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The Effectiveness of Empirically Supported Brief Interventions for Depressive and/or Anxiety Disorders for Primary Care Patients: A Systematic Review and Meta-Analysis

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by

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Dissertation

Presented to the Faculty of the Graduate School of

The University of Texas at Austin

in Partial Fulfillment

of the Requirements

for the Degree of

Doctor of Philosophy

The University of Texas at Austin

May 2018

Dedication

This dissertation is dedicated to my parents, Zuoping Zhang and Min Zhu, who have offered their enduring support for my academic pursuant; to my twin brother, Chang'ao Zhang, who is the best brother in the world; and his daughter (my niece), Xinyuan Zhang, who brings me endless joy and happy. Last but not least, to my grandmother, Jiafang He, who has taught me kindness, empathy, care, and love.

Acknowledgements

I am most appreciative to my mentor and friend, Dr. Cynthia Franklin, for her exceptional mentorship and for all her patience and kindness guiding me through my doctoral life. I am also thankful to my committee members, Drs. Beretvas, Choi, and DiNitto for their overwhelming support and patience with me during my dissertation writing. I, too, want to express my gratitude to my other mentors and collaborators who were all so kind and patient with me, Drs. Yuri Jang, Yolanda Padilla, Calvin Streeter, Barbara Jones, Susan De Luca, and Phyllis Solomon at the University of Pennsylvania.

Abstract

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Depressive and anxiety disorders (DADs) are highly prevalent in U.S. primary care systems. Consequences of DADs for primary care patients are real and substantial. While there exist many empirically supported interventions for DADs, only a few them have been adopted for a primary care population. To date, limited investigation has focused on the effectiveness of these empirically supported interventions for DADs when delivered in primary care settings. This dissertation aims to evaluate the effectiveness of empirically supported brief interventions for DADs for primary care patients. Using a systematic review and meta-analysis approach, this dissertation searches across seven electronic databases, six professional websites, peer-reviewed journal articles' reference list, and contact field experts for a pool of articles for meta-analysis. An initial pool of 1,140 articles are identified, after title/abstract screening and full-text review, a final sample of 65 articles are included for final summary and data analysis. Publication bias, risk of bias, and study quality rating are conducted in accordance with the Cochrane guidelines. In addition to descriptive statistics of individual studies, an overall treatment

effect, assuming a random-effect model, and moderator analysis, assuming a mixed-effect model, are performed using Robust Variance Estimation in Meta-regression. Meta-analytic results indicate an overall statistically significant treatment effect of included interventions for primary care patients' DADs. Single-predictor moderator analyses find percentage of married participants, treatment modality (individual versus group), and treatment composition (one versus combined approach) significantly moderates treatment effect size estimate. Multiple-predictor moderator analysis finds that, after controlling for other treatment characteristics, interventions delivered outside primary care settings reported significantly higher treatment effect than those delivered inside primary care settings. Discussions on these results and implications for social work practice, research, education and policy are presented.

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CHAPTER ONE: INTRODUCTION

BACKGROUND AND SIGNIFICANCE

Global and U.S. epidemiology of depressive and anxiety disorders

Depressive and anxiety disorders (DADs) constitute the top leading causes of all non-fatal burden of disease both internationally and in the United States (Murray et al., 2012; Whiteford et al., 2013). Epidemiological studies estimate a global average of lifetime prevalence of 9.2% and 7.3% for depressive and anxiety disorders, respectively (Baxter, Scott, Vos, & Whiteford, 2013; Kessler & Bromet, 2013; Moussavi et al., 2007). The World Health Organization (WHO) ranked depression as the fourth leading cause of disability worldwide and projected that by 2020, it will be the second leading cause (Murray & Lopez, 1996). Many other studies reported similar growth trajectories for anxiety disorders worldwide (Baxter et al., 2014; Kessler et al., 2011), and such growth has been found consistent regardless of a country's region, cultural context, or economic status (Bandelow & Michaelis, 2015). The United States and other developed countries share in this global epidemic and may experience higher prevalence rates than other countries (Ferrari et al., 2013; Scott et al., 2007).

Numerous studies based on nationally representative samples consistently show that depressive and anxiety disorders are the most prevalent mental disorders among the U.S. population. However, the studies differ in identifying which disorder is more prevalent due to changes in diagnostic criteria and classification systems (single disorder or spectrum of disorders). The National Comorbidity Survey and its replication (Kessler & Wang (2008) reported that of all mental disorders, anxiety disorders were the most

prevalent lifetime disorders (using the Diagnostic and Statistical Manual of Mental Disorders (DSM), Fourth Edition, Text Revision) (American Psychiatric Association, 2000) followed by mood disorders (including depressive disorders). Other studies show that major depressive disorder is the most prevalent individual lifetime disorder (16.2%) (González, Tarraf, Whitfield, & Vega, 2010; Strine et al., 2008), and over 35% of Americans have had at least one diagnosis of depressive spectrum disorders in their lifetime (Ford, Giles, & Dietz, 2002). Anxiety spectrum disorders are also among the most common mental disorders in the United States with a lifetime prevalence of 28.8% of the population (Kessler & Wang, 2008).

U.S. epidemiology of comorbid DADs in primary care

In addition to their individual prevalence in the United States, depressive and anxiety disorders often occur together, i.e., as comorbid conditions (Kessler, Chiu, Demler, Merikangas, & Walters, 2005). Patients with one or more anxiety spectrum disorders, including panic disorder, generalized anxiety disorder, social phobia, and others, are also frequently clinically depressed (Gorman, 1996). Studies show that over 70% of individuals with depressive disorders also have anxiety symptoms and that 40 to 70 percent of patients with depressive disorders simultaneously meet criteria for at least one type of anxiety disorder (Kessler, Merikangas, & Wang, 2007).

These high levels of comorbid depressive and anxiety disorders are also seen in U.S. primary care patients (Luxama & Dreyfus, 2014; Prins et al., 2011). A comprehensive review of comorbid depression and anxiety in primary care revealed that these disorders occur at rates that exceed other common medical illness (e.g., hypertension, diabetes), and, more strikingly, over 75% of clinically depressed primary care patients suffer from a current anxiety disorder (Hirschfeld, 2001). In addition,

estimates indicate that up to 90% of U.S. patients diagnosed with depression and anxiety are treated solely in primary care (Archer et al., 2012).

Also complicating the picture is the comorbidity between physical and mental health conditions (e.g., comorbid depression and chronic pain), and the multi-morbid physical and mental disorders (e.g., diabetes in combination with both depression and anxiety) seen in primary care settings. A systematic review and meta-analysis of the prevalence of comorbid depression in Type 2 diabetic patients (n=18,445) (Ali, Stone, Peters, Davies, & Khunti, 2006) reported an overall depression prevalence of 17.6% and indicated that diabetic patients are 1.8 times more likely to suffer from depression than their non-diabetic counterparts (OR=1.77, 95% CI 1.5–2.0). Another study of 2,091 primary care patients (Löwe et al., 2008) reported that over 50% had comorbidities among depression, anxiety, and somatization and that the combined contribution of these diagnoses to functional impairment substantially exceeded the contribution of each individual diagnosis. Empirical reviews of multi-morbidities among primary care patients reported a range from 12.9% to 95.1% prevalence rate across published studies in primary care settings (Violan et al., 2014). Theoretical literature also highlighted the importance of understanding multi-morbid mental and physical disorders in primary care as well as the development of new interventions for treating these disorders (Kemp & Quintana, 2013; Patten, 2013).

Significance and challenges

The high prevalence of co-/multi-morbid DADs in primary care has received growing attention given their detrimental impacts on primary care patients' health. Co-/multi-morbid DADs are associated with significantly higher rates of health care utilization and greater healthcare costs (Glynn et al., 2011). A recent systematic review

(Sinnott, Mc Hugh, Browne, & Bradley, 2013) revealed that primary care co-/multi-morbid DADs are associated more likely to receive disorganized and fragmented care, inadequate disease-specific treatments, challenges in delivering patient-centered care, and poorer doctor-patient communication. Many other studies report similar clinical findings that primary care co-/multi-morbid DADs are strongly correlated with health disparities (Salisbury, Johnson, Purdy, Valderas, & Montgomery, 2011), lower treatment adherence (Fortin, et al., 2006a), lower quality of life (Fortin, et al., 2006b), and poorer functioning (Noël, Frueh, Larme, & Pugh, 2005), among other negative outcomes.

Both the theoretical and empirical literature consistently identifies the need to develop new interventions to manage co-/multi-morbid DADs in primary care. Simms and colleagues (2012), for example, examined the structure of 91 anxiety, depression, and somatic symptoms in a sample of 5,433 primary care patients and emphasized the need to develop a new understanding of DADs symptomatology and clinical manifestations in primary care. In addition, Mercer and colleagues (2009) highlighted the complexity of treating primary care patients with DADs and further underscored an imperative to develop new interventions in terms of underlying theories as well as empirically-based practice guidelines. Wallace and colleagues (2015) emphasized the consistent prevalence of co-/multi-morbid DADs across heterogeneous primary care populations and the need to develop treatments targeted to different populations (e.g., those with chronic pain versus obesity).

In summary, the literature on DADs in primary care suggests that traditional psychosocial interventions often delivered in outpatient specialty mental health care settings may not be appropriate for primary care settings for several reasons. First, the clinical manifestation and etiology of depressive and/or anxiety disorders in primary care patients also being treated for physical health problems might differ from those of

patients seen in outpatient specialty mental health care settings (Katon, Lin, & Kroenke, 2007; Kroenke, 2003). Therefore, change theories of existing DADs interventions may need to be modified for use in primary care (Finucane & Mercer, 2006; Kirmayer, 2001). Second, existing psychosocial interventions may not be appropriate for primary care DADs given different service delivery characteristics. For example, in most outpatient specialty mental health care settings, psychosocial interventions for DADs are delivered on a weekly or bi-weekly basis and for a relatively longer period of time than interventions for DADs in primary care, which tend to be much briefer, less structured, and include fewer sessions (Archer et al., 2012; Cape, Whittington, Buszewicz, Wallace, & Underwood, 2010).

Despite the substantial number of studies identifying the extent of DADs in primary care patients published in recent decades, reviews of specific interventions for primary care patients with DADs are lacking. Other significant uncertainties remain concerning the effectiveness (Archer et al., 2012) and generalizability of existing empirically supported interventions (ESIs) for patients with DADs in various health care contexts such as primary care. The effectiveness of interventions for primary care patients with DADs may also vary by patient population, and models of intervention delivery in primary care settings also vary across published studies. As a result, it is important to update the understanding of interventions that have been delivered in primary care settings for patients with DADs. Examining the effectiveness of interventions in primary care is different from examining empirically supported interventions for DADs in specialty mental health outpatient clinics for reasons mentioned previously. This study will fill a gap in research by developing a better understanding of primary care-based DADs interventions and will answer the following questions to facilitate future research conversations: 1. Are any of the primary care

interventions for DADs effective? 2. If so, are identifiable characteristics of these interventions associated with higher levels of effectiveness? and 3. If other factors, like different populations, health conditions, treatment length, providers' clinical experience, moderate treatment effect size? This study uses systematic review and meta-analysis approaches to answer these questions.

THE RATIONALE FOR USING SYSTEMATIC REVIEW AND META-ANALYSIS

Systematic review

A systematic review is one that has been prepared using a systematic approach to minimizing biases and random errors and which is documented in a materials and methods analysis (Chalmers & Haynes, 1994). It offers researchers a principled way to synthesize empirical research in order to make generalizations (and recognize the limits of generalizations) (Cooper et al., 2009). Systematic reviews have gained wide popularity in evaluating the effectiveness of clinical interventions and practices for patients' health and mental health wellbeing–an area that needs comprehensive and unbiased research evidence.

Given that the literature on integrated psychosocial interventions for primary care depression and anxiety has increased exponentially over the past decade (Ballenger et al., 2001; Hirschfeld, 2001; Lemieux-Charles & McGuire, 2006; Roca et al., 2009), a new systematic review of the empirical literature is needed. While there are many promising interventions for treating depression and anxiety in primary care, most empirical studies are at preliminary stages and are far from conclusive. The systematic review methodologies can be useful for literature deconstruction (Gasteen, 2010), especially for fields that are evolving or when there are competing views of the effectiveness of

interventions being delivered in a new setting. In conjunction with quantitative meta-analysis, this systematic review may provide implications and guidance for both health care practice and policy development.

Meta-analysis

Meta-analysis is a statistical procedure for systematically combining pertinent quantitative study data from several individual scientific studies to develop a single conclusion that has greater statistical power (Cooper et al., 2009). Meta-analysis assumes a "common truth" behind all conceptually similar primary studies even though each study contains certain errors (e.g., sampling error) (Rothman, Greenland, & Associate, 2014). To estimate the "common truth," meta-analysis offers a quantitative approach to derive a pooled estimate that is closest to this unknown "common truth" while considering the errors within each primary study.

Meta-analysis fits particularly well with evaluating interventions' clinical effectiveness. Many primary studies on intervention effectiveness report only the difference between treatment and control groups and whether the difference is statistically significant. Statistical significance is often influenced by a primary study's sample size and offers limited information about an intervention's practical significance (effect size) (Kirk, 1996; Peeters, 2016), which can be obtained using meta-analysis.

In addition, meta-analysis also enables investigation into the heterogeneity among effect size estimates from different primary studies. In other words, it examines the sources of variance between effect size estimates beyond sampling errors. For example, meta-analysis uses moderator analysis (Borenstein, Hedges, Higgins, & Rothstein, 2009b) to understand if there are significant relationships between an intervention's length of treatment and its clinical effectiveness, or if providers' years of experience are

associated with greater treatment effects. Most importantly, in the context of integrated primary care interventions for comorbid depression and anxiety, a systematic review and meta-analysis allows for investigating two important questions: (1) Are the interventions included effective based on evidence reported in existing empirical studies? and (2) Are there factors across existing empirical studies that are strongly associated with greater clinical effectiveness?

Study purposes

Given (1) the high prevalence of DADs in primary care settings and in the general U.S. population, and (2) that interventions for treating depression and anxiety in primary care settings are still evolving and under on-going investigation, this study aims to systematically evaluate the current state of the empirical literature on interventions for primary care patients' depressive and anxiety disorders. In addition, using systematic review and meta-analysis procedures, this study aims to obtain a quantitative estimate of treatment effectiveness across primary studies that use Randomized Clinical Trial (RCT) designs to examine outcomes in depressive and anxiety disorders for primary care patients. Moderator analysis will also be conducted to investigate sources of possible heterogeneity among effect size estimates to determine the degree to which any particular intervention, design, provider type, and/or client characteristics may be associated with treatment effects.

DEFINITION OF KEY TERMS AND CONCEPTS

Primary studies: In a meta-analysis, the study sample is composed of individually published studies. To differentiate a meta-analysis from the individually published studies that compose the sample used in the meta-analysis, each individually

published studies is commonly referred to as "a primary study." Because this study is a meta-analysis of interventions used in primary care settings, it is important to clarify that a published study that was conducted in a primary care setting will be referred to as a "study in primary care settings," while an individually published study included in the meta-analysis will be referred to as a "primary study." Thus, an individual study that was conducted in a primary care setting would be referred to in this dissertation as a "primary study in primary care settings."

Mental health specialty outpatient setting: In this review, traditional mental health outpatient settings are referred to as "mental health specialty outpatient settings." Examples of these settings include but are not limited to community based mental health agencies and mental health or behavioral health departments affiliated with a university teaching hospital or a private hospital. In these settings, the primary focus of services is on patients or clients' mental wellbeing and physical health services may or may not also be provided.

Primary care based intervention: In this review, primary care based interventions explicitly refer to four types of mental health interventions: (1) In-person face-to-face interventions delivered in a primary care setting, typically by one or more members of the primary healthcare team. (2) Tele-health based interventions delivered in a primary care setting typically through platforms that utilize technologies such as computer-assisted cognitive behavioral therapy (CBT) or internet-based problem solving therapy. They may be pre-programed or delivered by a clinician in real time who is not physically present in the primary care setting. (3) In-person, face-to-face interventions prescribed by a primary care physician or other healthcare professional on the team but delivered outside the primary care setting by a mobile therapist or social work case manager. (4) Tele-health based interventions delivered outside the primary care setting,

for example, in a client's home or residential communities, typically utilizing technologies such as computer-based CBT.

CHAPTER TWO: CONCEPTUAL AND THEORETICAL FRAMEWORKS

Two sets of conceptual and theoretical frameworks guide this project: (1) Theories underlying depressive and/or anxiety disorders (DADs) and DADs' presentation in primary care; and (2) Frameworks for interventions (to be included in this review) for addressing DADs in primary care settings.

THEORIES UNDERLYING DADS

The puzzle of the relation between depressive and anxiety disorders is as old as the investigation of the syndromes themselves. While it has always been clear that depressive and anxiety disorders are conceptually and clinically distinct groups of disorders, both research and practice communities constantly struggle with clarifying the relation between the two types of disorders (Clark, 1989). Such struggles originate from two key taxonomic problems: comorbidity (an empirical overlap between constructs that are hypothesized to be distinct) and heterogeneity (when phenomena that ordinarily are collapsed together are found to be sufficiently distinctive for separation) (Watson, 2005). As a result, in addition to theories underlying depressive and anxiety disorders separately, this section also presents theories that elucidate the relation between depressive and anxiety disorders, including Clark and Watson's (1991) tripartite model and Mineka, Watson, and Clark's (1998) integrative hierarchical model.

Theories underlying depressive disorders

The term depression is derived from the Latin verb *deprimere*. Since the mid-19th century, the term has been used to refer to a psychiatric symptom and appeared in medical dictionaries for describing psychological status in the 1860s (Berrios, 1988). While there are many theories about the causes of depression, they can be broadly

grouped into two competing yet complementary views of depressive disorders' etiology: primarily biological or primarily psychological (Schwartz & Schwartz, 1993). The two views also reflect a widely agreed on nosology of endogenous and exogenous depressive disorders (Mendels & Cochrane, 1968). Chronologically, it is difficult to identify whether psychological theories of depression appeared before biological ones, or vice versa. It seems obvious that psychological theories were the primary theories in the early stages of understanding depression. However, with the development of modern bio-neuro-chemical technologies, biological theories have gained attention more recently (Schwartz & Schwartz, 1993).

The roots of the present psychological approaches to depression are found in the works of Karl Abraham and Sigmund Freud (Ebtinger, 1989; Freud, 1922; May, 2001). According to Freud, in melancholia – now a type of major depressive episode – a person has suffered a loss, real or imaginary, that may not even be identified consciously (Freud, 1922). As reflected in Freud's early psychoanalytical theories of depression, while the direct cause of melancholia is psychologically endogenous (derived from within), it is a result of reacting to external events, thus, essentially exogenous.

Extending Freud's psychoanalytic view of depression, Adolph Meyer was one of the very first psychiatrists to emphasize the influence of social and interpersonal (environmental), as well as biological factors in mental health, including depression (Lewis, 1934). Continuing with Freud's exogenous view of depression etiology and its connection to an individual's internal system, Meyer proposed a psychobiology framework of mental health (including depression) that views depression as being reactive to external stressful events. These reactions differ from each other because of the different life experiences that shape each individual's personality, vulnerability, and the genesis of psychiatric disorders (Rutter, 1986).

In the mid-20th century, other theorists extended Freud's and Meyer's work in understanding depression. While continuing the binary view (endogenous versus exogenous) of depression's etiology, most psychological theories focused on exogenous mechanisms, that is, how an individual responds to external stressful events that cause depression. Existential and humanistic theorists conceptualize depression as a result of an existential vacuum (Blair, 2004), the inability to construct a future (Schneider, Galvin, & Serlin, 2009), or when the world precludes a sense of 'richness' (Maslow, 1969).

Behavioral theorists Martin Seligman and Joseph Wolpe conceptualize depression using the framework of Pavlov's conditioning experiments. Seligman's theory of learned helplessness argues that, when faced with stressful events, those who had some success in easing the pain or distress seem to keep on trying and do not become depressed. The others, however, would develop learned helplessness and start to lose hope, resulting in depression (Peterson & Seligman, 1984). Wolpe's behavioral theories (1973) categorize depression into situational, biological, and neurotic depressions (Schwartz & Schwartz, 1993). Situational depressions are universal, i.e., when faced with certain situation, like deprivation or failure, it is "normal to be depressed" (Wolpe, 1973). Biological depressions have a variety of causes and may or may not be triggered by reactions to external events. Wolf argued that these depressions are best treated with biological approaches. Neurotic depressions, also called reactive depressions, are often reactive and triggered by external events. Many neurotic depressions, according to Wolf, result from a conditioning process and can be effectively treated with behavioral therapies.

