 43*, 245-265.

Cuijpers, P., Berking, M., Andersson, G., Quigley, L., Kleiboer, A., & Dobson, K. S. (2013). A meta-analysis of cognitive-behavioural therapy for adult depression, alone and in comparison with other treatments. *Canadian Journal of Psychiatry, 58*, 376-385.

Cuijpers, P., Smit, F., Bohlmeijer, E., Hollon, S. D., & Andersson, G. (2010). Efficacy of cognitive-behavioural therapy and other psychological treatments for adult depression: Meta-analytic study of publication bias. *British Journal of Psychiatry, 196*, 173-178.

DeRubeis, R. J., Evans, M. D., Hollon, S. D., Garvey, M. J., Grove, W. M., & Tuason, V. B. (1990). How does cognitive therapy work? Cognitive change and symptom change in cognitive therapy and pharmacotherapy for depression. *Journal of Consulting and Clinical Psychology, 58*, 862-869.

Dimidjian, S., Barrera, M., Martell, C., Munoz, R. F., & Lewinsohn, P. M. (2011). The origins and current status of behavioral activation treatments for depression. *Annual Review of Clinical Psychology, 7*, 1-38.

Dimidjian, S., Hollon, S. D., Dobson, K. S., Schmaling, K. B., Kohlenberg, R. J., Addis, M. E., . . . Jacobson, N. S. (2006). Randomized trial of behavioral activation, cognitive therapy, and antidepressant medication in the acute treatment of adults with major depression. *Journal of Consulting and Clinical Psychology, 74*, 658-670.

Dobson, K. S. (1989). A meta-analysis of the efficacy of cognitive therapy for depression. *Journal of Consulting and Clinical Psychology, 57*, 414–419.

Dobson, K. S. (2013). The science of CBT: Toward a metacognitive model of change? *Behavior Therapy, 44*, 224-227.

Dozios, D. J. A., Bieling, P. J., Patelis-Siotis, I., Hoar, L., Chudzik, S., & McCabe, K. (2009). Changes in self-schema structure in cognitive therapy for major depressive disorder: A randomized clinical trial. *Journal of Consulting and Clinical Psychology, 77*, 1078-1088.

Edelman, R. E., & Chambless, D. L. (1993). Compliance during sessions and homework in exposure-based treatment of agoraphobia. *Behaviour Research and Therapy, 31*, 767-773.

Fava, M., Bless, E., Otto, M. W., Pava, J. A., & Rosenbaum, J. F. (1994). Dysfunctional attitudes in major depression. *The Journal of Nervous and Mental Disease, 182,* 45-49.

Febbraro, G. A. R., & Clum, G. A. (1998). Meta-analytic investigation of the effectiveness of self-regulatory components in the treatment of adult problem behaviors. *Clinical Psychology Review, 18*, 143–161.

Fledderus, M., Bohlmeijer, E., & Pieterse, M. E. (2010). Does experiential avoidance mediate the effects of maladaptive coping styles on psychopathology and mental health? *Behavior Modification, 34,* 503-519.

Forman, E. M., Herbert, J. D., Moitra, E., Yeomans, P. D., & Geller, P. A. (2007). A randomized controlled effectiveness trial of acceptance and committment therapy and cognitive therapy for anxiety and depression. *Behavior Modification, 31*, 772-799.

Garratt, G., Ingram, R. E., Rand, K. L., & Sawalani, G. (2007). Cognitive processes in cognitive therapy: Evaluation of the mechanisms of change in the treatment of depression. *Clinical Psychology: Science and Practice, 14*, 224-239.

Gloaguen, V., Cottraux, J., Cucherat, M., & Blackburn, I. (1998). A meta-analysis of the effects of cognitive therapy in depressed patients. *Journal of Affective Disorders, 49*, 59–72.

Gratz, K. L., & Roemer, L. (2004). Multidimensional assessment of emotion regulation and dysregulation: Development, factor structure, and initial validation of the Difficulties in Emotion Regulation Scale. *Journal of Psychopathology and Behavioral Assessment, 26*, 41–54.

Greenwald, A. G., & Nosek, B. A. (2001). Health of the Implicit Association Test at age 3. *Zeitschrift für Experimentelle Psychologie, 48*, 85-93.