Cognitive theorists Albert Ellis and Aaron Beck argued that depression is a result of how people think (cognition) (Beck, 1967; Ellis, 1987). Ellis argued that depression stemmed from individuals' irrational "should" and/or "must" thoughts, which leads to inappropriate self-blame, low self-esteem, and other depressive symptoms. Beck's

original theory argued that depression stemmed from a "cognitive triad" of negative schemas about oneself, his/her future, and the world (Beck, Rush, Shaw, & Emery, 1979). Later, Beck integrated research findings from biology, neurology, medicine and other fields to modify his theories of depression. Beck's most recent update on his cognitive model of depression is termed the "Generic Cognitive Model" (Beck & Haigh, 2014). The generic cognitive model conceptualizes depression from an information processing perspective. It argues that different people process similar external stimuli (stressful event) differently and their focus during this information processing procedure also differs. Maladaptive thoughts and feelings (depression) happen when an individual automatically (with or without knowing) focuses on negative aspects of an event and overlooks the positive aspects. In his latest update, Beck articulated a model that integrates genetic and neurodevelopmental influences of an individual's scheme, which reflects a long history of biological theories in understanding depressive disorders.

Biological theories cross a wide spectrum of areas including genetics, neurology, hormonology, immunology, and neuroendocrinology. Given the complex nature of the biological system itself, biological theories of depression are often intertwined. Genetic theories draw heavily from findings from family, twin, and adoption studies and indicate that there is generic vulnerability for depression. For example, a review of twin studies finds that about one-third of the risk of major depression in adults derives from genetic differences between individuals (Kendler, Gatz, Gardner, & Pedersen, 2006), indicating the strong influence of genetics and heredity. Within the genetic theories of depression, several genetic polymorphisms have been linked to an increased risk of depression: the gene of the serotonin transporter, the serotonin concentration, and other serotonin system genes that are known to affect hypothalamic-pituitary-adrenal (HPA) axis functioning (Neumeister, Young, & Stastny, 2004). For example, Kendler and colleagues (2005)

found that individuals with one or two copies of the short allele of the serotonin transporter gene experienced greater depressive symptoms than individuals who are homozygous for the long allele. It should be noted that genetic factors not only impact internal depressogenic processes, they also have implications for the gene-environment correlation as related to depression. In other words, an individual's genetic composition not only indicates an individual's genetic vulnerability for developing depression, it also implicates an individual's response tendency when facing stressful events (Rice, Harold, & Thapar, 2005).

In addition to genetic theories, the theory that initially attracted attention in understanding the neurobiology of depression is the biogenic amine hypothesis of depression (Hirschfeld, 2000), also known as the monoamine hypothesis of depression. The biogenic amine theory stipulates that depression results from a deficiency of norepinephrine (NE) at a number of synapses in the brain. In other words, when depressed patients are stimulated, their neurotransmission process does not have enough chemical material (NE) to supply this process, resulting in neurological chemical imbalance, which further increases depression. Despite its many limitations (e.g. inability to explain the psychosocial triggers of depression), the amine hypothesis of depression offers significant implications for research on bio-neurological understandings of depression and the development of antidepressant drugs (Schwartz & Schwartz, 1993). With scientific research technologies becoming more and more sophisticated, researchers have begun to reach the consensus that while neurotransmitters are important regulators of mood, it is unlikely they could account for all the behavioral changes that are symptomatic of depression. This calls for other theories to further explain the bio-neurological theories of depression, such as neuroendocrine theories.

Neuroendocrine emerges as a dominant model of the neurobiology of depression that emphasizes the underlying dysregulation of the body's response to stress (Thase, 2009). Key components of this theory are the hypothalamic-pituitary-adrenal (HPA) axis and the related corticotrophin-releasing hormone (CRH), and locus coeruleus-norepinephrine (LC-NE) systems, which include limbic and cortical pathways bidirectionally interconnected through various neurotransmitter and hormonal circuits (Boyce & Ellis, 2005; Meyer, Chrousos, & Gold, 2001). When facing stressful events, the HPA axis produces higher levels of cortisol that trigger a cascade of functions to adapt to stressful events. Dysfunction happens if, after the stressful event, the inhibitory feedback processes in the HPA axis fail to normalize the cortisol, resulting in sustained high cortisol. This would then give rise to physiological changes that are connected with illness promotion, including depression. Neuroendocrine theory not only highlights genetic and environmental heterogeneity in understanding depression, it also provides a new perspective for future neurodevelopmental theories of depression like the neurogenesis theory of depression, which posits that impaired adult hippocampal neurogenesis (AHN) triggers depression and restoration of AHN leads to its recovery (Jacobs, van Praag, & Gage, 2000; Miller & Hen, 2016).

While other biological theories also attempt to explain the etiology of depression, like Wurtman's precursor theory which articulates depression is a result of insufficient neurotransmitter precursors in the human brain (Wurtman, Hefti, & Melamed, 1980), the genetic vulnerability and the neuroendocrine theories are the ones with the most empirical support (Hasler, 2010). However, whether a theory takes primarily a psychological or biological perspective, the field of psychiatry and mental health has reached a consensus on an integrative theoretical model in understanding depression. While acknowledging depressed patients' genetic vulnerability, studies consistently show

that the influence of genetic factors is around 30% to 40%, and 60% to 70% of the influence is posed by non-genetic factors (including gene-environment interaction) (Hasler, 2010). Therefore, it is important to develop thorough understanding of the psychological perspectives of depression as well as the interaction between psychological and biological aspects of depression.

Theories underlying anxiety disorders

Any attempts to theorize about the etiology of anxiety involves, in some way, the concepts of worry and fear in relation to a disturbance of brain function (Simpson, Neria, Lewis-Fernández, & Schneier, 2010). Therefore, even in some early psychological theories of anxiety, there were links between psychological and cognitive aspects of anxiety disorders (Behar, DiMarco, Hekler, Mohlman, & Staples, 2009). As a result, many popular psychological theories of anxiety are cognitive based. Among various theories underlying anxiety disorders, this paper reviews the Avoidance Model of Worry (Borkovec, Alcaine, & Behar, 2004), the Intolerance of Uncertainty Model (Robichaud, 2013), and the Meta-Cognitive Model (Wells, 1995).

The Avoidance Model of Worry (AMW) derives from behaviorists' two-stage theory of anxiety (Mowrer, 1956), which theorizes that classically conditioned acquisition of fear is followed by operantly conditioned avoidance of fear cues, resulting in fear maintenance due to a lack of exposure to those conditioned stimuli. Extending this behavioral theory, AMW posits that, at the cognitive-perceptual level, avoidance of (cognitive escape from) negative emotional experience and arousal is the core concept that contributes to the original development of anxious responding (Borkovec, 1979). AMW further articulates that worry is a central means to achieve cognitive avoidance; thus, this central component presents across anxiety disorders (Borkovec, 1994).

Therefore, addressing worry as a form of cognitive avoidance has been the focus of many psychosocial interventions targeting anxiety disorders, like cognitive behavioral therapy, exposure therapy, solution focused brief therapy, and others.

In addition to positioning worry at the center of anxiety disorders, AMW has also inspired more recent research on the causal role of worry itself in creating negative emotionality (Brosschot, 2010; Brosschot, Gerin, & Thayer, 2006; Stapinski, Abbott, & Rapee, 2010). This line of theoretical and empirical research has resulted in the appearance of the Contrast Avoidance hypothesis. While AMW articulates the functional role of worry in relation to anxiety, it fails to resolve the conflict between the purpose of worry—to avoid negative emotion—and the consequence of worry—negative emotion. The Contrast Avoidance hypothesis claims that individuals with anxiety disorders have developed a stronger aversive reaction to emotional contrast than non-anxious individuals. Emotional contrast refers to the change from positive to negative emotions. If an individual is constantly worried, he/she will experience lower level of emotional contrast because the baseline threshold is low. It is the avoidance of the emotional contrast, not necessarily the negative emotion itself, which motivates individuals to worry (Llera & Newman, 2014; Newman & Llera, 2011). In other words, anxious individuals put themselves under chronically negative emotion (associated with worry) to avoid being vulnerable to emotional contrast, which may entail a drastic increase in negative affect or distress. Whether to avoid experience negative emotions or emotional contrasts, both the AMW and contrast avoidance agree on worry's role of control and its certainty in anxiety disorders.

Another theory, the Intolerance of Uncertainty Model (IUM) (Koerner & Dugas, 2006) elaborates on this perspective. The IUM defines intolerance of uncertainty as "a dispositional characteristic that results from a set of negative beliefs about uncertainty

and its implications and involves the tendency to react negatively on an emotional, cognitive, and behavioral level to uncertain situations and events" (Buhr & Dugas, 2009 p. 216). Individuals with anxiety disorders find uncertain situations stressful and upsetting, and, in response, they experience worry. According to IUM, individuals with anxiety disorders believe that worry would either help them cope with feared incidents more effectively, or to prevent them from happening (Ladouceur, Gosselin, & Dugas, 2000). As the anxiety disorder further develops, both worry, and its accompanying anxious mood, further lead to negative problem orientation and stronger cognitions that maintain or sometimes worsen the anxiety.

Extending the understanding of worry in AMW, the Contrast Hypothesis, and the IUM, the Meta-Cognitive Model (MCM) proposed by Wells (Wells, 1995) posits that individuals with anxiety disorders experience two types of worry: worry about non-cognitive events (Type 1) and worry about worry (meta-worry, Type 2). Type 1 worry happens when individuals are initially faced with an anxiety-provoking incident. Type 2 worry happens when individuals, in addition to Type 1 worry, begin to worry about or fear that their Type 1 is becoming uncontrollable. MCM argues that it is the resultant Type 2 worry that distinguishes individuals with GAD from nonclinical worriers (Behar et al., 2009). When an individual's behavioral or cognitive strategies are ineffective in controlling anxious feelings or avoiding them, Type 2 worry starts to arise. Consequently, MCM offers clear guidance on intervening in anxiety disorders, such as cultivating effective cognitive strategies and behavioral activations among others.

The three psychological theories mentioned above along with other psychological theories put worry and cognition at the center of understanding anxiety disorders. While not explicitly indicated, most theories indicated the importance of at least complementing

psychological theories with biological theories if not fully incorporating them in understanding anxiety disorders.

Biological theories underlying anxiety disorders cut cross genetics, neurology, and the serotonergic system. Genetic research and theories of anxiety heavily draw from family and twin studies and molecular genetic study designs. Given the wide range of anxiety spectrum disorders, different genetic factors have been identified for specific disorders (Simpson et al., 2010). For panic disorders, multiple family studies and one meta-analytic report indicated familial aggregation of panic disorders with risk to first-degree relatives of probands ranging from 8% to 17% (Hettema, Neale, & Kendler, 2001; Horwath et al., 1995). High levels of heritability (estimated 30% to 40%) have also been reported and are supported by multiple twin studies (Bellodi et al., 1998; Kendler, Gardner, & Prescott, 2001). However, findings from candidate gene studies (genetic association studies focused on associations between genetic variation within pre-specified genes of interest and phenotypes or disease states) are inconsistent in identifying genes associated with panic disorder (Simpson et al., 2010), while anxiety studies of genes in the serotonergic system seem to reach preliminary consensus that the monoamine oxidase A (MAOA) gene, which codes for an enzyme that degrades amine such as serotonin, has been associated with panic disorder (Maron et al., 2005; Samochowiec et al., 2004).

For generalized anxiety disorders (GAD), family studies have reported heterogenous findings with greater risk for GAD in first-degree relatives of GAD probands ranging from 1.4% to 20% (Doherty & Owen, 2014; Newman & Bland, 2006). A meta-analytic twin study reported GAD's heritability to be 32% (Hettema et al., 2001). Molecular genetic studies have reported significant associations between GAD and the SLC6A4 gene (a serotonin transporter), and between GAD and monoamine oxidase (MAOA) (Samochowiec et al., 2004; You, Hu, Chen, & Zhang, 2005).

Another well studied anxiety spectrum disorder is obsessive compulsive disorder (OCD). Numerous family studies reported an approximate 9% to 25% OCD prevalence in first-degree relatives (Chacon et al., 2007; Do Rosario-Campos et al., 2005). Heritability estimates reported in twin studies range from 45% to 65% in children and from 39% to 50% in adults (van Grootheest, Cath, Beekman, & Boomsma, 2005). The serotonergic hypothesis of OCD argues that OCD is a result of dysregulation in serotonin, and, though not yet fully elucidated, evidence accumulates that 5-hydroxytryptamine receptors (serotonin receptors), in part, modulate OCD symptoms (Barr, Goodman, Price, McDougle, & Charney, 1992).

Compared to the anxiety disorders mentioned above, biological research and theories on posttraumatic stress disorder (PTSD) and on specific phobias are rather limited (Simpson et al., 2010), resulting in little agreement on possible theories explaining these two disorders. However, by looking for commonalities in biological research and theories across anxiety spectrum disorders, Hetteman and colleagues (2005) found two genetic factors to be common across anxiety disorders. The first factor indicated highest loadings on GAD, panic disorder, agoraphobia, and social anxiety disorder, whereas specific phobias were found to load most highly on a second genetic factor. Additionally, evidence of shared genetic liability among anxiety disorders has encouraged researchers to examine intermediary phenotypes considered to represent underlying vulnerability to multiple anxiety disorders including neuroticism, behavioral inhibition, and anxiety sensitivity (Simpson et al., 2010). As a result, even from a genetic perspective, psychosocial and environmental factors are believed to influence the etiology of anxiety spectrum disorders, highlighting the importance of an integrative framework for understanding and treating anxiety disorders.

Neurobiological theories view normal anxiety as an emotional state subserved by neuronal circuits (amygdala and the prefrontal cortex), while pathological anxiety may be viewed as maladaptive responsiveness of the same circuitry (Oathes, Patenaude, Schatzberg, & Etkin, 2015). A core component of anxiety is fear, and the most widely researched behavioral model of fear is "classical fear conditioning" (Lissek et al., 2005). Within this circuitry process, the amygdala (corpus amygdaloideum), buried deep inside the temporal lobe, is the key neural system subserving fear conditioning (Kim & Jung, 2006). It receives sensory inputs from diverse areas of the brain and then sends projections to various autonomic and somatomotor structures which are considered to mediate specific fear responses, such as the bed nucleus of stria terminalis for activating stress hormones or the lateral hypothalamus for sympathetic activation (LeDoux, 2003). The basolateral nucleus of the amygdala (BLA) plays a key role as the input area for environmental cues, and then sends projections to additional nuclei of the amygdala. Anxiety happens when BLA receives "normal" environmental cues but projects negatively to the rest of the amygdala neuclei (Bishop, Duncan, Brett, & Lawrence, 2004). Pathways through which BLA sends projections to other neuclei are the hippocampus and the cortex, and the latter has received a great deal of attention for its role in controlling anxiety.

The frontal cortices, especially the prefrontal cortex (PFC), resolve the conflict between "important" stimuli and those irrelevant to a task when complex stimuli arrive simultaneously (Egner, 2008). Decreased activation of PFC and hyperactivation of the amygdala will result in dysfunctional reactions and feelings toward normal external stimuli, manifesting potential symptoms of anxiety disorders. There is ample empirical support for this finding (Mathew, Price, & Charney, 2008; Milad et al., 2007; Quirk, Likhtik, Pelletier, & Paré, 2003), highlighting the clinical relevance and potential

theoretical contribution of studying the prefrontal cortex in understanding anxiety disorders.

Another important biological theory of anxiety involves the serotonergic system. Numerous neurotransmitter systems, such as glutamate, gamma-aminobutyric acid (GABA) noradrenaline, serotonin (5-HT), and numerous neuropeptides, have been found relevant in regulating anxiety responses (Millan, 2003). Out of different neurotransmitter systems, serotonin (5-HT) plays one of the most critical roles in regulating emotions, including anxiety, depression, and fear (Canli & Lesch, 2007). Serotonin exerts its effects through a complex system that includes multiple transporters and a wealth of receptors. Of the 14 different serotonergic receptors, several receptors, including the 5-HT1A, 5-HT1B, 5-HT2A, 5-HT2C, 5-HT4, and the serotonin transporter have been implicated in the regulation of anxiety states (Holmes, 2008).

Serotonin transporters (SERT) play an important role in determining the magnitude and duration of the serotonin response during a stressful situation. Imaging studies have connected SERT genotype with amygdalar activation in response to the presentation of fearful faces (Hariri et al., 2002). Other studies have also shown that SERT has a role at the circuit level. Carriers of the short allele (of SERT) show uncoupling of a cingulate-amygdala feedback circuit, which suggests that activity level of SERT has a role in developing essential circuits underlying anxiety responses (Pezawas et al., 2005).

From both biological and psychosocial perspectives, depression and anxiety are distinct disorders with overlapping etiologies. This highlights the importance of understanding comorbid depression and anxiety. Given the nature and focus of this dissertation, the following paragraphs elaborate on psychosocial theories explaining the

comorbidity between depressive and anxiety disorders, including the Tripartite Model, the Integrative Hierarchical Model, and the Bi-factor Model.

Theories underlying anxiety disorders

Tripartite Model: The tripartite model (Clark & Watson, 1991), illustrated in Figure 1, specifies that depressive and anxiety syndromes share a significant nonspecific component of generalized affective distress – negative affect (NA). This significant overlap in NA explains the high prevalence of comorbidity between depressive and anxiety disorders. Though NA is nonspecific, the model also indicates that it manifests differently in the presence of depressive or anxiety disorders. In depression, negative affect is marked by anhedonia or sadness, while in anxiety, negative affect is marked by anxious/somatic arousal associated with worry. Therefore, the tripartite model stipulates the use of two or more constructs to explain the relationship between anxiety and depression, both at the mood and syndrome level: a nonspecific negative affect factor at the mood level [to identify depression and/or anxiety] and a specific manifestation factor at the syndrome level that distinguishes them. Since the tripartite model was proposed, it has received substantial support in both conceptual and empirical literature (Cook, Orvaschel, Simco, Hersen, & Joiner, 2004; Simms, Grös, Watson, & O'Hara, 2008), which has resulted in more complex models being built on it. One representation of such models is Mineka, Watson, and Clark's (1998) integrative hierarchical model.

Integrative Hierarchical Model: The integrative hierarchical model is an integration of the tripartite model and the hierarchical model (Mineka et al., 1998). The hierarchical model, illustrated in Figure 2, separates components defining depressive and anxiety disorders into higher and lower order factors. Consistent with the tripartite model, higher order factors in the hierarchical model refer to the "absence of positive affects,"

such as “anxious apprehension” versus “sadness,” that differentiate depression and anxiety spectrum disorders from each other. In addition, the higher order factors contain six specific components that are used to differentiate the heterogeneous clinical manifestations within each disorder (depression and anxiety) (Brown, Chorpita, & Barlow, 1998; Simms et al., 2012).

An integration of the tripartite model and the hierarchical model forms the basis of the integrative hierarchical model of DADs (Figure 3). Depressive and anxiety disorders include common negative affect [tripartite model] that can be used to identify depressive and/or anxiety spectrum disorders. Higher order factors can then be used to differentiate [hierarchical model] depressive and anxiety spectrum disorders. These two components speak to the comorbidity between depressive and anxiety disorders while allowing for contrasts between the two disorders based on emotional factors. At the syndrome level [tripartite model], both disorder specific factors [tripartite model] and lower order factors [hierarchical model] distinguish heterogeneous manifestation of depressive or anxiety disorders.

Commonalities among DADs theories

The quantity and quality of theories underlying DADs has evolved substantially (Mineka, Watson, & Clark, 1998; Simms, Grös, Watson, & O’Hara, 2008; Watson, 2005); yet, one essential component, negative emotional distress (negative affect), which is often shared across depressive and anxiety disorders, has remained constant. Negative affect is a broad concept that can be summarized as feelings of emotional distress (Watson, Clark, & Tellegen, 1988). Negative affect and the dispositional tendency toward negative affect (called neuroticism, negative affectivity, or negative emotionality) are a large component of many forms of psychopathology including depressive and

anxiety disorders (Stringer, 2013). This has informed theoretical frameworks in explaining depressive and anxiety disorders, which consequently offer implications for treating DADs, i.e., to address the underlying negative affect.