Greenwald, A. G., Nosek, B. A., & Banaji, M. R. (2003). Understanding and using the Implicit Association Test: I. An improved scoring algorithm. *Journal of Personality and Social Psychology, 85*, 197-216.

Greenwald, A. G.., McGhee, D., & Schwartz, J. L. K. (1998). Measuring individual differences in implicit cognition: The Implicit Association Test. *Journal of Personality and Social Psychology, 6*, 1464-1480.

Gross, J. J. (1998). Antecedent- and response-focused emotion regulation: Divergent consequences for experience, expression, and physiology. *Journal of Personality and Social Psychology, 74*, 224-237.

Gunter, R. W., & Whittal, M. L. (2010). Dissemination of cognitive-behavioral treatments for anxiety disorders: Overcoming barriers and improving patient access. *Clinical Psychology Review, 30*, 194-202.

Hannesdottir, D. K., & Ollendick, T. H. (2007). The role of emotion regulation in the treatment of child anxiety disorders. *Clinical Child and Family Psychology Review, 10*, 275-293.

Harley, R., Susan, S., Safren, S., Jacobo, M., & Fava, M. (2008). Adaptation of dialectical behavior therapy skills training group for treatment-resistant depression. *Journal of Nervous and Mental Disease, 196*, 136-143.

Harned, M. S., Korslund, K. E., Foa, E. B., & Linehan, M. M. (2012). Treating PTSD in suicidal and self-injuring women with borderline personality disorder:

Development and preliminary evaluation of a Dialectical Behavior Therapy Prolonged Exposure Protocol. *Behaviour Research and Therapy, 50*, 381–386.

Hawley, K. M., & Weisz, J. R. (2005). Youth versus parent working alliance in usual clinical care: Distinctive associations with retention, satisfaction, and treatment outcome. *Journal of Clinical Child and Adolescent Psychology, 34*, 117-128.

Hayes, S. C., Strosahl, K. D., & Wilson, K. G. (1999). *Acceptance and commitment therapy: An experiential approach to behavior change*. New York: Guilford Press.

Hoffart, A. (1995). Cognitive mediators of situational fear in agoraphobia. *Journal of Behavior Therapy and Experimental Psychiatry, 26*, 313-320.

Hoffart, A., Sexton, H., Hedly, L. M., & Martinsen, E. W. (2008). Mechanisms of change in cognitive therapy for panic disorder with agoraphobia. *Journal of Behavior Therapy and Experimental Psychiatry, 39*, 262-275.

Hofmann, S. G. (2004). Cognitive mediation of treatment change in social phobia. *Journal of Consulting and Clinical Psychology, 72*, 392–399.

Hofmann, S. G., Meuret, A. E., Rosenfield, D., Suvak, M. K., Barlow, D. H., Gorman, J. M., et al. (2007). Preliminary evidence for cognitive mediation during cognitive-behavioral therapy of panic disorder. *Journal of Consulting and Clinical Psychology, 75*, 374-379.

Hollon, S. D., & Kendall, P. C. (1980). Cognitive self-statements in depression: Development of an automatic thoughts questionnaire. *Cognitive Therapy and Research, 4*, 383-395.

Hollon, S. D., Thase, M. E., & Markowitz, J. C. (2002). Treatment and prevention of depression. *Psychological Science in the Public Interest, 3*, 39-77.

Hothersall, D. (2004). *History of Psychology* (4th edition ed.). New York: McGraw Hill.

Jacobson, N. S., Dobson, K. S., Truax, P. A., Addis, M. E., Koerner, K., Gollan, J. K., et al. (1996). A component analysis of cognitive-behavioral treatment for depression. *Journal of Consulting and Clinical Psychology, 64*, 295-304.

Jamieson, J. P., Nock, M. K., & Mendes, W. B. (2012). Mind over matter: Reappraising arousal improves cardiovascular and cognitive responses to stress. *Journal of Experimental Psychology. General, 14*, 417-422.

Johansson, P., & Høglend, P. (2007). Identifying mechanisms of change in psychotherapy: Mediators of treatment outcome. *Clinical Psychology and Psychotherapy, 14*, 1-9.

Kashdan, T. B., Barrios, V., Forsyth, J. P., & Steger, M. F. (2006). Experiential avoidance as a generalized psychological vulnerability: Comparisons with coping and emotion regulation strategies. *Behaviour Research and Therapy, 44*, 1301-1320.

Kazdin, A. E. (1974). Reactive self-monitoring: The effects of response desirability, goal setting, and feedback. *Journal of Consulting and Clinical Psychology, 42*, 704-716.

Kazdin, A. E. (2000). *Psychotherapy for children and adolescents: Directions for research and practice*. New York: Oxford University Press.

Kazdin, A. E. (2007). Mediators and mechanisms of change in psychotherapy research. *Annual Review of Clinical Psychology, 3*, 1-27.

Kendall, P. C., & Hollon, S. D. . (1989). Anxious self-talk: Development of the Anxious Self-Statements Questionnaire (ASSQ). *Cognitive Therapy and Research, 13*, 81–93.

Kendall, P. C., Howard, B. L., & Hays, R. C. (1989). Self-referent speech and psychopathology: The balance of positive and negative thinking. *Cognitive Therapy and Research, 13*, 583-598.

Keng, S. L., Smoski, M. J., & Robins, C. J. (2011). Effects of mindfulness on psychological health: A review of empirical studies. *Clinical Psychology Review, 31*, 1041–1056.

Kleim, B., Grey, N., Wild, J., Nussbeck, F. W., Stott, R., Hackmann, A., . . . Ehlers, A. (2013). Cognitive change predicts symptom reduction with cognitive therapy for posttraumatic stress disorder. *Journal of Consulting and Clinical Psychology, 81*, 383-393.

Klein, A. M., Becker, E. S., & Rinck, M. (2011). Approach and avoidance tendencies in spider fearful children: The Approach-Avoidance Task. *Journal of Child and Family Studies, 20*, 224-231.

Kopp, J. (1988). Self-monitoring: A literature review of research and practice. *Social Work Research Abstracts, 24*, 8-20.

Kovacs, M., Sherrill, J., George, C. J., Pollock, M., Tumuluru, R. V., & Ho, V. (2006). Contextual emotion-regulation therapy for childhood depression: Description and pilot testing of a new intervention. *Journal of the American Academy of Child and Adolescent Psychiatry, 45*, 892–903.

Krieglmeier, R., Deutsch, R., De Houwer, J., & De Raedt, R. (2010). Being moved: Valence activates approach-avoidance behavior independent of evaluation and approach-avoidance intentions. *Psychological Science, 21,* 607-613.

Lang, P. J., Bradley, M. M., & Cuthbert, B. N. (1999). *International Affective Picture System (IAPS): Technical manual and affective ratings.* Gainesville, FL: The Center for Research in Psychophysiology, University of Florida.

Lappalainen, R., Lehtonen, T., Skarp, E., Taubert, E., Ojanen, M., & Hayes, S. C. (2007). The impact of CBT and ACT models using psychology trainee therapists: A preliminary controlled effectiveness trial. *Behavior Modification, 31*, 488-511.

LeBeau, R. T., Davies, C. D., Culver, N. C., & Craske, M. G. (2013). Homework compliance counts in cognitive-behavioral therapy. *Cognitive Behaviour Therapy, 42*, 171–179.

Lecrubier, Y., Sheehan, D., Weiller, E., Amorim, P., Bonora, I., Sheehan, K., . . . Dunbar, G. (1997). The M.I.N.I. International Neuropsychiatric Interview (M.I.N.I.) a short diagnostic structured interview: Reliability and validity according to the CIDI. *European Psychiatry, 12*, 224-231.

Leon, A. C., Shear, M. K., Porten, L., & Klerman, G. L. (1992). Assessing impairment in patients with panic disorder: The Sheehan Disability Scale. *Social Psychiatry and Psychiatric Epidemiology, 27*, 78-82.

Leung, A. W., & Heimberg, R. G. (1996). Homework compliance, perceptions of control, and outcome of cognitive-behavioral treatment of social phobia. *Behaviour Research and Therapy, 34*, 423-432.

Linehan, M. M., Comtois, K. A., Murray, A. M., Brown, M. Z., Gallop, R., Heard, H. L., . . . Lindenboim, N. (2006). Two-year randomized controlled trial and follow-up of dialectical behavior therapy vs therapy by experts for suicidal behaviors and borderline personality disorder. *Archives of General Psychiatry, 63*, 757-766.