THEORIES UNDERLYING INTERVENTIONS

The central role of targeting negative affect in treating DADs guided the selection of interventions included in this review, especially a strong theoretical foundation related to alleviating negative affect. To be included in this review, an intervention should be based on theoretical literature that points to the possible change mechanism that explains the effect of the intervention for treating DADs. While an intervention's theoretical foundation was the primary guiding principle, other factors also informed intervention selection including: (1) treatment brevity, so that it fits the pace of interventions in primary care (feasibility); (2) sufficient empirical support for effectively addressing DADs in other settings; and (3) preliminary empirical evidence on an intervention's effectiveness for in primary care settings. The following interventions meet all criteria mentioned: (1) Cognitive behavioral therapy (CBT), (2) Problem-solving therapy (PST), (3) Solution-focused brief therapy (SFBT), and (4) Motivational Interviewing (MI). In the meanwhile, considering the scope and resources available for this dissertation, only four interventions are included in this dissertation while there are other psychosocial interventions primarily target negative affect in DADs that are brief and feasible in primary care settings with preliminary empirical evidence supporting their effectiveness.

Cognitive behavioral therapy (CBT)

Clearly defining CBT is challenging because it is a broad concept and umbrella term used to refer to more than one psychotherapeutic approach [that share some

common features]. Aaron Beck developed a form of psychotherapy in the early 1960s that he originally termed "cognitive therapy" – a term that is now used synonymously with "cognitive-behavioral therapy" (CBT). However, Beck's model and cognitive-behavioral theory are also the basis for many other approaches in various stages of development. As Freeman et al. (2004) once said:

> "There was a time, not too long ago, when the term cognitive-behavioral therapy was considered an oxymoron … Only a quarter century ago, it was inconceivable to many that there could be anything legitimately called "mind sciences." Now it is difficult to imagine an adequate approach to psychotherapy that does not appreciate basic contributions from the cognitive sciences. (p. 5)"

This review adopts Dr. Judith Beck's (2011) definition that CBT refers to "a number of forms of cognitive behavior therapy that share characteristics of Beck's therapy, but whose conceptualization and emphases in treatment vary to some degree" (p. 2). Among those therapies are rational emotional behavior therapy (Ellis, 1962), dialectical behavior therapy (Linehan, 1993), problem-solving therapy (D'Zurilla & Nezu, 2006), acceptance and commitment therapy (Hayes, Follette, & Linehan, 2004), exposure therapy (Foa & Rothbaum, 1998), cognitive processing therapy (Resick & Schnicke, 1993), cognitive behavioral analysis system of psychotherapy (McCullough, 2003), behavioral activation (Martell, Addis, & Jacobson, 2001), cognitive behavior modification (Meichenbaum, 1977), and others. An understanding of Cognitive Behavioral Therapy requires discussing its historical and theoretical context because Cognitive Behavioral Therapy evolves as Cognitive Behavioral Theories change.

Theories underlying CBT

Cognitive-behavioral theory reflects ongoing evolutions in theorizing, clinical application, and empirical evidence. Cognitive-behavioral theory is based on the premises that thoughts, emotions, and behaviors are inextricably linked and that each of these aspects continuously impacts and influences the others (González-prendes & Resko, 2012). Specifically, cognitive-behavioral theory posits that thoughts about the self, relationships, the world, and the future shape emotions and behaviors (Beck, 2002). In turn, feelings/emotions and behaviors shape thoughts and thought processes in an ongoing reciprocal feedback loop.

Evolving from its early roots in behavior theory/therapy, one important facet of cognitive-behavioral theory is assuming the fundamental difference between "cognitive activity" and "behavior" (Watson, 1930). Watson and succeeding behavioral theorists, notably B. F. Skinner and Albert Bandura, shaped behavioral theory through extensive basic research and provocative theorizing about the implications of operant conditioning (Skinner, 1953), external stimuli (Bandura, 1986; Chomsky, 1959), and social (or vicarious) learning (Bandura, 1977a). The switch from pure behaviorism to valuing both behavior and cognition and their relationships to the environment was obvious even at the beginning stage of cognitive-behavioral theory development.

One of the most influential "cognitive behavioral" theories for depression and anxiety is Bandura's (1977a) self-efficacy theory of behavioral change. Though initially not called a cognitive behavioral theory, the connection between cognition and behavior was evident. The theory argues that psychological influences alter defensive behavior (to cope with depression and anxiety) by enhancing an individual's self-efficacy (Schwarzer, 2014). It is hypothesized that an individual's perception of personal efficacy will determine the initiation of coping behavior as well as the extent of effort to sustain these

behaviors. This theory establishes the reciprocal connection between an individual's perception/cognition and behavior, both of which are believed to reduce symptoms of depression and anxiety (Maddux & Gosselin, 2003; Muris, 2002). The influence of the self-efficacy theory of behavioral change remains evident in CBT, and also in problem solving therapy (Eskin, 2013; Nezu & Nezu, 2001) and motivational interviewing (Miller & Rose, 2009; Rollnick, Miller, & Butler, 2008).

Following Bandura's working on establishing the connection between cognition and behavior, Ellis and Beck are among the most influential cognitive behavioral theorists. Albert Ellis's rational-emotive behavior theory integrated behavioral, cognitive, and emotional components in one treatment model (Ellis, 2004), shifting the sole focus from behavior to other components (emotions and cognitive processes) in the fields of psychopathology and psychological intervention. Independent of Ellis, Beck (1963) was among the first researchers to offer a detailed account of the role of cognition in his cognitive therapy for psychopathology. Beck adhered to beliefs about the interactions among cognition, behavior, and emotion, but shifted the primary focus from behaviors to beliefs (cognition) (Beck, 1987). While Ellis started the shift from radical behaviorism to both behavior and cognition, Beck was regarded as a more influential theorist for advancing cognitive-behavioral theory/therapy, thus forming the second wave/generation of CBT.

Beck's cognitive theory and related advancements are most relevant for cognitive therapy related to depression and anxiety (Beck, 2005, 2008) and lead to the development of a General Cognitive model for emotional disorders. Though Beck originally focused on theorizing about depression, he later developed a general cognitive model of emotional disorders (or cognitive formulation of psychopathology) that addresses depressive, anxiety, and other emotional disorders (Beck & Haigh, 2014).

Briefly, Beck's cognitive model of emotional disorders proposes that three levels of cognition are responsible for the persistence of anxiety and depression: (1) schema: those enduring structural representations of human experience that direct the identification, interpretation, categorization, and evaluation of experience; (2) information-processing: a process in which external stimuli connect with the individual's inner world; and (3) automatic thoughts: a response to stimuli after information-processing (see Figure 4).

According to the cognitive model, schema or schematic contents that are negative in nature result in biased information processing, which in depression, involves preferential encoding and retrieval of negative self-referential information; whereas in anxiety, a selective processing of threat, danger, and helplessness is evident (Clark, Beck, 2010). Consequently, the culmination of biased information processing would result in the subjective experience of schema-congruent negative 'automatic' thoughts, images, and memories that perpetuate a subjectively adverse emotional state, including depression and anxiety.

Theoretically, tackling depressive and anxiety disorders requires identification of the client's underlying schema and relevant automatic thoughts in relation to the unique way s/he processes information. Then, various techniques can be used to "challenge" the client's schema and automatic negative thoughts. Once the client's automatic negative thoughts and/or irrational schema are corrected, the theory suggests that the negative affect those thoughts and/or schemas perpetuate will decline and eventually disappear. Thus, the cognitive-emotional-behavioral triangle switches from a negative loop to a positive one with more neutral or positive thoughts about life experiences that lead to lower adverse emotions, which promote positive, active behavioral performance that

further improves one's negative schema or automatic thoughts. This is how CBT addresses depressive and/or anxiety disorders.

Problem solving therapy (PST)

In addition to cognitive-behavioral therapy, (social) problem solving therapy (PST) is a modified version of CBT for treating depressive and/or anxiety disorders within a shorter time period. Like CBT, PST is non-pharmacological. It can be defined as a social competence-based clinical intervention approach in which clients are taught a step-by-step approach to constructive problem solving to maximize effective solutions to daily problems (D'Zurilla & Nezu, 2006). In addition to its behavioral activation components, PST is widely known as a social problem solving therapy to emphasize its focus on contextual factors and an individual's problem-solving orientation (Bell & D'Zurilla, 2009). According to D'Zurilla and Nezu (2006), it is important to understand that "the adjective social in the term social problem solving is not meant to limit the study of problem solving to any particular type of problem; rather, it is used only to highlight the fact that the focus of study is on problem solving that occurs within the natural social environment" (p. 12). Originally developed in the late 1960s and early 1970s, both cognitive-behavioral theories and Richard Lazarus's relational model of stress largely influenced PST (Harris, 2001; Lazarus, 1966). PST continues to be refined.

Theories underlying problem-solving therapy

Strongly influenced of by the general cognitive-behavioral model (Beck, 2002; A. Ellis, 1985), PST's founders developed the social problem-solving model hypothesizing that problem-solving abilities are comprised of two major, partially independent processes: problem orientation and problem-solving skills (D'Zurilla & Goldfried, 1971;

D'Zurilla & Nezu, 1990). Problem orientation refers to a set of orienting responses when an individual is confronted with a problematic situation. These orienting responses include an attentional set to either recognize or ignore problems and a set of relatively stable cognitive-emotional schemas (e.g., beliefs) which describe how an individual generally thinks and feels about life's problems and his/her ability to solve them. Both attentional set and cognitive emotional schemas are metacognitive processes that are independent of any specific problem but may be activated when a person confronts a problem and experiences stress. Problem-solving skills, on the other hand, are the activities an individual uses to understand and consequently address his/her problems in everyday living. The four major problem-solving skills identified in PST are: (1) problem definition and formulation; (2) generation of alternative solutions; (3) decision making; and (4) solution implementation and verification (D'Zurilla & Goldfried, 1971).

D'Zurilla and colleagues (2004) developed a revised, five-dimensional social problem-solving model that comprises two different, albeit related, problem orientation dimensions and three different problem-solving styles. The two problem orientation dimensions are positive and negative problem orientation, whereas the three problem-solving styles are rational problem solving, impulsivity/carelessness style, and avoidance style. If an individual has a positive problem orientation, it is hypothesized that he/she addresses daily challenges using rational problem-solving skills such as considering alternatives and consequences before acting (D'Zurilla & Nezu, 2006). With a negative problem-solving style, an individual solves daily problem with impulsive/careless or avoidant problem-solving styles like rushing, being thoughtless [impulsive/careless], or procrastination and inactivity [avoidance] (Chang & Sanna, 2001). Figure 5 provides a relational diagram of the five dimensions. As discussed in detail elsewhere (D'Zurilla & Nezu, 2006; Robert, Ladouceur, Blais, Freeston, & Dugas, 1998), positive problem

orientation and rational problem solving are constructive dimensions that increase the likelihood of positive problem-solving outcomes, whereas negative problem orientation, impulsivity/carelessness style, and avoidance style are dysfunctional dimensions that disrupt or inhibit effective problem solving, leading to negative personal and social outcomes.

The problem-solving model explains the relationship between an individual's perception of life experiences or events and his/her problem-solving skills and expected outcomes. Richard Lazarus's (1999) relational model of stress is another theoretical framework that guides PST. It further clarifies how PST addresses psychological disorders including depressive and anxiety disorders. The relational model of stress includes two types of stressful life events that are major negative events (e.g., a job loss) and daily problems (e.g., job searches), which are assumed to influence each other. Both types of events have direct, negative impacts on an individual's well-being (i.e., increase psychological distress) and also have indirect effects via an individual's problem solving style as described in the problem-solving model (see Figure 6).

This model assumes that problem-solving functions mediate or moderate between life events and well-being. According to this relational model, there are two different mediational hypotheses. The first hypothesis is based on the ABC model (inherent in the cognitive-behavioral model), where stressful life events like job loss (A) set the occasion for problem-solving behavior like actively looking for a job (B) versus hibernating at home, which in turn results in personal and social consequences like getting another job rather quickly (C), which is likely to result in relief versus isolating oneself at home, which is likely to reduce psychological disorders, like feeling depressed. The second mediation hypothesis assumes problem-solving as an intervening variable in a causal chain, in which stressful life events negatively impact problem-solving, which

consequently decreases well-being. The moderator hypothesis assumes that stressful life events interact with problem-solving ability to influence well-being. Poor problem-solving ability increases the negative impact of stress on well-being, whereas positive problem-solving functions as a "buffer" to reduce the negative impact of stress on well-being. Based on both types of hypotheses, improving individuals' problem-solving skills and/or coping has a protective effect, potentially buffering individuals from psychological hardship like depressive and anxiety disorders.

Motivational interviewing (MI)

Miller and Rollnick (2012) define Motivational Interviewing (MI) as "a collaborative conversation style for strengthening a person's own motivation and commitment to change" (p. 12). William R. Miller and Stephen Rollnick originally developed this approach primarily for treating problem drinkers. Miller (1983) first described MI in detail and Miller and Rollnick (2012) further elaborated its clinical procedures. MI is grounded in a respectful and client-centered stance with a central clinical procedure of identifying, examining, and resolving clients' ambivalence about changing behaviors (Levensky, Forcehimes, O'Donohue, & Beitz, 2007; Miller, 1983). Closely tied to Rogers' (1973) interpersonal approach, MI is based on four general practice principles: (1) express empathy, (2) develop discrepancy between current behavior and important goals, (3) "roll with" or avoid struggling against resistance and ambivalence, and (4) support self-efficacy for change (Markland, Ryan, Tobin, & Rollnick, 2005; Thyrian et al., 2007). MI's four processes are: (1) engage (establish a connection and collaborative relationship with the client); (2) focus (clarify direction, the horizon toward which the client intends to move); (3) evoke (elicit the client's own) motivations for change; and (4) plan (work together with the client to develop

commitment to change and to formulate a specific action plan) (Miller & Rollnick, 2012). Core interviewing skills include asking Open-ended questions and Affirming, Reflecting, and Summarizing (OARS).

Theories underlying motivational interviewing

The main change theory underlying motivational interviewing is the Trans-theoretical Model of Behavior Change (TTM) (Prochaska & Velicer, 1997). The TTM construes behavior change as an intentional process that unfolds over time and involves progressing through a series of six stages of change (Prochaska, Diclemente, & Norcross, 1993): pre-contemplation, contemplation, preparation, action, maintenance, and termination (see Figure 7). However, most behavior change does not happen in a linear manner; instead, it is likely a client will move back and forth between the six stages.

In pre-contemplation, clients have no intention of taking action in the near future (6 months), i.e., they are not even thinking about change. Contemplation is the stage in which clients intend to change in the next 6 months, but not immediately (i.e., in the next month). While clients in the contemplation stage have stronger motivation to change than those in the pre-contemplation stage, they are not ready to start the process immediately. Once clients intend to take action (change) in the next month, they have moved into the preparation stage. Now with the client fully or somewhat motivated and ready for change, the emphasis of this stage is supporting the client in becoming well-prepared for change. In the action stage, change is typically overt and observable, and behavior change is often equated with action. Not all behavior modification counts as action in this model (West, 2006). There is clear consensus now that, in smoking cessation, for example, only total abstinence counts as action because other changes do not necessarily lead to quitting and do not lower risks associated with zero smoking. Change theories require the MI therapist

to identify a client's current stage of change and to use corresponding processes, techniques, and skills to assist the client in moving towards the next stage of change.

In addition to the TTM, two other important theories underlie some typical MI skills: Festinger's (1957) formulation of cognitive dissonance and Bem's (1972) reformulation as self-perception theory. According to Festinger's cognitive dissonance theory, individuals have an inner drive to hold all their inner attitudes and beliefs in harmony. Disharmony (or dissonance) serves as a powerful motivation for individuals to correct it and maintain consistency (Festinger, 1957). In the case of behavior change, it is, then, the provider's job to help the client recognize inconsistency (caused by maladaptive behaviors) so that he/she will become sufficiently motivated to resolve the maladaptive behaviors (which cause inconsistency) to maintain cognitive consistency. With regard to depressive and anxiety disorders, MI therapists increase clients' awareness of their negative emotions and connect negative emotion with clients' maladaptive thoughts and behaviors to create dissonance. Then, using the motivation generated from the dissonance, therapists work with clients to move along the spectrum of behavioral change, which should lead to further improvement in clients' sad mood (core components of depression) and/or worrisome thoughts (ingredients of anxiety).

Equally important to the change theories of MI is Bem's reformulation as self-perception theory. Bem (1972) asserted that when lacking an initial attitude due to a lack of experience, people develop attitudes based on observing their own and others' behaviors and drawing conclusions about what attitudes must have caused it. This theory offers grounds for behavioral change, i.e., clients are capable of hypothesizing feelings and attitudes without actually experiencing them. Therefore, for example, a client who had had alcohol use disorder for over 25 years is still capable of recognizing that alcoholics who no longer drink tend to have balance; that losing balance (the negative

behavior) is related to their drinking; and that they will regain balance once they quit (a good thing [attitude]). In fact, self-awareness is argued to be a form of metacognition (as discussed in cognitive behavioral theories) concerning one's own self-concept/perception (Samsonovich, Kitsantas, Dabbagh, & De Jong, 2008). This framework, in the case of depression and anxiety treatment, asserts that clients are capable of experiencing/envisioning alternative emotions (other than depression and/or anxiety) and evaluating the causes and consequences of their behaviors. Thus, formulation of cognitive dissonance and reformulation as self-perception theory are coupled with the TTM to guide MI change processes and techniques. Because clients are capable of evaluating and experiencing alternative positive emotions, if the therapists can bring these alternative emotion and positive behaviors to clients' awareness, cognitive dissonance will be invoked, which will lead to motivating clients to move forward on the TTM spectrum. With these strong and coherent theoretical foundations, it is not surprisingly that MI has been further expanded to various areas of interventions including depression and anxiety.

Solution-focused brief therapy (SFBT)

SFBT is a strength-based, client-centered, and future-oriented brief mental health intervention approach (Franklin, 2015; Gingerich & Peterson, 2013). Research on SFBT is growing rapidly. Originating in the early 1980s at the Brief Family Therapy Center in Milwaukee, Wisconsin, a group of master family therapists led by social workers Steve de Shazer (1988) and Insoo Kim Berg (Berg & DeJong, 2005) developed SFBT inductively. Grounded in social constructivism theories, SFBT focuses on patients' resources and knowledge rather than their histories and problems. Instead of focusing on "what caused clients' problems," SFBT practitioners are most interested in "what can

[and will] the clients do to get out of their problems in the future?" (Franklin, 2012). Taking a curious and respectful stance, SFBT practitioners work collaboratively with patients to co-construct solutions to their problems by drawing on past successes and what clients perceive to work well in solving their own problems (Flatt & Curtis, 2013).

The Solution-focused Brief Therapy Association (SFBTA) Research Committee developed the first SFBT treatment manual in 2008 and updated it in 2013 (Bavelas et al., 2013). The manual identifies active ingredients and the core processes of conversations important in SFBT. These ingredients involve conversations that involve a therapeutic process of co-constructing, altering, or creating new meanings with clients. Co-construction is a collaborative process in communication where speaker and listener collaborate to negotiate meanings, and this jointly-produced information in turn acts to shift meanings and social interactions (Bavelas et al., 2013). According to the SFBTA treatment manual, clients are specifically asked to co-construct a vision of a preferred future and draw on their past successes, strengths, and resources to make that vision a part of their everyday lives.

Theories underlying solution-focused brief therapy

Though debate on whether SFBT is theoretical or atheoretical (i.e., whether theory drives SFBT's change processes) continues, more and more studies point to a few theories that underlie SFBT's change process. First, SFBT was originally grounded in the constructivist approaches to communication and social interactional theories (de Shazer, 1988). Over time SFBT also became associated with social constructionism and the philosophical, post-structural views of language such as Wittgenstein's language games (Chang & Nylund, 2013). As Foord (n.d.) nicely state: "For a large class of cases of the

employment of the word 'meaning'—though not for all—this word can be explained in this way: the meaning of a word is its use in the language" (p. 43).

This quote reflects a later phase of Wittgenstein's thoughts on change in understanding 'meaning'–from a conception of meaning as representation to a view which looks to the meaning making process itself. Wittgenstein's philosophical thoughts, which essentially argue that language is directional (see, for example, Wittgenstein, 1967), influenced De Shazer's later thinking (De Jong & Berg, 2001; de Shazer, Dolan, Konnan, & Berg, 1997). Wittgenstein's thoughts were most influential to SFBT in describing the underlying change process in social constructivism/constructionism, thus the co-construction of meaning and solutions. McGee, Del Vento, and Bavelas (2005) effectively explained the process of co-construction by stating:

> "Because the client must provide information that the therapist does not have, he or she discovers and presents information consistent with the embedded presuppositions [of the therapist's questions]. So whether the client discovers, on one hand, abilities and positive qualities or, on the other hand, disabilities and pathology, he or she has been intimately involved in co-constructing this new common ground." (p. 5)

Therefore, because language and meaning are subject to change and alternation, and clients are an essential part of this process, the conversations (co-construction of solutions) between SFBT providers and clients are essential to therapeutic change. In fact, a systematic review and meta-synthesis (Franklin, Zhang, Froerer, & Johnson, 2017) of SFBT's change processes identified co-construction as one of SBFT's best empirically supported practices. Furthermore, under the broad framework of co-construction, researchers have also demonstrated that the specific questioning techniques (e.g., miracle questions, scaling, etc.) are an important means of facilitating change with clients

(Beyebach, 2014), which is further explained through another important theoretical underpinning of SFBT: theories of positive emotions.

Theories of positive emotions argue that increasing positive expectancies and positive emotion such as hope and optimism may be associated with positive outcomes in SFBT (Kim & Franklin, 2015). In the case of treating depression, anxiety, and other mental disorders, SFBT therapists use language to help clients construct their own narratives and co-construct with them to create a common ground for a "new reality or narrative" that includes solutions and positive emotions. Given this theoretical framework, SFBT techniques are tools used to enhance clients' positive emotions and, in turn, positive emotions expand clients' thought-action repertoire, which allows them to both perceive and become open to new ideas, behavioral changes, and other critical aspects of therapeutic change (Bannink, 2007; Kim & Franklin, 2015). Figure 8 presents SFBT change processes that Kim and Franklin (2015) propose. With both behavioral changes (solutions) and positive emotions and the interactive benefits of the two (more positive changes lead to more positive emotions and vice versa), clients are expected to be more open to change, engage in more behavioral improvements, and gain a greater sense of competence and capacity to reverse negative emotions. In summary, SFBT is grounded in social constructivism and co-construction with clients to develop new meanings, positive emotions, and solutions to maladaptive behaviors and emotions.

CHAPTER THREE: LITERATURE REVIEW AND GAPS IN THE LITERATURE

Each of the four interventions included in this review has garnered sufficient empirical evidence of its effectiveness in treating both depressive and anxiety disorders in mental health outpatient specialty care settings, but evidence is still accumulating regarding the effectiveness of these four approaches for treating depressive and anxiety disorders in primary care settings. This section reviews literature on the four interventions and aims to identify gaps in the existing literature.

Cognitive behavioral therapy

In general, cognitive behavioral therapies' effectiveness in treating depressive and/or anxiety disorders is well supported. Several meta-analyses (Hofmann, Asnaani, Vonk, Sawyer, & Fang, 2012; Spek et al., 2007; Stewart & Chambless, 2009) report that effect sizes of CBT for both depression and anxiety are robust with an average large treatment effect size (d = 0.96). For treating depression, Cuijpers et al. (2013) reported that CBT's efficacy for depression was mixed with some studies suggesting strong and others weak evidence. In treating anxiety, Hans and Hiller (2013) confirmed CBT's well-established efficacy for treating adults with anxiety in traditional outpatient clinics but identified the need to further examine CBT's efficacy for treating anxiety in other practice settings including primary care settings.

In addition to the general CBT literature on depression and anxiety, many studies have examined non-interpersonal CBT for DADs. Spek and colleagues (2007), for example, conducted a meta-analysis of internet-based cognitive behavior therapy for symptoms of depression and anxiety (d = .60 and d = .96, respectively) and found that internet-based CBT has effects comparable to interpersonal CBT. Similarly, Kaltenthaler

and Cavanagh (2010) found that computerized cognitive behavioral therapy is as effective as therapist-led cognitive behavioral therapy. However, the effectiveness of non-interpersonal CBT for anxiety disorders is unclear. A systematic review of computerized cognitive behavior therapy for phobias and panic disorder shows that computerized cognitive behavioral therapy is not as effective as conventional therapist-led cognitive behavioral therapy for treating phobias and/or panic disorder, though it is superior to relaxation training and being on a waiting list (Ferriter, Kaltenthaler, Parry, & Beverley, 2008). Another review, by Coull and Morris (2011), of cognitive-behavioral therapy-based guided self-help in treating depressive and/or anxiety disorders reached similar conclusions. Although there is support for the effectiveness of this type of intervention, RCTs included in the review had limited effectiveness in routine clinical practice, thus the authors were unable to conclude that CBT-based guided self-help treatment has comparable effects to its interpersonal counterparts.

Both the in-person and tele-health based intervention literature has confirmed CBT's clinical efficacy for treating depressive and/or anxiety disorders, with stronger evidence for interpersonal CBT. However, review studies of CBT in primary care did not appear until after 2010.

Twomey, O'Reilly, and Byrne (2015), for example, conducted a meta-analysis of CBT-focused RCTs (n = 29) for treating depression and anxiety in primary care and reported that multi-modal CBT (CBT delivered in using a combination of modalities like internet and primary care based) was more effective than a no treatment control condition (d = .59) and routine primary care TAU (d = .48). A review of studies of CBT for depression and anxiety disorders delivered in primary care by primary care therapists found that both interpersonal and non-interpersonal CBT delivered in primary care can be potentially beneficial, but results were inclusive because the quality of primary studies

was unsatisfactory (Hoifodt et al., 2011). Despite some promising findings about primary care-based CBT, results remain inconclusive, warranting further investigation (Coull & Morris, 2011; Hans & Hiller, 2013).

In summary, evidence on CBT interventions for treating depression and anxiety in primary care remain unclear. Like Hofmann and colleagues (2012) in their overview of meta-analyses (i.e., a systematic review of meta-analysis studies), the author of this dissertation believes it is important to further evaluate the empirical evidence of CBT among subgroups and in various settings, including primary care.

Problem solving therapy

There is strong empirical support for problem-solving therapy's effectiveness in treating depressive disorders. In a meta-analysis, Cuipers et al. (2013) found PST's mean effect size for depression was 0.83 (95% CI: 0.45-1.21) using a random-effect model, and it was as or more effective than other psychosocial and pharmacological treatments. Bell and D'Zurilla (2009) reached a similar conclusion in their study with a mean difference effect size of 0.40 ranging from -1.15 to 3.8. Gellis and Kenaly (2008) systematically reviewed all problem-solving therapy studies for adult depression, concluding that it was superior to several alternative interventions in reducing depressive symptomatology and that effects were maintained for substantial periods beyond treatment cessation. A meta-analysis of PST for major depressive disorder in older adults published in 2016 found that PST significantly decreased depressive symptoms with an effect size of d = 1.15, 95% CI: 0.55 – 1.76 (Kirkham, Choi, & Seitz, 2016). It should be noted that PST for depression treatment has been specifically adapted for primary care settings (PST-PC) (Hegel et al., 1999) and can be delivered by a broad range of healthcare providers using fewer and shorter sessions than traditional PST. Emerging literature indicates that PST-

PC is not only feasible for use in primary care settings, it is also effective, with Zhang, Park, Sullivan, and Jing's, (2018) meta-analysis reporting an overall statistically significant treatment effect of PST for primary care depression and anxiety ($d = 0.637$, $p < 0.001$).

In addition to the strong empirical support for interpersonal PST, several clinical trials have also demonstrated the efficacy of tele-health based PST, especially for depressive disorders among older adults. Buntrock and colleagues (2017), for example, reported that a web-based PST intervention for adults with subthreshold depression significantly improved participants' depression free years with additional reduced risk of developing a major depressive disorder. More importantly, Choi, Marti, and Conwell (2016) reported that participants receiving tele-PST, but not in-person PST, exhibited lower suicidal ideation and depression ratings across the follow-up period. In another study, Choi et al. (2014) also reported that while both tele-PST and in-person PST were efficacious for improving geriatric depression and disability outcomes, Tele-PST had a larger treatment effect size than in-person PST ($d = .68$ versus $d = .20$).

Though PST's treatment efficacy for depressive disorders is well supported, only a few clinical trials have tested it in treating depression and results have been mixed. Kleiboer et al. (2015) did find PST effective in decreasing anxiety when coupled with support services. In another randomized controlled trial, Mikami et al. (2014) found PST effective in preventing new onset of post-stroke generalized anxiety disorder (GAD) when delivered in a healthcare setting. Hoek et al.'s (2012) randomized controlled trial also found that internet-based guided self-help problem-solving therapy reduced both recipients' depression and anxiety scores.

In general, the empirical literature shows that PST is effective in treating depressive and anxiety disorders (Malouff, Thorsteinsson, & Schutte, 2007), and

evidence favors its effectiveness in treating depression. While a few studies have tried to examine PST's effectiveness for treating mental disorders in healthcare settings, results were inconclusive for most of them (Perri et al., 2001; Steiner et al., 2002), emphasizing the need to systematically examine PST's effectiveness for depressive and anxiety disorders in healthcare settings.

Problem-solving therapy was derived from CBT, but PST has been modified into much briefer interventions than CBT while being equally effective. Two other brief mental health interventions, Motivational Interviewing (MI) and Solution-Focused Brief Therapy (SFBT), were developed in other fields (i.e., substance use treatment and family therapy, respectively). They are as brief as PST, and research on their use in treating depression and anxiety disorders is accumulating.

Motivational Interviewing

Overall, the empirical evidence on motivational interviewing for treating depression and anxiety in primary care is relatively weak compared to its effectiveness in treating problematic substance use and addictive behaviors. One meta-analytic review (Burke, Arkowitz, & Menchola, 2003) of MI revealed moderate to large treatment effects (d = .25 to d = .57) with an average statistically significant treatment effect size of d = .47 for problems involving alcohol, drugs, and diet and exercise. Similar reviews also reported moderate to large treatment effects for MI addressing clients' health behaviors such as weight loss and exercise, d = 1.417 (Amstrong et al., 2011), smoking cessation, OR = 1.45 (Heckman, Egleston, & Hofmann, 2010), and alcohol consumption, d = .43 (Vasilaki, Hosier, & Cox, 2006).

While the evidence on MI for behavioral change is generally positive, findings on MI's effectiveness for depressive and/or anxiety disorders in primary care and other

health care settings is inconsistent. While numerous randomized controlled trials support MI as an effective and useful add-on to other psychotherapies for depression and anxiety (Hsieh et al., 2012; Seal et al., 2012; Westra, Arkowitz, & Dozois, 2009), these studies were unable to clearly differentiate the treatment effects that can be attributed to MI. In fact, a few systematic and/or meta-analytic reviews were unable to identify MI-only studies for depressive and/or anxiety disorders (Lundahl & Burke, 2009; Rubak et al., 2005) because most individual studies did not use MI as the sole treatment approach for these disorders. Westra, Aviram, and Doell (2011) further confirmed this understanding of the MI literature in stating that "while preliminary findings are promising in supporting the addition of MI to existing therapies for many major mental health problem, research is in the early stages, with existing studies having numerous methodological limitations" (p. 643). In fact, Lundahl et al.'s (2013) systematic review and meta-analysis of MI RCTs in medical settings found no significant overall treatment effect and possibly worse treatment effects of MI for mental health conditions, especially compared to other specific treatment like CBT.

Given the contrasting empirical evidence on MI's treatment effects for health behavior and substance use problems versus mental health problems (including depression and anxiety), a more thorough examination of the literature on MI for treating depressive and/or anxiety disorders in healthcare settings like primary care is needed. A few MI trials for alcohol consumption in primary care (D'Amico, Miles, Stern, & Meredith, 2008; Kaner et al., 2007) along with one systematic review and meta-analysis (Bertholet, Daeppen, Wietlisbach, Fleming, & Burnand, 2005) supported MI's treatment effectiveness for substance use problems in primary care. However, whether MI's treatment effects transfer to mental health problems in primary care needs further investigation. A recent systematic review and meta-analysis of MI (Vanbuskirk &

Wetherell, 2014) focused exclusively on MI with primary care populations, but it did not include mental health-related outcomes and focused only on MI's effect on improving primary care patients' health behaviors (e.g., daily exercise, smoking cessation).

Solution-focused brief therapy

Over the past decade, numerous clinical trials have supported SFBT's effectiveness in decreasing psychological distress (Franklin, 2015; Gingerich & Peterson, 2013), especially internalizing (including depressive and/or anxiety) disorders. Several systematic and meta-analytic reviews support SFBT's effectiveness for treating depressive and anxiety disorders across various service settings including hospitals. Kim (Kim, 2008) conducted the first systematic review and meta-analysis of SFBT in the United States and reported significant, though small, positive treatment effects for internalizing outcomes (d = .26, p < .05). Over 50% of the internalizing outcomes were measures of depression and/or anxiety, lending strong support to SFBT's effectiveness in addressing these disorders. Other systematic and/or meta-analytic reviews of SFBT in schools (d = .23, p<.05 for externalizing and d = .40, p < .05 for internalizing outcomes) (Franklin, Kim, & Tripodi, 2009), for internalizing outcomes across all settings (d = .23, p < .05) (Schmit, Schmit, & Lenz, 2015), and most relevant to the context of this review, in hospital settings (d=0.94, p<.001) (Zhang, Franklin, Currin-McCulloch, & Kim, 2017) support SFBT, especially for treating depression and anxiety.

A qualitative/descriptive review of SFBT (Gingerich & Peterson, 2013) found SFBT effective for treating mood-related mental disorders, especially depressive disorders and noted that SFBT can achieve similar, if not greater, treatment effects than alternative interventions (e.g., medication management or interpersonal psychotherapy) with fewer sessions. Therefore, it has been suggested that SFBT has significant potential

for depression and anxiety treatment in healthcare settings, which general requires briefer forms of treatment. Researchers such as Franklin (2015), Gingerich and Peterson (2013) encourage further examination of SFBT for its utility in healthcare, including primary care, settings.

Summary of the empirical literature

In summary, empirical studies favor, albeit at different levels of confidence, the effectiveness of the interventions mentioned above for treating depressive and/or anxiety disorders, especially in mental health specialty outpatient settings, but when delivered in various health care settings, including primary care, their effectiveness remains inconclusive. The empirical literature on primary care-based interventions is growing rapidly with more and more studies focusing on the use of technology, brief therapy, and a combination of both, opening the way for more systematic reviews and meta-analyses.

Not surprisingly, of the four interventions discussed here, CBT has received the greatest attention from the research community. While the empirical evidence for CBT for patients with DADs in primary care settings seems promising overall, some RCTs and a few meta-analyses did not show findings favoring CBT. PST seems to be a very promising intervention for primary care patients with DADs. In addition to its already strong empirical foundation for depression treatment in typical mental health specialty care settings, studies show that PST interventions, and especially its primary care-based version, PST-PC, are not only highly feasible but also can be delivered to primary care patients with high fidelity. More importantly, numerous tele-health-based PST (tele-PST) studies also indicate that tele-PST can potentially benefit primary care patients with depression and anxiety. With accumulating empirical evidence supporting PST's benefits in treating primary care patients with DADs, a current systematic evaluation of these

evidence is warranted. In contrast, MI and SFBT have received relatively less support than CBT and PST when used in primary care settings. This may not be surprising given that MI originated in the substance abuse field and SFBT grew out of the family therapy field. MI has gained significant empirical support over the past decade, and there have been some investigations of its effectiveness for depressive and/or anxiety disorders. Similarly, SFBT is a relatively young intervention which has received greater empirical support for treating depression and anxiety over the years. Both the MI and SFBT literature have reached a stage that calls for a systematic evaluation of the effectiveness of these interventions for primary care patients' depressive and/or anxiety disorders to summarize past research and identify a future research agenda.

An important line of literature, though not addressed in this dissertation, is the stepped care model of mental health interventions in primary care settings (Haaga, 2000; Richards, 2012). The stepped care model acknowledges a significant gap between the demand for psychological therapy services and available resources. As a result, a minimalist approach of psychosocial intervention is adopted to enhance the efficiency of service provision (Bower & Gilbody, 2005). The stepped care model requires treatments of differing intensity to better match the needs of different clients. Typically, in the first step, clients in need are put on a "watchful waiting list" to determine if further interventions are needed. One rationale behind this list is that almost half of all patients with a depressive episode recover spontaneously within three months with no intervention (Spijker et al., 2002). A second step involves guided self-help interventions so that high functioning clients can manage their clinical symptoms using accessible, cost-efficient and effective interventions. If a client's symptoms are not managed well after the first two steps, the third step, brief face-to-face interventions, is introduced.

Finally, if a brief face-to-face intervention has not been effective, the fourth step, longer-term, face-to-face psychotherapy and medication, is considered.

The stepped care model makes it obvious that its rationale is to match intervention intensity to clients' level of functioning and clinical severities in order to maximize the use of limited psychosocial intervention resources at the population level. While a detailed conceptual and theoretical discussion can be found elsewhere (Bower & Gilbody, 2005; van Straten, Seekles, van 't Veer-Tazelaar, Beekman, & Cuijpers, 2010), increasing empirical literature supports the effectiveness of stepped care models (Katon et al., 1999; Seekles, Van, Beekman, Van, & Cuijpers, 2011), and they have become increasingly important in managing mental disorders. However, since an intervention of the type investigated in this meta-analysis is embedded in only one step, i.e., step three (a brief face-to-face intervention), and a client may or may not receive that intervention after beginning a stepped care program, stepped care models are not included in this dissertation.

CHAPTER FOUR: METHODS

Using a systematic review and meta-analysis of clinical trials, this study sought to evaluate the overall effectiveness of the four brief interventions described above when delivered to treat anxiety and depressive disorders in primary care settings. Additional objectives of this project include: (1) Determine if effectiveness differs across the interventions; (2) Evaluate whether any factor (e.g., treatment length, study design, years of provider experience) moderates the treatment effect estimates; and (3) Identify gaps remaining in the empirical literature and a future research agenda.

Study selection

Inclusion criteria

To be eligible for inclusion, a study must have examined one of the four interventions targeted in this review, cognitive behavioral therapy, problem-solving therapy, motivational interviewing, or solution-focused brief therapy. The study must also have been a randomized controlled trial (RCT). RCT designs are considered as the highest quality research evidence in healthcare research (Burns, Rohrich, & Chong, 2011). While acknowledging the value and contributions of other experimental designs and qualitative research methodologies, the review includes only RCTs for two reasons. First, a preliminary scoping review of the literature identified a sufficient number of eligible RCTs for conducting a meaningful synthesis. Second, given the nature of RCT designs, primary studies' results should provide strong internal validity for understanding the clinical effectiveness of the four interventions for treating depression and anxiety in primary care.

When the studies selected contained more than one treatment group, and each treatment group received a different intervention, that study was further screened to

determine if the treatment effect (or lack thereof) can be attributed to the intervention of interest. For example, if a study examined CBT (one of the four interventions of interest in this study) for one treatment group and interpersonal therapy (not one of the four interventions of interest) for another treatment group and compared their effectiveness to a treatment-as-usual (TAU) control group, the study was included, and CBT's treatment effect was obtained by comparing CBT with interpersonal therapy and with TAU. Studies that examined an intervention of interest plus an intervention that is not a focus of this review were generally excluded. For example, a study that compared CBT plus interpersonal therapy for the treatment group versus TAU was excluded unless there was explicit evidence that treatment group participants received an average dose of CBT that exceeded 60% of the entire treatment dosage. When a study included two interventions of interest, for example, CBT plus MI for the treatment group compared to TAU, that study was included for estimating an overall treatment effect but was excluded from potential moderator analysis.

Another inclusion criterion was that a study had to report at least one depressive or anxiety outcome. When an outcome measure contained measures of depression and/or anxiety, the study was further examined for its eligibility. If a measure assessed both depression and anxiety but did not report separate scores for the two constructs, that study was excluded. For example, the Brief Symptom Inventory–18 (BSI-18) (Derogatis, 1993) reports an overall score of psychological distress that is the sum of sub-dimensions of depressive symptoms, anxiety symptoms, and somatic symptom scores. If a study reported only the overall BSI-18 score, it did not meet inclusion criteria and was excluded, but if it reported BSI-18 sub-dimension scores for depressive and/or anxiety symptoms, it was included.

A final inclusion criterion was that a study had to examine a primary care-based intervention as defined in Chapter One. To reiterate, primary care based intervention include: (1) an intervention delivered in a primary care setting by a health care provider or through a technological platform or a combination of both, or (2) an intervention delivered outside a primary care setting by a health care provider or through a technological platform, or a combination of both, but directly connected with or prescribed by a primary care health care provider.

Exclusion criteria

A study was excluded if it did not include one of the four identified interventions, was not an RCT, did not report one or more distinct measures of depressive or anxiety outcomes, or did not examine primary care-based interventions.

Search strategies

Materials included in the search contained both published manuscripts and unpublished studies, including dissertations, grey literature, and documents obtained from additional literature searches (Higgins & Green, 2011; Petticrew & Roberts, 2006).