Linehan, M. M., Dimeff, L. A., Reynolds, M., Comtois, K. A., Welch, S. S., Heagerty, P., & Kivlahan, D. R. (2002). Dialectical behavior therapy versus comprehensive validation therapy plus 12-step for the treatment of opioid dependent women meeting criteria for borderline personality disorder. *Drug and Alcohol Dependence, 67*, 13-26.

Loerinc, A. G. , Meuret, A., Twohig, M., & Craske, M. G. (In submission). CBT is effective but not sufficient for anxiety disorders: an evaluation of responder rates.

Longmore, R. J., & Worrell, M. (2007). Do we need to challenge thoughts in cognitive behavioral therapy? *Clinical Psychology Review, 27*, 173.

Lovibond, S. H., & Lovibond, P. F. (1995). *Manual for the Depression Anxiety Stress Scales* (2nd ed.). Sydney: Psychology Foundation.

Maddux, J. E., Sherer, M., & Rogers, R. W. (1982). Self-efficacy expectancy and outcome expectancy: Their relationship and their effects on behavioral intentions. *Cognitive Therapy and Research, 6*, 207-211.

Mazzucchelli, T., Kane, R., & Rees, C. (2009). Behavioral activation treatments for depression in adults: A meta-analysis and review. *Clinical Psychology: Science and Practice, 16*, 383–411.

Mennin, D. S. (2004). Emotion regulation therapy for generalized anxiety disorder. *Clinical Psychology and Psychotherapy, 11*, 17–29.

Moeyaert, M., Ferron, J., Beretvas, S. N., & Van den Noortgate, W. (2014). From a single-level analysis to a multilevel analysis of single-case experimental designs. *Journal of School Psychology, 52*, 83-103.

Naifeh, J. A., Tull, M. T., & Gratz, K. L. (2012). Anxiety sensitivity, emotional avoidance, and PTSD symptom severity among crack/cocaine dependent patients in residential treatment. *Cognitive Therapy and Research, 36*, 247–257.

Neimeyer, R. A., & Feixas, G. (1990). The role of homework and skill acquisition in the outcome of group cognitive therapy for depression. *Behavior Therapy, 21*, 281–292.

Norton, P. J., & Price, E. C. (2007). A meta-analytic review of adult cognitive-behavioral treatment outcome across the anxiety disorders. *The Journal of Nervous and Mental Disease, 6*, 521-531.

Nosek, B. A., Greenwald, A. G., & Banaji, M. R. (2007). The Implicit Association Test at age 7: A methodological and conceptual review. In J. A. Bargh (Ed.), *Automatic Processes in Social Thinking and Behavior*: Psychology Press.

Olatunji, B. O., Rosenfield, D., Tart, C. D., Cottraux, J., Powers, M. B., & Smits, J. A. J. (2013). Behavioral versus cognitive treatment of obsessive-compulsive disorder: An examination of outcome and mediators of change. *Journal of Consulting and Clinical Psychology, 81*, 415-428.

Ong, J. C., Shapiro, S. L., & Manber, R. (2008). Combining mindfulness meditation with cognitive-behavior therapy for insomnia: A treatment-development study. *Behavior Therapy, 39*, 171–182.

Ougrin, D. (2011). Efficacy of exposure versus cognitive therapy in anxiety disorders: Systematic review and meta-analysis. *BMC Psychiatry, 11*, 200.

Peacock, E. J., & Wong, P. T. P. (1990). The Stress Appraisal Measure (SAM): A multidimensional appraoch to cognitive appraisal. *Stress Medicine, 6*, 227-236.

Pricea, M., Andersona, P., Henricha, C. C., & Olasov Rothbaumb, B. (2008). Greater expectations: Using hierarchical linear modeling to examine expectancy for treatment outcome as a predictor of treatment response. *Behavior Therapy, 39*, 398–405.

Quinn, E. P., Brandon, T. H., & Copeland, A. L. (1996). Is task persistence related to smoking and substance abuse: The application of learned industriousness theory to addictive behaviors. *Experimental and Clinical Psychopharmacology, 4*, 186-190.

Raes, A K., Koster, E. H. W., Loeys, T., & De Raedt, R. (2011). Pathways to change in one-session exposure with and without cognitive intervention: An exploratory study in spider phobia. *Journal of Anxiety Disorders, 25*, 964-971.