Five strategies were followed to create an initial pool of potential studies for screening and review that included searches of (1) electronic databases, (2) professional websites, (3) dissertation abstract databases, and (4) reference lists in included studies and systematic reviews, as well as (5) contacting experts in the field. First, using a pre-defined set of key words (described later), seven electronic databases were searched for materials appearing from 1900 to April 2016 including (1) Academic Search Complete (ASC), (2) PsycINFO, (3) Cumulative Index of Nursing and Allied Health Literature (CINAHL), (4) PUBMED, (5) Medline, (6) The Cochrane library/database of systematic

reviews and controlled trials, and (7) ProQuest Dissertations & Theses Global. Also searched were professional websites relevant to the interventions reviewed or the disorders targeted, including Academy of Cognitive Therapy (www.academyofct.org), the IMPACT evidence-based depression care (www.impact-uw.org), Motivational Interviewing Network of Trainers (http://www.motivationalinterviewing.org), Solution-focused Brief Therapy Association (www.sfbta.org), European Brief Therapy Association (www.ebta.eu), and Anxiety and Depression Association of America (www.adaa.org). Finally, experts and well-known researchers of the four interventions reviewed were contacted to determine if additional studies were available.

Within each database, three sets of key words were used to identify (1) the four brief interventions targeted, (2) depressive and/or anxiety disorders, and (3) primary care settings. To identify cognitive-behavioral therapy, the key words used were "cognitive behavior therapy" or "cognitive-behavior therapy" or "cognitive therapy" or "CBT." To identify problem solving therapy, the key words were "problem solving therapy" or "problem-solving therapy" or "problem solving" or "PST." To identify motivational interviewing, the key words were "motivational interviewing" or "motivational interview" or "MI." To identify solution-focused brief therapy, the key words were "solution-focused brief therapy" or "solution focused brief therapy" or "solution focused" or "SFBT."

During keyword searches for titles and abstracts, depressive and/or anxiety disorders were identified using the key words "depression" or "depressive" (to cover the entire spectrum of depressive disorders) or "anxiety" or "panic" or "phobia" (to cover the whole spectrum of anxiety disorders including generalized anxiety disorder and social anxiety disorder). To identify primary care settings, the key words used were

"primarycare" or "primary care" or "PCP" or "family medicine" or "family doctor." Figure 9 presents the Search Procedure chart of the review process.

Data extraction and coding

The author of this review coded all included studies. Two other coders (one with a PhD in psychology and one finishing a PhD in social work) coded 50% of the studies as a confirmation check using a pre-developed coding sheet. In addition to bibliographical information, participants' demographic information was recorded, including age, gender, and race/ethnicity. Given that this review included only randomized controlled trials, the nature of the comparison group was recorded (e.g., treatment as usual, another treatment, waitlist) and whether an intervention was delivered in the physical setting of a primary care practice or other setting was also recorded. Also coded for primary studies were treatment modality and dosage and providers' professional background. Diagnostic criteria and (un)standardized measures of depressive and/or anxiety outcomes were also coded.

Data analysis

Data analysis proceeded in three stages and all analyses were conducted using R software (R Development Core Team, 2016). First, descriptive statistics were calculated (% for categorical variables and means and standard deviations for continuous variables) for study characteristics (e.g., sample size, research design, treatment modality [individual versus group]). Second, treatment effect size estimates were calculated for each individual study to determine treatment effect magnitude. For continuous outcomes, the standardized mean difference (SMD) was calculated using the group mean in the treatment condition minus the mean in the control condition and then dividing the

difference by the pooled within-group standard deviation (Equation [1]) (Cooper et al., 2009). The pooled within-group standard deviation was obtained with a function specified in the equation below, where df_1 was the degrees of freedom in the treatment group, df_2 was the degrees of freedom in the comparison group, S_1 was the standard deviation in the treatment group, and S_2 was the standard deviation in the comparison group.

$$\mathrm{SMD} = \frac{\mathrm{Y_{trt}} - \mathrm{Y_{con}}}{\mathrm{S_{pooled}}} \quad [1]$$

$$\mathrm{S_{pooled}} = \sqrt{\frac{(\mathrm{df_1})\mathrm{S_1^2} + (\mathrm{df_2})\mathrm{S_2^2}}{(\mathrm{df_1} + \mathrm{df_2})}} \quad [2]$$

Because the SMD statistic tends to over-estimate the "true" treatment effect parameter (Ellis, 2009), Cooper and colleagues (2009) suggest using small sample size bias correction for the SMD statistic using a J function (Equation [3]). The J function incorporates a study's sample size, in the form of degrees of freedom, to further correct for studies with small sample sizes.

$$\mathrm{J(df)} = 1 - \frac{3}{4\mathrm{df} - 1} \quad [3]$$

For binary treatment outcomes, effect size estimates were calculated as follows: First, an odds ratio (OR) was calculated using Equation [4], where Pt is the percentage of participants who improved or had a positive outcome in the treatment group and Pc is the percentage for the comparison group.

$$\mathrm{OR} = \frac{[\mathrm{P_t}/(1-\mathrm{P_t})]}{[\mathrm{P_c}/(1-\mathrm{P_c})]} \quad [4]$$

Second, a log odds ratio was calculated by obtaining the log transformation of the odds ratio [ln(OR)]. Finally, log odds ratio (LOR) was transformed into the same effect size metric as the SMD effect size estimates using Equation [5] (Cooper et al., 2009).

$$\text{transformed LOR} = \left[\frac{\sqrt{3}}{\pi}\right][Ln(OR)] \quad [5]$$

Both the small sample size corrected SMD effect size estimates and the transformed log odds ratio effect size estimates were based on the same metric and noted as d in this review.

Synthesizing effect size estimates and moderator analyses

Meta-regression (Borenstein, Hedges, Higgins, & Rothstein, 2009a), a regression-like procedure used in meta-analysis, was used to synthesize effect size estimates and moderator analysis. In meta-regression, the outcome variable is the treatment effect size reported in primary studies and the covariates are study-level characteristics, like average minutes per session of the intervention or service providers' educational background. An intercept only meta-regression model offers an overall average of treatment effect sizes across studies. Adding a covariate(s) to the meta-regression model allows for investigating the effect of potential moderators on treatment effect sizes.

A common challenge when synthesizing effect size estimates across individual studies is handling statistical dependence when multiple effect sizes were reported for a

single study construct. When several measures of the same construct are used in a single study, the same group of participants has been measured multiple times; therefore, the scores of these measures are not independent of each other. In addition, when a study compared more than one treatment group to the same control group, for each treatment-control dyad, there is one or more effect size estimates. However, because the difference in measure(s) is based on the same control group, the measures are not independent of each other.

In such situations, the analysis must account for possible dependence among the treatment effect sizes (Cooper et al., 2009). To handle dependence, two types of approaches are most commonly used: ad hoc and post hoc. In ad hoc approaches, the researcher manually removes dependent effect size estimates, leaving only one effect size per study to ensure independence. Typically, the researcher either selects one measure arbitrarily or takes a weighted average of multiple measures for the same construct within the study. Consequently, only one treatment effect size, either selected arbitrarily or a weighted average, is included in the final meta-analysis. These ad hoc approaches, however, introduce researcher bias in the final analysis, and there is no way to evaluate such bias. Therefore, these ad hoc approaches were not used in this project.

In contrast, post hoc approaches are more principled and involve using statistical methods (rather than researcher discretion) to handle the dependence, including the Generalized Least Square (GLS) method (Gleser & Olkin, 2009) and the Hierarchical Linear Modeling (HLM) method (Van den Noortgate et al., 2013). However, both of these post hoc approaches have disadvantages. To effectively account for the within study dependency of multiple effect sizes, the GLS method requires knowledge of the covariance structure of dependent effect sizes (Gleser & Olkin, 2009), which are often not reported in primary studies. As a result, GLS is often not a feasible approach for

meta-analysts, as was the case in this project. The HLM method avoids this problem but carries strong assumptions about effect size estimates' sampling distributions, which a dataset may or may not meet (Raudenbush & Bryk, 2002).

A relatively new method that addresses the challenges mentioned with the GLS and HLM methods is Robust Variance Estimation (RVE) (Hedges, Tipton, & Johnson, 2010; Tipton & Pustejovsky, 2015). RVE better fits the data used in this study because it makes no assumptions about effect size estimates' sampling distribution and can estimate the covariance structure of the dependent effect sizes without actually knowing it (Hedges et al., 2010; Tipton & Pustejovsky, 2015). Additionally, simulation studies suggest that RVE may yield accurate estimation of an average treatment effect with as few as 10 primary studies and has satisfactory performance for moderator analysis with 20 to 40 studies (Tipton & Pustejovsky, 2015). To control for possible inflated Type I error of test statistics and confidence intervals, this study incorporated small sample size correction into meta-regression with robust variance estimation (Tipton, 2015; Tipton & Pustejovsky, 2015).

Publication bias

Publication bias describes the situation in which published research literature is systematically unrepresentative of the population of completed studies (Borenstein, Rothstein, & Sutton, 2006). Studies with insignificant findings are less likely to be published than studies with statistically significant treatment effects. Including only published studies may introduce an upward bias into the estimation of an overall treatment effect across studies (Cooper et al., 2009). While published and unpublished studies and grey literature were searched for this project, publication bias was still assessed to inform interpretation of the results.

Quality of studies rating and risk of bias

To assess study quality, the Jadad Scale (Jadad et al., 1996), often called the Oxford quality scoring system, was used. A systematic review of RCT quality rating scales reported that the Jadad Scale is the most widely used quality rating scale in health care research, and more importantly, that it has the highest validity and reliability in evaluating RCTs (Olivo et al., 2008).

To assess risk of bias, the Cochrane Collaboration's tool for assessing risk of bias in randomized trials (Higgins et al., 2011) was used. The risk of bias tool examines six domains of bias in an RCT and offers a clear visual presentation of assessment results. An interdisciplinary team of experts developed the tool in 2005, and it was evaluated in 2009. While the tool's reliability has not been extensively studied, a review indicated the risk of bias tool can effectively identify an individual trial's risk of bias based on the use of this tool in previous empirical studies (Higgins et al., 2011).

CHAPTER FIVE: RESLTS

Search results

Figure 9 presents detailed steps and results of the literature search. An initial pool of 1,140 articles (from both electronic database search and manual search) were identified for initial screening after duplicates were removed. Of the 1,140 articles, 961 articles were excluded based on title and abstract review. Of the 961 articles excluded, 838 articles were excluded based on title review and 123 articles were excluded based on abstract review. This resulted in a sample of 179 articles for full text review. Also excluded after review of full text and statistical eligibility were 114 articles for reasons such as the article reported on a study that was not conducted in a primary care setting, reported a study protocol (not study results), or did not report sufficient statistical information for calculating effect size estimates. When a study met all inclusion criteria except sufficient statistical information, efforts were made to contact the study author(s). An analytical sample of 65 primary studies was included in the final meta-analysis.

Quality of studies and risk of bias:

Using the Jadad Scale (Table 1) for Reporting Randomized Controlled Trials, the 65 trials had an average score of 3.22 (SD = 1.21) out of 5.0, indicating acceptable overall study quality among included primary studies. The primary studies were rated g in mentioning randomization (65/65), and acceptable in tracking all participants (47/65), appropriate randomization (44/65) and mentioning blinding (39/65). They were, however, not satisfactory using appropriate blinding, if at all blinding was used (14/65). Using the Cochrane Collaboration's tool for assessing risk of bias (Table 2), studies were rated most satisfactorily in random sequence generation (65/65), selective outcome reporting (58/65), and handling incomplete outcome data (43/65). Risk of bias was observed in

allocation concealment (28/65) and blinding of outcome data assessment (26/65). The greatest risk of bias occurred across studies due to poor blinding of study participants and personnel (5/65), with the majority of studies explicitly reporting inability to blind participants and personnel.

Publication bias:

Publication bias was assessed by plotting observed treatment effect size estimates against their standard errors (Figure 10). Overall, the distribution of effect size estimates was reasonably symmetric. While a few effect size estimates (n = 4) were greater than the observed average treatment effect size and have large standard error, they only counted for 2% of the total number of effect size estimates. Thus, funnel plots indicate that publication bias is not a concern.

Study characteristics

Study characteristics are presented in Table 3, 4, and 5. The 65 primary studies included studies examining cognitive-behavioral therapy (n = 47), problem-solving therapy (n = 12), and motivational interviewing (n = 6). Using the search strategy and inclusion/exclusion criteria, no study of solution focused brief therapy (n = 0) was identified that meet the criteria. Most of the studies primarily investigated depressive outcomes (n = 54, 83.08%), while 10 studies (15.38%) investigated anxiety outcomes and one study investigated co-morbid depression and anxiety. Taken together, the 65 primary studies included a total sample of 10,951 participants. Sixty-one studies reported participants' ages ranging from 14.6 to 77.0 years old with a mean age of 45.17 (SD = 15.76). Thirty-seven primary studies reported participants' racial background with an average of 64.18% of participants being non-Hispanic White. Four of the 37 studies were

racially specific intervention studies (e.g., CBT for Asians or PST for African Americans) in which the percentage of non-Hispanic White participants was coded as 0%. After excluding these racial specific intervention studies (n = 32), the average was 73.91% non-Hispanic White participants. Sixty studies reported participants' gender with an average of 68.75% of participants being female, and 42 studies reported that half (49.98%) of the participants were married. While efforts were made to collect other demographic and socio-economic background information, significant amounts of missingness (50% or higher) prevented meaningful syntheses of these characteristics including participants' education, income, socioeconomic status, and family support among others.

Most interventions were delivered in primary care settings (n = 42, 64.62%) while in 18 studies interventions were delivered outside primary care settings, and in 5 studies the intervention was delivered both in and outside primary care settings. Fifty-eight (89.23%) studies used individual interventions and 7 studies (10.77%) studies used group interventions. Most studies (n = 40, 61.5%) used non-telehealth interventions only, 18 (27.7%) used tele-health-based interventions only, and 7 (10.8%) used a combination of tele-health and in-person approaches. The average number of individual sessions was 7.91 with a range of 3 to 15 sessions. Sessions averaged at 49 minutes each (SD = 20.69), and the duration of individual interventions averaged at 10.86 weeks ranging from 3 to 52 weeks across studies. Group interventions reported an average 89 minutes per session (SD = 34.71), ranging from 60 to 145 minutes. Total number of group sessions averaged at 8.86 sessions (SD = 2.45), ranging from 5 to 12 sessions, lasting on average over 8.43 weeks (SD = 2.44), ranging from 5 to 12 weeks. Forty-three studies reported service providers' educational background: 4 studies used bachelor's level providers, 23 used master's level providers, 13 used doctoral level providers, and 3 used both master's and

doctoral level providers. Of the 61 studies that reported primary care physicians' involvement in delivering psychosocial interventions, 50 studies (82.0%) did not involve primary care physicians, 10 (16.4%) involved primary care physicians in various ways (e.g., co-provider, supervisor, medication manager), and in one study (1.6%) primary care physicians were the primary treatment providers. Significant missingness (89.2% missing) was observed across studies in reporting practitioners' years of experience, thus meaningful synthesis was forbidden.

With regard to primary study designs, 49 studies (75.4%) used an active control group design, 10 (15.4%) used a medication only control condition, and 6 (9.2%) used placebo or waitlist controls. Most primary studies (n = 42, 64.6%) examined one intervention independently. Twenty primary studies (30.7%) reported that the intervention of interest was delivered in conjunction with other therapeutic techniques or approaches but explicitly reported that the investigated intervention was the primary treatment.

Meta-analytic results

Between study heterogeneity was assessed using the "metafor" package with its rmv.mv function in R to calculate between study variability, $\sigma^2 = 0.253$, $p < 0.001$. Results indicated significant between study heterogeneity, supporting the decision to pool effect size estimates using a random effect model and conducting moderator analyses using mixed-effect models. Results of an overall treatment effect size estimate and subgroup-analyses are presented in Table 6. Overall, the 65 primary studies included 198 reported effect size estimates with a pooled averaged treatment effect of d = 0.462, $t(39) = 7.36$, $p < 0.001$, 95% CI (0.335, 0.589). This means that, on average, the psychosocial interventions were 0.462 standard deviations higher (with higher indicating better) in

depressive and/or anxiety outcomes among participants in the treatment group than their counterparts in the control group. Additionally, this average treatment effect size estimate was statistically significant as evidenced by a 95% confidence level that did not include zero. Subgroup analyses revealed an overall statistically significant treatment effect for depressive outcomes, $d = 0.424$, $t(43.3) = 6.21$, $p < 0.001$, 95% CI (0.286, 0.561) and for anxiety outcomes, $d = 0.547$, $t(11) = 6.1$, $p < 0.001$, 95% CI (0.350, 0.744). Studies of interventions conducted in primary care settings reported an overall statistically significant treatment effect for depressive and/or anxiety outcomes, $d = 0.450$, $t(23.2) = 6.77$, $p < 0.001$, 95% CI (0.312, 0.587). Studies of interventions outside primary care settings or delivered in and outside primary care settings reported an overall statistically significant treatment effect for depressive and/or anxiety outcomes, d = 0.450, $t(23.2) = 6.77$, $p < 0.001$, 95% CI (0.312, 0.587), and $d = 0.478$, $t(18.5) = 3.31$, $p < 0.01$, 95% CI (0.175, 0.780), respectively.

Twelve studies reported 31 treatment effect size estimates for problem-solving therapy and indicated an overall statistically significant treatment effect, $d = 0.45$, $t(8.44) = 2.46$, $p < 0.05$, 95% CI (0.032, 0.869). An overall treatment effect was also statistically significant for cognitive behavioral therapy studies (48 studies, 157 effect sizes), $d = 0.474$, $t(28.3) = 6.82$, $p < 0.001$, 95% CI (0.331, 0.616) but not for motivational interviewing studies (6 studies, 10 effect sizes), $d = 0.282$, $t(4.07) = 1.11$, $p = 0.329$, 95% CI (-0.419, 0.983). Both primary care physician (PCP) involved and not PCP-involved treatment effects were statistically significant with treatment effect estimates of $d = 0.559$, $t(7.25) = 2.45$, $p < 0.05$, 95% CI (0.0234, 1.090) and $d = 0.461$, $t(36.4) = 6.37$, $p < 0.001$, 95% CI (0.315, 0.608), respectively. Additionally, treatment effects for both tele-health and in-person (not tele-health) interventions were statistically significant with $d = 0.411$, $t(19.6) = 3.08$, $p < 0.01$, 95% CI (0.132, 0.690) and $d = 0.484$, $t(22.3) = 7.02$, $p <$

0.001, 95% CI (0.341, 0.627), respectively. Finally, treatment effects were statistically significant for both individual based and non-individual based interventions with d = 0.487, $t(34.8) = 7.06$, $p < 0.001$, 95% CI (0.347, 0.627) and $d = 0.24$, $t(4.45) = 5.05$, $p <$ 0.01, 95% CI (0.113, 0.367), respectively.

Moderator analyses

Study participants' demographic characteristics and study and intervention characteristics were first entered individually into the model to determine if any single moderator explained heterogeneity between reported treatment effect size estimates (which were coded so that higher means better). Results of the single-predictor analysis are presented in Table 7. Treatment outcome was not a moderator, $b = -0.0792$, $t(27.6) =$ -0.776, $p = 0.445$, 95% CI (-0.289, 0.130). Age (mean centered), gender, and race did not moderate treatment effect sizes. However, the coefficient estimate for intercept in the single predictor model of age was statistically significant, $b = 0.462$, $t(43.5) = 6.552$, $p <$ 0.001, 95% CI (0.320, 0.604), indicating that the treatment was statistically significant for a participant at the average age of 45.17 years old. Percentage of married participants moderated treatment effect sizes, $b = 0.006$, $t(5.72) = 3.027$, $p < 0.05$, 95% CI (0.001, 0.011), meaning greater treatment effect is associated with higher proportion of participants who are married.

Treatment effect sizes did not differ significantly among different interventions, different types of control conditions, and across delivery settings (inside versus outside primary care). However, treatment modality (individual versus others) and treatment composition (if an intervention was delivered solely or in combination with another intervention or other techniques) significantly moderated effect size estimates. While subgroup analysis indicated that both individual based and non-individual based

interventions were statistically significant, individual based interventions reported significantly greater treatment effect sizes than non-individual based interventions, b = 0.244, $t(6.13) = 2.91$, $p < 0.05$. Treatment composition revealed that studies which utilized one intervention only reported significantly smaller treatment effects than a primary intervention used in conjunction with other therapeutic approaches or techniques, b = -0.330, $t(34.3) = -2.10$, $p < 0.05$, 95% CI (-0.651, -0.010).

Treatment dosage factors, including number of sessions and minutes per session and their cross product (entire dosage) and treatment duration (number of weeks an intervention lasted) did not significantly moderate treatment effect sizes. Similarly, none of the following factors was a significant moderator of treatment effect sizes: whether an intervention was tele-health based, providers' educational background, and if a physician was involved in the intervention.

While other multiple-predictor meta-regression models did not identify any significant moderators, the model that included all intervention characteristics (outcome type, PCP involvement, telehealth or not, number of sessions, minutes per session, treatment composition, treatment modality, and delivery setting) indicated that delivery setting was a significant moderator holding other factors constant, b = -0.863, $t(6.83) = -2.547$, $p < 0.05$, 95% CI (-1.668, -0.058) (presented in Table 8). Controlling for other intervention characteristics, interventions delivered outside primary care settings reported significantly greater treatment effect size estimates than those delivered inside primary care settings.

CHAPTER SIX: DISCUSSION

Primary care based psychosocial interventions have the potential to effectively alleviate DADs while simultaneously removing service barriers such as stigma of receiving mental health services, the financial burden of commuting to mental health outpatient clinics but despite these potential benefits have not been examined for their overall effectiveness in systematic reviews. Following Cochrane guidelines, this dissertation aimed to address this gap in research by examining the effectiveness of four psychosocial interventions (CBT, PST, MI and SFBT) for treating patients with DADs in primary care. The results of the search for primary studies only found (CBT, PST & MI) that met the study criteria. SFBT has been shown to improve depression and anxiety in other systematic reviews (Gingerich & Peterson, 2013) and has demonstrated psychosocial outcomes in medical settings (Zhang et al., 2018) but this dissertation did not find any primary SFBT studies with specific measures of depression and anxiety that were also delivered in primary care settings. Overall, results of this meta-analysis showed that there was a statistically significant treatment effect that pooled across the three interventions that met study criteria (CBT, PST and MI) for primary care DADs, $d = 0.462$, $p < 0.001$. This overall treatment effect size estimate is considered moderate and indicates that, on average, participants receiving primary care based interventions for DADs are 0.462 standard deviations higher (i.e., more improved) on outcome measures than their counterparts in control conditions. This finding supports the effectiveness of delivering psychosocial interventions for DADs in primary care settings and shows that psychosocial interventions may be feasible for use to manage DADs in primary care.

Importantly, the positive outcomes were impacted by the individual interventions delivered within the primary care settings. Close to three quarters of the primary studies investigated CBT (n = 47, 72.3%), which seems consistent with the psychosocial

intervention literature indicating that CBT remains one of the most extensively utilized, examined, and supported types of psychosocial interventions for treating depression and anxiety (Need a citation for this fact). After combining studies of PST (n = 12), often considered a sub-type of CBT, with other CBT studies, most primary studies (n = 59, 90.8%) included utilized behaviorally oriented interventions for treating DADs in primary care. Significant treatment effects were found for CBT (d = 0.474, $p < 0.001$) and PST (d = 0.450, $p < 0.05$) but not MI (d = 0.282, p = 0.329). This may not be surprising given the nature of MI, which receives most empirical support for its effectiveness in increasing motivation to changes but not necessarily in changing behaviors themselves (Lundahl et al., 2013; Westra, 2004). There is, however, stronger evidence for MI's effectiveness in substance abuse-related behavioral changes (D'Amico et al., 2015; Tanner-Smith & Lipsey, 2015), and MI holds promise for being applied in co-morbid conditions of substance use and DADs.

In comparison, however, it is not surprising to find that both CBT and PST resulted in statistically significant treatment effects and MI did not show significant results for two reasons: (1) Both these behaviorally oriented interventions are well supported for treating DADs in various settings (e.g., Zhang et al., 2018; Weitz, Kleiboer, van Straten, & Cuijpers, 2018) and (2) Both employ change mechanisms that are consistent with the theoretical literature on the etiology of DADs such as negative cognition and affect (e.g., Beck & Haigh, 2014) The results of this meta-analysis suggest that health care professionals may have the greatest impact on DAD's in primary care by addressing clients' behaviors and, simultaneously and consequently, their cognitions (e.g., self-efficacy, positive thinking) and emotions (negative affect) following the change processes of behaviorally oriented interventions that have also been shown to have efficacy in psychotherapy studies (Schwarzer, Lippke, & Luszczynska, 2011).

The overall treatment effect was additionally statistically significant for both depressive disorders and anxiety disorders with no statistically significant difference between the two. Thus, overall study results indicate that the interventions investigated are equally effective for both depressive and anxiety disorders. This finding was expected, first, because each, intervention investigated in primary studies was empirically supported for both depressive and anxiety disorders. Thus, it was reasonable to expect them to be effective when delivered in primary care settings with appropriate modification for each type of disorder. Second, most primary studies (n = 54, 83.08%) investigated treatment for depression with secondary measures for anxiety outcomes reported in the same study. Considering the nature of comorbidity between depressive and anxiety, as well as the overlapping etiology of DADs (negative affect), improvement in depressive disorders (hence in negative affect) is also likely to have a positive treatment effect on anxiety and vice versa.

While the overall treatment effect size identified is promising, study results are most relevant to Caucasians (who composed 64.18% or 73.91% of study participants) and are limited with regard to racial/ethnic minority populations. While there existed low difference in participants' gender and marital status, other participants' demographic information was insufficiently reported, like educational, socioeconomic backgrounds, and other psychosocial factors. Insufficient knowledge of participants' demographic and socio-economic backgrounds, opens the results subject to unknown biases that are relevant to this missing information. Future intervention research in primary care with DADs should pay more attention to health disparities. While health and mental health service gaps are evident in the general population, they are much larger among under represented, ethnic minority populations (Jackson, Knight, & Rafferty, 2010; Williams, & Sternthal, 2010). Intervention research should not only continue its commitment to the

overall goals of science in identifying effective interventions for treating DADs in primary care, it must also address prevalent health disparities that impact the health and well-being of a large numbers of individuals who are at high risk of multi/co-morbid disorders including DADs.

Interventions delivered outside primary care settings were more effective than those delivered inside primary care settings. In other words, if two interventions are the same in other treatment characteristics (e.g., both 40-min, 8 sessions of in-person CBT), the one delivered outside primary care is likely to have greater treatment effect than the one delivered inside primary care. *It is possible that* interventions outside primary care are less stigmatize, thus improving clients' willingness to participate and cooperate. The interventions outside primary care may also be more accessible and focused on mental health outcomes thus easier for clients to receive the full dosage. With greater participation/compliance and higher chance of receiving the full dosage, it is reasonable to expect interventions outside primary care settings being more effective

Both individual and group interventions were found to be effective, but individual interventions had significantly greater treatment effects, $b = 0.244$, $p < 0.05$. One possible explanation for this difference may be the specific population targeted in this study. A major reason people with mental health problems seek help in primary care settings is that they do not want to share their situations with others (often due to stigma). Thus, it is reasonable to expect that they may not respond as well to interventions in a group setting where sharing and openly talking about their mental health conditions are essential to therapeutic improvement. In addition, individual interventions may better target a patient/client's individualized needs. Primary care patients may include those experiencing unique co-morbid or multi-morbid mental and physical health disorders,

which often requires individualized care management. This may help to explain why individual based interventions are significantly more effective than group interventions.

Primary studies also reported significant treatment effect sizes for interventions delivered inside (mostly in-person interventions) and outside (mostly technology-based interventions) primary care settings, indicating robustness of treatment effect regardless of treatment setting. Both technology-assisted and in-person interventions showed significant treatment effects for primary care patients with DADs. The body of literature on technology assisted psychosocial interventions for depressive and anxiety disorders continues to grow (Choi, Hegel, et al., 2014; Choi, Marti, et al., 2014; Khann & Kendall, 2010). An increasing number of clinical trials have endorsed the feasibility and effectiveness of technology assisted psychosocial interventions for DADs (Benavides-Vaello, Strode, & Sheeran, 2013; Eccleston et al., 2014), and federal grants are allowing researchers to examine their sustainability when delivered in various settings across the nation.

This finding is particularly encouraging, especially for underserved populations. While primary care based psychosocial interventions for DADs have greatly reduced treatment barriers for many clients in need, for those individuals from communities with extremely limited health care resources (e.g., rural areas) and significant health disparities, accessing primary care services can still be a significant challenge. Knowing that interventions delivered outside primary care settings for DADs may be as effective as those delivered inside primary care assists in recommending alternatives that can best serve individuals facing chronic health conditions, house bound individuals and/or may have transportation problems. In particular, tele-health interventions are feasible ways to reach patients with DADs and can reduce stigma and privacy concerns associated with treatment in a specialty mental health care. Tele-health can eliminate access barriers

because interventions can be delivered more flexibly at times and locations that best suit patients' needs especially at times when and in locations where patients may need help the most. The result of this study supports an increasing body of empirical literature of pilot RCTs (Gellis & Kenaley, 2014) and multi-site RCTs (Choi et al., 2014) on tele-health. This research need to be carried forward with replications and also with studies that examine specific change mechanisms related to transferring and delivering interventions in tele-health settings.

The results of this study further indicated that an intervention delivered on its own was less effective than an intervention delivered in conjunction with other therapeutic techniques. This is not surprising because the interventions being compared were mostly behaviorally oriented and had elements in common that may be used in relationship to one another and to improve the treatment effect. It is also true that it may be difficult to deliver all components of a CBT intervention in primary care and that briefer interventions are needed. The findings suggest that being able to combine elements from briefer interventions may have a positive and potentially favorable impact on patients. For example, CBT is a highly effective, manualized intervention that is typically delivered in 12 to 14 sessions (Dobson, 2009) but when it is not possible to deliver this many sessions of CBT in various healthcare settings, CBT is often delivered in conjunction with MI with significantly reduced number of sessions (e.g., Barrowclough et al., 2010; Hsieh et al., 2012; Ponsford et al., 2016). These results, however, have implications for treatment fidelity and future studies may want to examine how to effectively combine therapeutic techniques from empirical interventions and further study how common elements from efficacious interventions can be delivered within primary care settings.

Implications for social work practice, research, education and policy

A recently workforce study indicates that a large number of social workers are employed in health care settings making these findings of particular relevance to social work practice (Salsberg, Quigley, Acquaviva, Wyche, & Sliwa, 2018). The results of this meta-analysis show that social workers that are employed in health care have several efficacious interventions that have potential for use in primary care and can impact DADs. The behaviorally oriented interventions that improve cognitions (e.g., self-efficacy, positive thinking) and emotions (negative affect) are most effective suggesting that social workers need to be well trained in these interventions. Being able to combine interventions and use brief interventions are also important to social work practice in primary care. This study also pointed out that while the interventions studied here are potentially relevant and efficacious that there is limited data on their relevance for patients/clients of color and other underserved populations. The majority of study participants were non-Hispanic White making it impossible to ascertain whether the same statistically significant treatment effect for DADs can be applied to other populations. While racial and ethnic backgrounds did not moderate this effect in this study, there were not enough participants to ascertain whether treatment effects are similar or different across racial and ethnic groups. A small number of studies on racial-specific interventions (n = 4) reported a statistically significant treatment effect ($d = 0.89$, $p < 0.05$), however, this result should be interpreted with caution. Social work (and other) practitioners are obliged to incorporate scientifically supported empirical evidence into their daily practices (National Association of Social Workers, 2017) and this study has identified a gap in intervention literature that requires further research to remedy. The underrepresentation of racial and ethnic minority in studies indicates that social work

researchers need to conduct more research with ethnic minority populations with DADs in primary care.

Important also for social work practice is that is that most primary studies investigated depressive outcomes (n = 54, 83.08%), 10 (15.38%) investigated anxiety outcomes, and one investigated co-morbid depression and anxiety. The attention to effective primary care based depression interventions is encouraging, but the disproportionally limited investigation of anxiety disorders and, especially on co-morbid depressive and anxiety disorders, is important to consider when working with patients in primary care because in these practice settings there is a high co-morbidity between depression and anxiety (Hirschfeld, 2011). Social workers and other mental and physical health practitioners encounter patients/clients with depression and/or anxiety in primary care settings, and both disorders are equally prevalent in primary care. Knowing most primary studies focused on targeting depressive outcomes, social work practitioners can have greater confidence in treating depressed clients than those with anxiety disorders with the interventions that were reviewed here. While treating depression may help alleviate symptoms of anxiety or vice versa in primary care patients, whether similar treatment effects can be achieved for co-morbid depression and anxiety is not known suggesting a caution in the implementation of empirical supported treatments and a need educate clients on the limitations of interventions. It also highlights the need for social work practitioners and researchers to engage in more research on interventions within primary care based settings for anxiety and comorbid depression and anxiety.

Other important findings that provide significant implications for social work practitioners, educators, and researchers are that tele-health based interventions are equally effective as interpersonal interventions, and interventions delivered outside primary care settings (most of them are tele-health based) are significantly more effective

than services inside primary care. With recent significant advances in technology and accessibility of technologies, technology has started to play a more important role in social work practice (McCarty & Claney, 2002). Study results may be encouraging and reassuring that tele-health based interventions, which offers much greater flexibility and accessibility, can be as effective as interpersonal psychosocial interventions. It is also promising to know that services delivered outside primary care settings are at least as effect, if not more than, as delivered inside primary care settings. Services delivered outside primary care settings (e.g., home based, community based) may bring psychosocial services to clients that are much less stigmatized, more accessible, and flexible.

For policy makers and social work policy advocators, the most important "take away message" is that integrated mental health services work. With accumulating evidence, this dissertation adds to an already compelling literature that, with appropriate modification and adjustment, psychosocial interventions can effectively address clients' mental health concerns while simultaneously reducing significant treatment barriers including treatment stigma, accessibility, affordability, adherence and compliance, among others. At a policy level, a more supportive health care infrastructure that facilitates integrative mental health services in primary care settings should be encouraged. Equally important, resources should continue to support the training of future mental health workforces across healthcare settings, including primary care settings. One example of such effort is the Health Resources & Services Administration (HRSA) grant that has and is still supporting education programs to train/prepare students to work collaboratively with other healthcare professions.

Across the nation, integrated behavioral health model are expanding vastly in federally qualified health care centers (FQHC), with studies supporting the effectiveness

of this holistic care model of care for patients' bio-psycho-social wellbeing (Fortney et al., 2013; Scharf et al., 2013). Policy makers and advocators are encouraged to establish a pipeline of work forces from education, to training, to practice so that more clients in need can benefit from a holistic care of their wellbeing.

Limitations

Despite the noteworthy findings discussed several limitations exist in this current study. Many of this dissertation's limitations are inherent to systematic reviews and meta-analysis. First, it includes only four interventions, and only three of them met study criteria and could be analyzed. Many other empirically supported psychosocial interventions were not investigated due to time and other resource constraints. Future investigations should examine other interventions. Second, resource constraints allowed for double coding only slightly more than half of the studies. Coding of the other studies may be subject to the author's personal judgement bias and human error. Third, efforts were made to conduct an extensive literature search, it is impossible to ensure that all eligible literature has been included. Fourth, while most included studies have reflected overall good quality and low risk of bias, a few were of poorer quality and had high risk of bias, which may have unknown influence on the overall findings. Fifth, single-predictor meta-regression was conducted for multiple times with a fixed p value of 0.05, which may cause an inflated Type I Error. While this multiple-time statistical test was necessary, its consequence should be noted. Finally, while the number of studies and effect sizes were sufficiently reasonable for meta-regression, moderator analysis in meta-regression uses case-wise deletion when missing values are present. Therefore, in cases of substantial missing values for a specific moderator, statistical power may be

insufficient to identify a significant coefficient when a true moderating effect actually exists.

Overall, all included studies had moderately satisfactory qualities; however, many study either did not report blinding or was unable to blind. Based on risk of bias scores and plot of publication bias, it is reasonably to believe that biases are low among the primary studies included.

Despite its limitations, this dissertation lends strong empirical support for the effectiveness of brief psychosocial interventions for primary care patients' depressive and/or anxiety disorders and suggests avenues for improving these interventions to more effectively reduce the burden of these disorders cause.

Conclusion

This dissertation addressed the epidemic of depressive and/or anxiety disorders (DADs) in the United States by examining different types of psychosocial interventions that are being studied in primary care settings. DADs are prevalent in U.S. primary care settings and the potential of primary care based psychosocial interventions in effectively addressing DADs and removing treatment barriers simultaneously through different delivery methods within primary care is of considerable importance to health care delivery. Considering the overall ratings of study qualities and risk of biases, meta-analysis results for interventions' treatment effects and potential moderators can be interpreted with moderate to high levels of confidence for three of the interventions (CBT, PST, MI) that were analyzed in this study. Even though, no primary SFBT studies were found for this review this intervention has received sufficient empirical support for its effectiveness for both depressive and/or anxiety disorders. Therefore, future research needs to study the effectiveness of SFBT for DADs in primary care settings.

There were also two important limitations discovered in the primary studies that need to be improved on in future research. One limitation is the lack of underserved and ethnic minority patients in the samples and this limits generalizability of these findings to health care practice. Second, lack of outcomes focusing on anxiety and co-morbid anxiety disorders also limits positive interpretations of findings for anxiety disorders in routine practice. This study showed that it is feasible and flexible to deliver the three interventions (CBT, PST and MI) using different methods including in primary care offices and outpatient auxiliary settings and through technology such as telehealth suggesting that psychosocial interventions can be feasibly and effectively integrated into primary care settings.

Table 1. Quality Rating using Jadad Scale for Reporting Randomized Controlled Trials

	Randomization		Blinding		An account of all patients	Total score
	Mentioned randomization	Appropriate randomization	Mentioned blinding	Appropriate blinding	All patients' fate stated	
Asarnow et al. (2005)	1	1	0	0	1	3
Barrett et al. (2001)	1	1	0	0	1	3
Buntrock et al. (2016)	1	1	1	1	1	5
Cape et al. (2016)	1	1	1	0	1	4
Carmody et al. (2013)	1	0	0	0	1	3
Carta et al. (2012)	1	0	1	0	0	2
Chinanda et al. (2014)	1	1	0	0	1	3
Chinanda et al. (2016)	1	1	0	0	1	3
Clarke et al. (2005)	1	0	1	0	0	2
Clarke et al. (2016)	1	1	1	0	1	4
Conradi et al. (2008)	1	0	0	0	0	1
Cramer et al. (2011)	1	1	1	0	1	4
Craske et al. (2011)	1	1	1	1	1	5
De Graaf et al. (2009)	1	0	0	0	1	2
Dwight-Johnson et al.	1	0	1	0	0	2

(2011)						
Forsyth et al. (2015)	1	0	0	0	1	2
Gilbody et al. (2015)	1	1	1	0	1	4
Hange et al. (2017)	1	1	0	0	1	3
Hegerl et al. (2010)	1	1	1	1	1	5
Hoek et al. (2011)	1	1	1	0	1	4
Høifødt et al. (2013)	1	1	1	0	1	4
Katon et al. (2004)	1	0	1	1	0	3
Kay-Lambkin et al. (2009)	1	1	1	1	1	5
Keeley et al. (2016)	1	1	1	1	1	5
Kessler et al. (2009)	1	1	0	0	1	3
King et al. (2013)	1	1	1	0	1	4
Kivi et al. (2014)	1	1	1	0	1	4
Kuyken et al. (2015)	1	1	1	0	1	4
Laidlaw et al. (2008)	1	1	1	1	1	5
Lam et al. (2010)	1	1	1	1	0	4
Lamer et al. (2010)	1	1	1	0	1	4
Leliefeld et al. (2017)	1	1	1	0	1	4
Levesque et al. (2011)	1	0	0	0	1	2
Ludman et al. (2007)	1	0	1	0	1	3

Lynch et al. (2004)	1	0	0	0	0	1
Martin et al. (2015)	1	0	0	0	1	2
McCusker et al. (2009)	1	1	1	0	1	4
Milgrom et al. (2011)	1	0	1	0	0	2
Morrell et al. (2009)	1	1	1	0	1	4
Mynor-Wallis et al. (2000)	1	1	1	1	1	5
Naeem et al. (2011)	1	0	1	0	1	3
Newby et al. (2013)	1	0	1	0	0	2
Nordgren et al. (2014)	1	1	0	0	1	3
Oxman et al. (2008)	1	1	0	0	1	3
Pigeon et al. (2017)	1	1	0	0	1	3
Power et al. (1989)	1	0	0	0	0	1
Power et al. (2012)	1	1	1	1	0	4
Proudfoot et al. (2003)	1	1	0	0	0	2
Proudfoot et al. (2004)	1	1	0	0	1	2
Reynolds et al. (2014)	1	1	1	1	0	4
Richards et al. (2016)	1	1	1	0	1	4
Roy-Byrne et al. (2010)	1	1	1	1	1	5
Schmaling et al. (2002)	1	0	0	0	0	1
Scott et al. (1997)	1	0	0	0	0	1