Rinck, M., & Becker, E. S. (2007). Approach and avoidance in fear of spiders. *Journal of Behaviour Therapy and Experimental Psychiatry, 38*, 105–120.

Rindskopf, D. M. (2014). Nonlinear bayesian analysis for single case designs. *Journal of School Psychology, 52*, 71–81.

Roelofs, K., Putman, P., Schouten, S., Lange, W.-G., Volman, I., & Rinck, M. (2010). Gaze direction differentially affects avoidance tendencies to happy and angry faces in socially anxious individuals. *Behaviour Research and Therapy, 48*, 290-294.

Safran, J. D., Vallis, T. M., Segal, Z. V., Shaw, B. F., Balog, W., Epstein, L., et al. (1987). Measuring session change in cognitive therapy. *Journal of Cognitive Psychotherapy: An International Quarterly, 1*, 117–128.

Safren, S. A., Heimberg, R. G., & Juster, H. R. (1997). Clients' expectancies and their relationship to pretreatment symptomatology and outcome of cognitive-behavioral group treatment for social phobia. *Journal of Consulting and Clinical Psychology, 65*, 694-698.

Safren, S. A., Heimberg, R. G., Lerner, J., Henin, A., Warman, M., & Kendall, P. C. (2000). Differentiating anxious and depressive self-statements: Combined factor structure of the Anxious Self-Statements Questionnaire and the Automatic Thoughts Questionnaire-Revised. *Cognitive Therapy and Research, 24*, 327-344.

Schwarzer, R., & Born, A. (1997). Optimistic self-beliefs: Assessment of general perceived self-efficacy in thirteen cultures. *World Psychology, 3*, 177-190.

Schwarzer, R., & Jerusalem, M. (1995). Generalized Self-Efficacy scale. In J. Weinman, S. Wright & M. Johnston (Eds.), *Measures in health psychology: A user's portfolio. Causal and control beliefs* (pp. 35-37). Windsor, UK: NFER-NELSON.

Shadish, W. R. (2014). Analysis and meta-analysis of single-case designs: An introduction. *Journal of School Psychology, 52*, 109–122.

Shadish, W. R., Hedges, L. V., & Pustejovsky, J. E. (2014). Analysis and meta-analysis of single-case designs with a standardized mean difference statistic: A primer and applications. *Journal of School Psychology, 52*, 15–39.

Sheehan, D. V. (1983). *The Anxiety Disease*. New York: Charles Scribner and Sons.

Sheehan, D. V., Lecrubier, Y., Harnett-Sheehan, K., Amorim, P., Janavs, J., Weiller, E., . . . Dunbar, G. (1998). The M.I.N.I. International Neuropsychiatric Interview (M.I.N.I.): The development and validation of a structured diagnostic psychiatric interview. *Journal of Clinical Psychiatry, 59*, 22-33.

Sheehan, D. V., Lecrubier, Y., Harnett-Sheehan, K., Janavs, J., Weiller, E., Bonara, L. I., . . . Dunbar, G. C. (1997). Reliability and validity of the M.I.N.I. International Neuropsychiatric Interview (M.I.N.I.): According to the SCID-P. *European Psychiatry, 12*, 232-241.

Slee, N., Spinhoven, P., Garnefski, N., & Arensman, E. (2008). Emotion regulation as mediator of treatment outcome in therapy for deliberate self-harm. *Clinical Psychology and Psychotherapy, 15*, 205–216

Sotsky, S. M., Glass, D. R., Shea, T., Pilkonis, P. A., Collins, J. F., Elkin, I., . . . Oliveri, M. E. (1991). Patient predictors of response to psychotherapy and pharmacotherapy: Findings in the NIMH Treatment of Depression Collaborative Research Program. *American Journal of Psychiatry, 148*, 997-1008.

Stewart-Williams, S., & Podd, J. (2004). The placebo effect: Dissolving the expectancy versus conditioning debate. *Psychological Bulletin, 130*, 324-340.

Strong, D. R., Lejuez, C. W., Daughters, S., Marinello, M., Kahler, C. W., & Brown, R. A. (2003). *The computerized mirror tracing task*. Unpublished Manual.