Serfaty et al. (2009)	1	1	1	0	0	3
Sharp et al. (1998)	1	0	0	0	0	1
Sharp et al. (2004)	1	1	0	0	1	3
Stanley et al. (2003)	1	0	0	0	0	1
Stanley et al. (2009)	1	1	1	0	1	4
Stanley et al. (2014)	1	0	1	0	1	3
Ward et al. (2000)	1	1	0	0	1	3
Wiles et al. (2013)	1	1	1	0	1	4
Wiles et al. (2016)	1	1	1	0	1	4
Williams et al. (2000)	1	1	1	1	1	5
Williams et al. (2013)	1	1	0	1	1	4
Total Score	65	44	39	14	47	

Table 2. Cochrane Collaboration's tool for assessing risk of bias*

	Random sequence generation	Allocation concealment	Blinding of participants and personnel	Blinding of outcome data	Incomplete outcome data	Selective reporting
Asarnow et al. (2005)	+	-	-	-	+	+
Barrett et al. (2001)	+	+	-	?	?	-
Buntrock et al. (2016)	+	+	?	+	+	+
Cape et al. (2016)	+	?	-	+	+	+
Carmody et al. (2013)	+	?	-	-	-	+
Carta et al. (2012)	+	-	-	+	?	+
Chinanda et al. (2014)	+	-	-	-	-	+
Chinanda et al. (2014)	+	-	-	-	-	+
Clarke et al. (2005)	+	?	-	+	+	+
Clarke et al. (2016)	+	-	-	+	+	+
Conradi et al. (2008)	+	-	-	-	?	-
Cramer et al. (2011)	+	-	-	+	-	+
Craske et al. (2011)	+	+	?	-	+	+
De Graaf et al. (2009)	+	-	-	-	+	+
Dwight-Johnson et al. (2011)	+	-	-	-	+	+
Forsyth et al. (2015)	+	?	-	-	+	+

Gilbody et al. (2015)	+	+	-	-	+	+
Hange et al. (2017)	+	+	-	-	+	+
Hegerl et al. (2010)	+	+	-	+	+	+
Hoek et al. (2011)	+	?	?	+	+	+
Høifødt et al. (2013)	+	-	+	+	+	+
Katon et al. (2004)	+	+	-	+	?	-
Kay-Lambkin et al. (2009)	+	+	?	+	+	+
Keeley et al. (2016)	+	+	?	+	+	+
Kessler et al. (2009)	+	+	?	?	+	+
King et al. (2013)	+	-	-	-	+	+
Kivi et al. (2014)	+	+	+	?	+	+
Kuyken et al. (2015)	+	+	-	+	+	+
Laidlaw et al. (2008)	+	+	?	+	-	+
Lam et al. (2010)	+	+	?	+	+	+
Lamer et al. (2010)	+	+	-	-	+	+
Leliefeld et al. (2017)	+	+	+	+	+	+
Levesque et al. (2011)	+	?	-	-	+	?
Ludman et al. (2007)	+	-	-	+	+	+
Lynch et al. (2004)	+	-	-	?	-	+
Martin et al. (2015)	+	-	-	-	?	+

McCusker et al. (2009)	+	+	?	-	-	+
Milgrom et al. (2011)	+	?	-	-	+	+
Morrell et al. (2009)	+	?	-	+	+	+
Mynor-Wallis et al. (2000)	+	+	?	+	?	+
Naeem et al. (2011)	+	?	?	+	+	+
Newby et al. (2013)	+	-	-	-	+	+
Nordgren et al. (2014)	+	?	?	?	+	+
Oxman et al. (2008)	+	+	-	-	?	+
Pigeon et al. (2017)	+	-	?	-	+	+
Power et al. (1989)	+	-	-	?	?	+
Power et al. (2012)	+	?	+	+	+	?
Proudfoot et al. (2003)	+	+	-	-	+	+
Proudfoot et al. (2004)	+	?	-	-	?	+
Reynolds et al. (2014)	+	+	+	+	?	?
Richards et al. (2016)	+	+	?	+	+	+
Roy-Byrne et al. (2010)	+	+	+	+	+	+
Schmaling et al. (2002)	+	-	-	?	?	+
Scott et al. (1997)	+	-	-	-	-	?
Serfaty et al. (2009)	+	?	-	+	+	+
Sharp et al. (1998)	+	-	-	-	-	+

Sharp et al. (2004)	+	?	-	+	+	+
Stanley et al. (2003)	+	-	-	-	-	+
Stanley et al. (2009)	+	+	-	?	+	+
Stanley et al. (2014)	+	+	-	-	+	+
Ward et al. (2000)	+	+	-	-	-	+
Wiles et al. (2013)	+	-	-	?	+	+
Wiles et al. (2016)	+	-	-	?	+	+
Williams et al. (2000)	+	+	?	-	+	+
Williams et al. (2013)	+	+	-	+	+	+
	65 +s	28 +s	5 +s	26 +s	43 +s	58 +s

* “+” low risk of bias; “-” high risk of bias; “?” unclear risk of bias

Table 3. Study characteristics of problem-solving therapy.

Author	Sample†	Demographics††	Control †††	Provider and PCP's role in PST (if applicable)	PST/PST-PC Dosage	Diagnostic Criteria	Depression and/or Anxiety Measures	Setting and disorders †††††
Barrett et al. (2001)	T = 80 C1 = 80* C2 = 81	44.1 years old (SD NR), 36.1% male, 90% white.	MED Placebo	Ph.D-level psychologists. PCP no involvement	6 PST-PC sessions, lasting about 1 hour for the first visit and 30 minutes for subsequent visits.	DSM-III-R, HDRS, PRIME-MD	HSCL-D-20 HDRS	PC, depression
Chibanda et al. (2014)	T = 30 C = 28	24.5 years old (SD = 4.9) % male NR Race NR	MED	Trained Peer Counselor. PCP no involvement	12 sessions (60 mins per session) group PST session which were modeled after a 7-step management plan for depression published earlier (Abbas et al., 1994)	DSM-IV	EPDS	PC, postnatal depression
Chibanda et al. (2016)	T = 286 C = 287	35.1 years old (SD = 11) 13.6% male, Race NR	TAU	Lay health workers, all female PCP no involved	6 sessions of PST. Other information of PST was referred back to the Chibana (2014) study [above]	SSQ-14	GAD-7 PHQ-9	PC, depression and anxiety
Katon et al. (2004)	T = 164 C = 165	58.3 years old (SD = 12), 35% male, 75.4% white.	TAU	Registered nurses in collaboration with the PCP	Medication OR PST-PC, there is a stepped-care algorithm ***	PHQ-9 Did not require diagnostic criteria	SCL-90 depression	Combined, diabetes and depression
Lam et al. (2010)	T = 149 C = 150	71.8 years old (SD = 7.0) 43.14% male, Race NR	AC††††	Primary care physicians	3 sessions of modified PST-PC (Mynors-Wallis et al., 2000), first session 30-45 min. session 2 & 3 20-30 min.	HADS score	HADS (AS), HADS (DS) SF-36 mental	PC, depression and anxiety
Lynch et al. (2004)	T = 9 C1 = 9 C2 = 13	38.5 years old (SD = 13.7), 17% male Race NR	AC††††† TAU	Registered nurses. PCP referral, no other involvement	6 sessions of telephone-based PST (adopted Nezu, Nezu, & Perri, 1989)	PRIME-MD HRSD	PRIME-MD, HRSD BDI, DHP-D-A	PC
Mccusker et al. (2008).	T = 36 C = 32	73.3 years old (SD = 8.6),	TAU	Depression care practitioner supervised	4 sessions PST intervention (60- minute	PHQ-2	SCL-20, SF-12	Combined

		33.8% male Race NR		by (and in collaboration with) PCP	first session, 30 mins for the rest) developed based on IMPACT		SCID	
Mynors-Wallis et al. (2000)	T1 = 80 T2 = 35 C = 36	35 years old (SD = NR), 23% male, 95% white	MED	General practitioner Nurse and General Practitioner (PCP)	6 sessions PST-PC, with first session 1 hr, others 30 minutes	RDC HDRS score	HDRS BDI-I	NIPC
Oxman et al. (2008)	T = 72 C = 69	55.2 years old (SD = 16), 41.8% male, 96.5% white	TAU	Masters level counselor. PCP referral, no other involvement	6 sessions PST-PC, with first session 1 hr, others 30 minutes	DSM-IV, HAM-D, PRIME-MD	HAM-D, MADRS HSCL-D-20	PC
Reynolds et al. (2014)	T = 125 C = 122	36.5 years old (SD = 10.9) 28.7% male 62.3% white	TAU	Social workers and mental health nurses. PCP referral, no other involvement.	6 to 8 sessions PST-PC, with first session 1 hr, the rest 30 minutes	CES-D, DSM-IV; MMSS	SCID/DSM-IV; BDI, SF-12 CIRSG, BSI - Anxiety	PC
Schmaling et al. (2002)	T = 31** C1 = 31 C2 = 30	42.8 years old (SD = 10.7) 39.1% male 88.0% white	MED Placebo	Trained therapists with no further specification. PCP referral, no other involvement.	6 sessions PST-PC, with first session 1 hr, others 30 minutes	DSM-III-TR PRIME-MD, HRSC	HAM-D (17-item) HSCL-D (20-item)	PC
Williams et al. (2000)	T = 138 C1 = 137 C2 = 140	71 years old (SD = 7.0), 58.5% male, 78.2% white	MED Placebo	PhD Psychologists, Social workers, and Psychology Counselors. PCP no involvement	6 sessions PST-PC, with first session 1 hr, others 30 minutes	DSM-III-R, HDRS DSM-IV, PRIME-MD	HSCL-D-20 HDRS	PC

† Sample size: T = Treatment, T_2 = Treatment 2 if applicable, C = Control.

†† Demographic: NR = Not Reported

††† Control: TAU = Treatment as usual, W/NT = Waitlist or no treatment, MED = Medication, Placebo = Placebo medication, †††† AC = Active control (health education video), †††††Active control (stress management),

††††† PC = Primary care setting, NIPC = Not in primary care, Combined = When part of the participants received treatment in primary while others did not

* C1 = medication Paroxetine, C2 = Placebo

** specific breakdown of the numbers was not reported in article, thus assigned arbitrarily

*** 68.7% of participants in the treatment group received PST. Therefore, the authors believed the effect of intervention can be attributed to PST. Because sensitivity analysis that excluded this study did not alter the overall treatment effect, we included and presented this study in final analysis.

**** DCS = Depression Care Specialist

BSI: Brief Symptoms Inventory. **CES-D**: Center for Epidemiology Scale – Depression. **CIRSG**: Cumulative Illness Rating Scale for Geriatrics. **DFD:**

Depression Free Days. **DHP-D-A**: Duke Health Profile-Depression-Anxiety. **EPDS:** 10-item Edinburgh Postnatal Depression Scale. **GAD-7:** Generalized Anxiety Disorder – 7 items. **HAM-D:** Hamilton Rating Scale for Depression. **HADS**: Hospital Anxiety and Depression Scale. **HDRS**: Hamilton Depression Rating Scale. **HRSD**: Hamilton Rating Scale for Depression. **HSCL-D-20**: Hopkins Depression self-report scale. **MADRS**: Montgomery–Åsberg Depression Rating Scale. **PHQ-2**: Patient Health Questionnaire, 2-item. **PHQ-9**: Patient Health Questionnaire, 9-item. **PRIME-MD**: Primary Care Evaluation of Mental Disorders. **RDC:** Research Diagnostic Criteria. **SCL-20**: Hopkins depression symptom checklist. **SCL-90 depression**: Hopkins Symptom Checklist – 90 depression questions. **SCID/DSM-IV**: Structured Clinical Interview for DSM-IV Axis II Personality Disorders (SCID-II). **SF-12**: SF-36 Health Survey 12-item version. **SSQ-14:** The Shona Symptom Questionnaire.

Reference in the table:

Abbas M, Broadhead JC, Mbape P, Khumalo-Sakatukwa G. Defeating depression in the developing word: A Zimbabwean model. *British Journal of Psychiatry, 164*(3): 293 – 296.

Mynors-Wallis LM, Gath DH, Day A, Baker F. Randomised controlled trial of problem solving treatment, antidepressant medication, and combined treatment for major depression in primary care. *British Medical Journal, 320*: 26 – 30.

Nezu, A., Nezu, C., & Perri, M. (1989). *Problem-solving therapy for depression.* New York: Wiley.

Table 4. Study characteristics of cognitive-behavioral therapy.

Author	Sample†	Demographics††	Control †††	Provider and PCP's role in CBT (if applicable)	PST/PST-PC Dosage	Diagnostic Criteria	Depression and/or Anxiety Measures	Setting and disorders ††††
Asarnow et al. (2005)	T = 211 C = 207	17.2 years old, 22% male, 12.7% White	TAU	Psychotherapist in mental health or nursing with master or Ph.D. degree, with 1-day training., PCP no involvement	Manualized CBT with special adaptation. 5 sessions and 50 minutes per session	CIDI-12 CES-D	CES-D MCS-12	PC, depression
Biesheuvel-Leliefeld et al. (2016)	T = 124 C = 124	48.7 years old, 30.2% male, Race NR	TAU	24 counselors (mental health nurses and psychologists) trained. PCP no involvement	8-week supported self-help, manualized preventive CT. 60 minutes per session.	SCID/DSM-IV	QIDS-sr FDSQ-A	PC, depression
Buntrock et al., (2016)	T = 202 C = 204	45.04 years old, 26.1% male, 83.5% White	TAU	Provided guidance to online program. Graduate students and healthcare professionals supervised by a psychologist	Six 30-minute sessions. Mean duration of 6 weeks.	CES-D DSM-IV	CES-D HADS Anxiety BADS-SF PSWQ	NIPC, depression
Carmody et al. (2013)	T = 50 C = 51	67.5 years old, 97% male, 68.4% White	EDU	4 masters level practitioner, experienced, trained	12 telephone sessions over 20 weeks.	MINI	BDI-II	NIPC, chronic pain and depression
Carta et al. (2012)	T = 42 C = 42	42.5 years old, 34.4% male, Race NR	TAU	2 trained psychologists, PCP no involvement provided TAU	12 sessions over 6 months.	DSM-IV-TR	BDI WHOQOL CGI	PC, depression
Clarke et al. (2005)	T = 77 C = 75	15.3 years old, 22.5% male, Race NR	MED	Therapists were trained for CBT but other information not reported	Five to nine 60-minute sessions. 5.3 averaged session	K-SADS-PL DSM-IV	CES-D HDRS CBCL-D YSR-I	PC, depression
Clarke et al. (2017)	T = 106 C = 106	14.6 years old, 31.6% male, 72.2 White	TAU	Minimal master's degree with several years of experience	2 4-session modules. Minutes per session unspecified	DSM-IV-TR	CDRS-R CES-D	PC, depression

				delivering CBT				
Conradi et al. (2008)	T = 41 C1 = 104 C2 = 63	Demographic information NR (Short Report)	TAU	NR	10 to 12 protocolised CBT sessions	NR	BDI	PC, depression
Cramer et al. (2011)	T = 52 C = 21	42.5 years old, 0% male, 87.7% White.	TAU	Trained facilitators who are not professionals. PCP no involvement	Group Intervention: Based on principles from CBT and PST 12 sessions over 10 consecutive weeks and 2 booster sessions at the end	PHQ-9	PHQ-9 BAI	PC, depression
Craske et al. (2011)	T = 503 C = 501	43.2 years old, 28.88% male, 56.57% White.	TAU	6 social workers, 5 nurses, 2 master's psychologist, 1 doc. Psychologist. PCP no involvement	8 individual and online modules, from 8 to 10 weeks, length of session not reported.	DSM-IV	GADSS PDSS-SR SPIN PCL-C	Combined, anxiety
de Graaf et al. (2009)	T1 = 100 T2 = 100 C = 103	44.9 years old, 43.2% male, Race NR	TAU	Computerized program	Online eight 30 min sessions and a ninth booster session	DSM-III-R BDI-II CIDI	BDI-II	NIPC, depression
Dwight-Johnson et al. (2011)	T = 50 C = 51	39.8 years old, 22% male, Race NR	TAU	Generally 5 part-time MSW therapists with various experiences, PCP no involvement	8 core sessions with 2 to 4 booster sessions. 45 to 50 minutes per session.	PHQ-9 MDQ	SCL-D PHQ-9	NIPC, depression
Gilbody et al. (2015)	T1 = 210 T2 = 242 C = 239	39.9 years old, 33.0% male, Race NR	TAU	Computerized program	T1: 15-min intro and eight 50-min session T2: http://moodgym.anu.edu.au	PHQ-9	PHQ-9	NIPC, depression
Hange et al. (2017)	T = 46 C = 31	77 years old, 32% male, Race NR	TAU	Internet-based CBT program	Period defined 12 weeks, seven modules, reported 35 minutes per session on average	MINI MADRS-S	MADRS-S	NIPC, depression
Hegerl et al. (2010)	T = 61 C1 = 83 C2 = 83 C3 = 59	46.4 years old, 31.8% male, Race NR	MED PLC TAU	NR	Group session, 90-min, 10 weeks.	HAMD DSM-IV	HAMD IDS	PC, depression
Hoifodt, et al. (2013)	T = 52 C = 54	36.1 years old, 27.4% male,	W/NT	Pre-designed online modules.	Web-based CBT MoodGYM version (5	BDI-II	BDI-II BAI	Combined,

		Race NR		Therapists' credential not reported. PCP no involvement	modules), Personal therapist support, E-mail support.		HADS	depression
Kessler et al. (2009)	T = 149 C = 148	34.9 years old, 32% male, Race NR	TAU	Experienced trained psychologists in CBT, PCP no involvement (control)	10 sessions with 55 mins per session.	BDI ICD-10	BDI	NIPC, depression
King et al. (2013)	T = 58 C1 = 49 C2 = 23	34.9 years old, 25% male, 89.3% White.	TAU PCM	Accredited counselors and psychologist, PCP no involvement	12, 50-min sessions on a weekly basis	ICD-10 BDI	BDI BSI	PC, depression
Kivi et al. (2014)	T = 45 C = 47	36.6 years old, (SD = 11.3) 34% male, Race NR.	TAU	Licensed doctoral level provider, PCP no involvement	Internet therapy, 7 modules, over 8 to 12 weeks, therapists were in touch weekly for maintenance.	MINI DSM-IV	BDI-II MADRS-S BAI	NIPC, depression
Kuyken et al. (2015)	T = 212 C = 212	52.0 years old, 23.3% male, 99% White	MED	NR, did cover the evaluation of therapists	Eight 2.25-hour group sessions.	DSM-IV	Depression-free-days BDI GRID-HAMD	PC, depression ns ns
Laidlaw et al. (2008)	T = 21 C = 23	74 years old 27.5% male Race NR	TAU	One doctoral psychologist and the rest master level psychologist, PCP no involvement	Averaged 8-session CBT session, ranging from 2 – 7 sessions.	DSM-IV SADS-L HDRS BDI-II	HRSD BDI-II GDS BHS PSWQ WHOQOL	PC, late life depression
Lamers et al. (2010)	T = 96 C = 91	71 years old, 60% male, Race NR	TAU	Registered nurses. PCP part of the training team who trained the nurses	Individual, 2 to 10 visits by nurses, over three months, averaged 4 intervention contact each lasting 60 minutes.	PHQ-9, MINI, HDRS	BDI post SCL-A post BDI 9 month SCL-A 9 month	NIPC, depression and COPD
Leibowitz et al., (2016)	T = 19 C = 120	42.2 years old, 40.2% male, 66.1% White	TAU	Two practitioners, recent graduates, with psychology undergrad degree.	Five weekly 90-min group treatment sessions.	Identified concern about sleeping	PHQ-9 GAD-7	PC, depression and insomnia