Swaminathan, H., Rogers, H. J., & Horner, R. H. (2014). An effect size measure and Bayesian analysis of single-case designs. *Journal of School Psychology, 52*, 105–122.

Taylor, I. (1985). The reactive effect of self-monitoring of target activities in agoraphobics: A pilot study. *Scandinavian Journal of Behaviour Therapy, 14*, 17-22.

Teachman, B. A., Marker, C. D., & Smith-Janik, S. B. (2008). Automatic associations and panic disorder: Trajectories of change over the course of treatment. *Journal of Consulting and Clinical Psychology, 76*, 988-1002.

Teachman, B. A., & Woody, S. R. (2003). Automatic processing in spider phobia: Implicit fear associations over the course of treatment. *Journal of Abnormal Psychology, 112*, 100-109.

Teasdale, J. D., & Fennell, M. J. V. (1982). Immediate effects on depression of cognitive therapy interventions. *Cognitive Therapy and Research, 6*, 343-352.

Teasdale, J. D., Scott, J., Moore, R. G., Hayhurst, H., Pope, M., & Paykel, E. S. (2001). How does cognitive therapy prevent relapse in residual depression? Evidence from a controlled trial. *Journal of Consulting and Clinical Psychology, 69*, 347-357.

Tull, M. T., & Gratz, K. L. (2008). Further examination of the relationship between anxiety sensitivity and depression: The mediating role of experiential avoidance and difficulties engaging in goal-directed behavior when distressed. *Journal of Anxiety Disorders, 22*, 199–210.

Warmerdam, L., van Straten, A., Jongsma, J., Twisk, J., & Cuijpers, P. (2010). Online cognitive behavioral therapy and problem-solving therapy for depressive symptoms: Exploring mechanisms of change. *Journal of Behavior Therapy and Experimental Psychiatry, 41*, 64-70.

Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: The PANAS scales. *Journal of Personality and Social Psychology, 54*, 1063-1070.

Web of Knowledge. (2012). Results analysis: Publication years 2001-2011 from Thomas Reuters.

Weinrib, A. Z. (2011). *Investigating experiential avoidance as a mechanism of action in a mindfulness intervention.* (Doctor of Philosophy), University of Iowa. Retrieved from http://ir.uiowa.edu/etd/1105

Weisz, J. R., Doss, A. J., & Hawley, K. M. (2005). Youth psychotherapy outcome research: A review and critique of the evidence base. *Annual Review of Psychology, 56*, 337-363.

Westen, D., & Morrison, K. (2001). A multidimensional meta-analysis of treatments for depression, panic, and generalized anxiety disorder: An empirical examination of the status of empirically supported therapies. *Journal of Consulting and Clinical Psychology, 69*, 875-899.

Westen, D., Novotny, C. M., & Thompson-Brenner, H. (2004). The empirical status of empirically supported psychotherapies: Assumptions, findings, and reporting in controlled clinical trials. *Psychological Bulletin, 130*, 631-663.

Westra, H. A., Dozios, D. J. A., & Marcus, M. (2007). Expectancy, homework compliance, and initial change in cognitive-behavioral therapy for anxiety. *Journal of Consulting and Clinical Psychology, 75*, 363-373.

Williams, S. L., Dooseman, G., & Kleinfield, E. (1984). Comparative effectiveness of guided mastery and exposure treatments for intractable phobias. *Journal of Consulting and Clinical Psychology, 52*, 505–518.

Wisco, B. E., & Nolen-Hoeksema, S. (2010). Interpretation bias and depressive symptoms: The role of self-relevance. *Behaviour Research and Therapy, 48*, 1113-1122.

Wolitzky-Taylor, K., Arch, J. J., Rosenfield, D., & Craske, M. G. (2012). Moderators and non-specific predictors of treatment outcome for anxiety disorders: A comparison of cognitive-behavioral therapy to acceptance and commitment therapy. *Journal of Consulting and Clinical Psychology, 80*, 786-799.

Woud, M. L., Becker, E. S., & Rinck, M. (2008). Implicit evaluation bias induced by approach and avoidance. *Cognition and Emotion, 22*, 1187–1197.

Zettle, R. D. (2003). Acceptance and Commitment Therapy (ACT) vs. systematic desensitization in treatment of mathematics anxiety *The Psychological Record, 53*, 197-215.