Ludman et al. (2007)	T = 198 C = 195	44.4 years old, 24.1% male, 77.4% White	TAU	Master's-level psychotherapists with at least 1 year of experiences, PCP prescribe anti-depressant	Eight core sessions followed by two to four booster sessions over 1 year, 30 to 40 mins per session.	HSCL DSM-IV	HSCL PHQ-9	NIPC, depression
Martin et al. (2015)	T = 36 C = 30	40.6 years old, 25.75% male, Race NR.	TAU	Clinical psychologist, PCP no involvement, provided control intervention	Individual, in person, 12-session, 50-min, weekly	CIDI-LT (Depression and Anxiety)	BDI-II BAI PHQ-9	PC, headache and depression
Milgrom et al. (2011)	T = 23 T_2 = 22 C = 23	31.5 years old, 0% male, 86.76% White.	TAU	Psychologist T1 Nurse T2 PCP control, PCP also is part of the treatment condition	Individual, in person, 6-session over 6 weeks. Length per session not reported	EPDS	BDI-II DASS	PC, postnatal depression
Morrell et al., (2009)	T = 271 C = 147	30.9 years old, 0% male, Race NR	TAU	Providers that were systematically trained, background not reported, PCP no involvement	8 individual session with 60 minutes per session, weekly	EPDS SF-12	EPDS STAI	PC, postnatal depression
Naeem et al. (2010)	T = 17 C = 17	32.9 years old 26.5% male, Race NR	MED	1 psychiatrist 2 psychologists	9 sessions of CBT without session's length reported	ICD-10-DCR	HADS anxiety HADS depression BSI	PC, depression
Newby et al. (2013)	T = 49 C = 60	44.3 years old, 22.2% male, Race NR	W/NT	Internet CBT, therapist assisted, PCP no involvement	6 sessions, length not specified	ICD-10 PHQ-9 GAD-7	PHQ-9 GAD-7 BDI-II	NIPC, anxiety and depression
Nordgren et al. (2014)	T = 50 C = 50	35.4 years old, 37% male, Race NR	W/NT	Pre-design online module, therapist served as a collaborator, PCP no involvement	Internet module, 13 modules across [probably] 13 weeks.	DSM-IV (Anxiety)	CORE-OM BAI MADRS-S	NIPC, anxiety
Pigeon et al. (2017)	T = 13 C = 14	58.46 years old, 89% male, 77.48% White.	TAU	Graduate level psychologist student trained for the study.	Four sessions, one week apart, session 1: 40 mins, session 2: 20 mins, session	PHQ-2 PHQ-9 ISI	PHQ-9	Combined, insomnia and

				PCP no involvement	3: 30 mins, session 4: 20 mins [through phone]			depression
Power et al. (1989)	T = 10 C1 = 10 C2 = 11	34.2 years old, 12.9% male, Race NR	MED PLC	Psychologist therapist (with no further information), PCP offer two additional assessment appointments	4 sessions, 50 minutes per session, and 2 15-minute PC session over 6 weeks.	Initial GP and psychologist assessor evaluation	HRSA	PC, anxiety
Power & Freeman (2012)	T = 65 T_2 = 64 C = 28	36.1 years old, 38.2% male, Race NR	TAU	Therapists with clinical experiences, PCP no involvement in treatment	12 to 16 sessions, followed Beck's manual.	SCID-R	BDI-II	PC, anxiety
Proudfoot et al. (2003)	T = 89 C = 78	44.6 years old, 26.3% male, 74.9% White.	TAU	Computerized program, PCP no involvement	8 therapy sessions, 50 mins per session.	ICD-10	BDI BAI	NIPC, anxiety and depression
Proudfoot et al. (2004)	T = 146 C = 128	43.5 years old, 26.3% male, 80.3% White.	TAU	Computerized program, PCP no involvement	8 therapy sessions, 50 mins per session.	GHQ-12 CIS-R	BDI BAI	NIPC, anxiety and depression
Roy-Byrne et al. (2010)	T = 503 C = 501	43.5 years old, 28.9% male, 56.6% White.	TAU	6 social workers, 5 nurses, 2 master's psychologist, 1 doc. Psychologist, PCP, offer medication	Individual and online modules, from 8 to 10 weeks, length of session not reported.	DSM-IV OASIS	BSI-12 PHQ-8	PC, anxiety
Scott et al. (1997)	T = 24 C = 24	41 years old, 33.3% male, Race NR	TAU	One post-graduate therapist of cognitive therapy, PCP no involvement	6 weeks of CBT with 30 minutes per session.	DSM-III-R	BDI HRSD	PC, depression
Serfaty et al. (2009)	T1 = 70 C1 = 67 C2 = 67	74.1 years old 20.6% male, 75.5% White	TC*** TAU	Accredited therapist with 5 years CBT experiences, PCP no involvement	6 to 8 sessions with possibility up to 12 sessions.	GMSHES BDI-II	BDI-II BAI-II	PC, depression
Sharp et al. (1998)	T = 92 C = 57	Demographic information not reported	MED PLC	Provider information NR, PCP no involvement	9 sessions from 30 to 60 minutes per session.	DSM-III-R	HAM-A SRT FQ-AG	PC, panic disorder

Sharp et al. (2004)**	T1 = 20 (CBT_G) T2 = 31 (CBT_I) C = 19	37.7 years old Gender NR Race NR	W/NT	NR	Group: Twelve 1- hour session over 12 weeks. Individual session exactly as the group.	DSM-IV HAS MADRS	HAM-A SRT MADRS FQ-AG	PC, panic disorder and agoraphobia
Stanley et al. (2003)	T = 6 C = 6	70.6 years old, 16.7% male, 50% White.	TAU	Therapists with no further information	CBT with component of PST. Eight sessions over eight weeks.	PRIME-MD MMSE DSM-IV (SCID-I/P)	PSWQ BAI BDI SCID-GAD	PC, anxiety - GAD
Stanley et al. (2009)	N = 134	64 years old, 21.6% male, 70.2% White.	TAU	3 masters with 2 years' experiences, 1 pre-doctoral with 3 yrs experiences and 1 bachelors with 5 yrs experiences. PCP no involvement	Individual, in person, 7.4 sessions over 12 weeks. Length per session not reported.	MINI DSM-IV	PSWQ GADSS SIGH-A BDI-II	PC, anxiety
Stanley et al. (2014)	T = 76 T_2 = 74 C = 73	66.9 years old, 46.64% male, 78.92% White.	TAU	Mental health provider, Psychologist	Individual, in person and telephone therapy, up to 10 sessions, over 6 months, length of session not specified	DSM-IV (GAD)	PSWQ-A GADSS STAI-T SIGH-A PHQ-8	PC, older adults GAD
Ward et al. (2000)	T = 63 C1 = 67 C2 = 67	36.7 years old, 22.8% male, 89.8% White.	TAU TAU2	Six counselors and three psychologists	Six sessions but maximum of 12 appointments, 50 mins per-session	ICD-10	BDI BSI	PC, depression
Wiles et al. (2013)	T = 234 C = 235	49.6 years old, 27.72% male, 97.87% White.	TAU	Psychotherapist education not reported	Individual, in person, 12-18 sessions and 50-60 minutes per session over 6.3 months.	BDI-II ICD-10	BDI-II PHQ-9 GAD-7 Panic Score	PC, depression
Wiles et al. (2016)	T = 234 C = 235	49.6 years old, 27.72% male, 97.87% White.	TAU	Psychotherapist education not reported	Individual, in person, 12-18 sessions and 50-60 minutes per session over 6.3 months.	BDI-II ICD-10	BDI-II PHQ-9 GAD-7	PC, treatment-resistant depression
Williams et al. (2013)	T = 141 C = 140	41.7 years old, 31.7% male, Race NR	TAU	Online CBT	3 to 4 40-min sessions with on additional session if needed	BDI-II	BDI-II	NIPC, depression

† Sample size: T = Treatment, T2 = Treatment 2 if applicable, C = Control.

†† Demographic: NR = Not Reported
††† Control: TAU = Treatment as usual, W/NT = Waitlist or no treatment, MED = Medication, EDU = Psycho-education, PLC = Placebo, PCM: Primary care management
†††† PC = Primary care setting, NIPC = Not in primary care,
* If does not meet DSM criteria but CES-D cut off score, still included in the trial. ** Treatment 1 = group CBT and treatment 2 = individual CBT. *** TC = TAU + talking control. **** Diazepam = 22, Placebo = 19, Cognitive-behaviour therapy = 21, Diazepam + Cognitive-behaviour therapy = 21, Placebo + Cognitive-behaviour therapy = 18.

ASI: Anxiety Severity Index. **BADS-SF**: Behavioral Activation for Depression Scale – Short Form. **BAI**: Beck Anxiety Inventory. **BAI-II**: Beck Anxiety Inventory II. **BDI**: Beck Depression Inventory. **BDI-II**: Beck Depression Inventory II. **BSI**: Brief Symptoms Inventory. **BSI-12**: Brief Symptoms Inventory, 12 items. **CDRS-R**: Children's Depression Rating Scale – Revised. **CES-D**: Center for Epidemiology Scale – Depression. **CGI**: Clinical Global Impression. **CIDI-(LT)**: Composite International Diagnostic Interview (Life Time Version). **CIS-R**: Clinical Interview Schedule – Revised. **CORE-OM**: The Clinical Outcomes in Routine Evaluation Outcome Measure. **DASS**: Depression and Anxiety Stress Scale. **EPDS:** Edinburgh Postnatal Depression Scale. **FDSQ-A**: Four-Dimensional Symptom Questionnaire – Anxiety. **FQ-AG**: agoraphobia subscale of the Fear Questionnaire **GADSS**: Generalized Anxiety Disorder Severity Scale. **GAD-7**: Generalized Anxiety Disorder 7-item. **GHQ-12**: General Health Questionnaire. **GMSHES**: Geriatric Mental State and History and Etiology Schedule. **GRID-HAMD**: Interview version of the Hamilton Depression Rating Scale. **HADS**: Hospital Anxiety and Depression Scale. **HAM-A**: Hamilton Anxiety Rating Scale. **HAMD**: Hamilton Depression Rating Scale. **HAS**: Hamilton Anxiety Scale. **HDRS**: Hamilton Depression Rating Scale. **HRSA**: Hamilton Rating Scale for Anxiety. **HRSD**: Hamilton Rating Scale for Depression. **ICD-10-DCR**: ICD-10 Diagnostic Criteria for Research. **IDS**: Inventory for Depressive Symptomatology. **ISI**: Insomnia Severity Index. **MADRS**: Montgomery–Åsberg Depression Rating Scale. **MADRS-S**: Montgomery–Åsberg Depression Rating Scale – Self Reported. **MINI**: Mini International Neuropsychiatric Interview. **MMSE**: Mini–Mental State Examination **OASIS**: Overall Anxiety Severity and Impairment Scale. **PHQ-2**: Patient Health Questionnaire -2. **PHQ-8**: Patient Health Questionnaire – 8. **PHQ-9**: Patient Health Questionnaire, 9-item. **PCL-C**: PTSD Checklist – Civilian Version. **PDSS-SR**: Panic Disorder Severity Scale – Self-report. **PRIME-MD**: Primary Care Evaluation of Mental Disorders. **PSWQ**: Penn State Worry Questionnaire. **PSWQ-A**: Penn State Worry Questionnaire – Abbreviated. **QIDS-sr:** Quick Inventory of Depressive Symptomatology – Self Report. **SCID-R**: Structured Clinical Interview for DSM-IV, Research version. **SCL**: Hopkins Symptom Checklist. **SCID/DSM-IV**: Structured Clinical Interview for DSM-IV Axis II Personality Disorders (SCID-II). **SF-12**: SF-36 Health Survey 12-item version. **SOI**: Severity of Illness [for generalized anxiety disorder]. **SRT**: patient-rated Symptom Rating Test. **STAI**: State-Trait Anxiety Inventory. **STAI-T**: The trait subscale of the Spielberger State-Trait Anxiety Inventory. **SIGH-A**: Structured Interview Guide for the Hamilton Anxiety Scale. **WHOQOL**: the Quality of the Life Questionnaire of the World Health Organization.

Table 5. Study characteristics of Motivational Interviewing*.

Author	Sample†	Demographic††	Control †††	Provider and PCP's role in MI (if applicable)	MI Modality and Dosage	Diagnostic Criteria	Measures	Setting ††††
Forsyth et al. (2015)	T = 61 C = 56	Age NR, Gender NR, Race NR	TAU**	Master of Science in Nutrition, PCP no involvement	A motivational interviewing approach is used throughout the consultation. Six visits.	DASS	DASS DASS-depress DASS-anxiety	PC, depression and anxiety
Hoek et al. (2011)	T = 43 C = 40	17.5 years old, 43.3% male, 60% White	TAU	PCP providing MI in the treatment condition	PCP MI + 14 sessions of online treatment	PHQ-A DSM-IV	CESD-10 PHQ-A depressive	NIPC, depression
Kay-Lambkin et al. (2009)	T1 = 35 T2 = 32 C = 30	35.4 years old, 46% male, Race NR	W/NT	CBT + MI, therapist delivered versus computer delivered. Qualification NR other than psychologist, PCP no involvement	Nine sessions of motivational interviewing and cognitive behavior therapy	DSM-IV BDI-II	BDI-II	NIPC, substance and depression
Keeley et al. (2016)	T = 88 C = 80	47.51 years old, 29.05% male, 24.64% White	TAU	PCP is the provider of intervention	At least 4 visits during a period of 36 weeks no other details specified	PHQ-2 PHQ-9 MINI	PHQ-9	PC, depression
Levesque et al. (2011)	T = 174 C = 176	Age range reported, mean and SD NR, 33.4% male, 54.9% White	W/NT	Computer based	Session dosage not reported	PHQ-9	BDI-II	NIPC, depression
Marko-Holguin et al. (2016)	T = 24 C = 20	17.5 years old, 40.91% male, 60% White	TAU	PCP providing MI in the treatment condition	PCP MI + 14 sessions of online treatment	PHQ-A DSM-IV	CESD-10 PHQ-A depressive	NIPC, depression

† Sample size: T = Treatment, T2 = Treatment 2 if applicable, C = Control.
†† Demographic: NR = Not Reported
††† Control: TAU = Treatment as usual, W/NT = Waitlist or no treatment, MED = Medication, EDU = Psycho-education, PLC = Placebo, PCM: Primary care management
†††† PC = Primary care setting, NIPC = Not in primary care,

*Four studies were eligible but sub-studies or follow-ups of the Hoek et al. article – thus, these three studies were excluded. **Attention control with phone calls;
BDI-II: Beck Depression Inventory; **DASS**: Depression Anxiety and Stress Scale; **MINI:** Mini International Neuropsychiatric Inventory; **PHQ-A**: Patient Health Questionnaire – Adolescents; **PHQ-2**: Patient Health Questionnaire – 2 items; **PHQ-9**: Patient Health Questionnaire – 9 items;

Table 6. Overall and Sub-Group Meta-analysis

	Estimate	t (df)	K / N	95% CI	
Overall treatment effect	0.462	7.36 (39)	65 / 198	[0.355, 0.589]	p < 0.001
Outcome type					
Depressive outcomes	0.424	6.21 (43.3)	59 / 113	[0.286, 0.561]	p < 0.001
Anxiety outcomes	0.547	6.1 (11)	32 / 85	[0.350, 0.744]	p < 0.001
Setting of delivery					
Delivered in primary care	0.450	6.77 (23.2)	41 / 139	[0.312, 0.587]	p < 0.001
Delivered outside primary care	0.478	3.31 (18.5)	25 / 59	[0.175, 0.780]	p < 0.01
Types of intervention					
Problem-solving therapy	0.450	2.46 (8.44)	12 / 31	[0.032, 0.616]	p < 0.05
Cognitive behavioral therapy	0.474	6.82 (28.3)	48 / 157	[0.331, 0.616]	p < 0.001
Motivational interviewing	0.282	1.11 (4.07)	5 / 10	[-0.419, 0.983]	
PCP involvement					
PCP involved intervention	0.559	2.45 (7.25)	11 / 25	[0.023, 1.090]	p < 0.05
Non-PCP involved intervention	0.461	6.37 (36.4)	50 / 146	[0.315, 0.608]	p < 0.001
Tele-health or not					
Tele-health based intervention	0.411	3.08 (19.6)	26 / 62	[0.132, 0.690]	p < 0.01
Not tele-health based intervention	0.484	7.02 (22.3)	40 / 136	[0.341, 0.627]	p < 0.001
Treatment modality					

Individual based intervention	0.487	7.06 (34.8)	59 / 180	[0.347, 0.627]	p < 0.001
Non-individual based intervention	0.240	5.05 (4.45)	7 / 18	[0.113, 0.367]	p < 0.01

*** *p < 0.001, ** p < 0.01, * p < 0.05*

K = number of studies, N = number of effect size estimates

Table 7. Single-predictor Meta-Regression Analysis

	Estimate	t (df)	K / N	95% CI	
Outcome Type (*ref: anxiety*) b_0	0.508	5.437 (11.8)	65/198	[0.304, 0.712]	p < 0.001
Depression b_1	-0.079	-0.776 (27.6)	65/198	[-0.289, 0.130]	p = 0.445
Age (mean centered) b_0	0.462	6.552 (43.5)	61/174	[0.220, 0.604]	p < 0.001
Age b_1	-0.001	-0.394 (16.5)	61/174	[-0.009, 0.006]	p = 0.699
Gender (% female) b_0	0.167	0.656 (11.2)	61/167	[-0.393, 0.727]	p = 0.525
% female b_1	0.004	1.130 (12.1)	61/167	[-0.004, 0.012]	p = 0.281
Race (% White) b_0	1.001	2.660 (6.78)	37/97	[0.197, 1.895]	p < 0.05
% White b_1	-0.007	-1.50 (8.27)	37/97	[-0.018, 0.004]	p = 0.170
Marital status (% married) b_0	0.113	0.848 (4.96)	39/109	[-0.231, 0.457]	p = 0.435
% Married b_1	0.006	3.027 (5.72)	39/109	[0.001, 0.011]	p < 0.05
Different interventions (ref: PST) b_0	0.460	2.494 (8.38)	65/198	[0.038, 0.882]	p < 0.05
Cognitive behavioral therapy b_1	0.013	0.068 (11.66)	65/198	[-0.417, 0.444]	p = 0.947
Motivational interviewing b_2	-0.176	-0.562 (6.87)	65/198	[-0.918, 0.566]	p = 0.592
Control group (ref: active control) b_0	0.555	6.82 (7.36)	65/198	[0.365, 0.746]	p < 0.001
Medication only, or placebo, or wait listing b_1	-0.136	-1.18 (15.02)	65/198	[-0.381, 0.109]	p = 0.255
Delivery setting (ref: inside primary care) b_0	0.476	3.293 (18.6)	65/198	[0.173, 0.779]	p < 0.001
Outside primary care or mixture b_1	-0.020	-0.123 (33.1)	65/198	[-0.343, 0.304]	p = 0.903

Treatment modality (ref: non-individual) b_0	0.241	5.120 (5.04)	65/198	[0.120, 0.361]	p < 0.01
Individual based intervention b_1	0.244	2.910 (6.13)	65/198	[0.040, 0.449]	p < 0.05
Treatment composition (ref: combined) b_0	0.69	4.550 (19.9)	65/198	[0.375, 1.011]	p < 0.001
Primary targeted intervention only b_1	-0.330	-2.100 (34.3)	65/198	[-0.651, -0.010]	p < 0.05
Minutes per session (mean centered) b_0	0.389	5.931 (25.3)	45/136	[0.253, 0.524]	p < 0.001
Minutes per session b_1	0.001	0.343 (3.80)	45/136	[-0.007, 0.008]	p = 0.750
Number of sessions (mean centered) b_0	0.466	7.348 (38.7)	64/196	[0.338, 0.594]	p < 0.001
Number of sessions b_1	0.003	0.153 (23.5)	64/196	[-0.040, 0.023]	p = 0.879
Duration (number of weeks) b_0	0.473	7.353 (37.97)	63/193	[0.343, 0.603]	p < 0.01
Duration (number of weeks) b_1	0.003	0.389 (5.57)	63/193	[-0.017, 0.024]	p = 0.712
Dosage (mean centered [min*number]) b_0	0.407	5.769 (20.20)	45/136	[0.260, 0.554]	p < 0.001
Dosage (mean centered [min*number]) b_1	-0.002	-0.964 (7.24)	45/136	[-0.007, 0.003]	p = 0.366
Tele-health intervention (ref: not tele-health) b_0	0.487	7.077 (22.2)	65/198	[0.344, 0.630]	p < 0.001
Tele-health based intervention b_1	-0.079	-0.524 (35.4)	65/198	[-0.383, 0.226]	p = 0.603
Educational background (ref: bachelors) b_0	0.288	1.605 (3.6)	43/124	[-0.233, 0.808]	p = 0.192
Masters b_1	0.275	1.286 (5.54)	43/124	[-0.261, 0.810]	p = 0.250
Doctoral b_2	0.066	0.308 (6.50)	43/124	[-0.444, 0.575]	p = 0.767
Physician involvement (ref: not involved) b_0	0.464	6.398 (36.47)	61/171	[0.317, 0.611]	p < 0.001
Physician involved b_1	0.093	0.386 (9.92)	61/171	[-0.445, 0.631]	p = 0.708

Fidelity measure (ref: no use of fidelity) b_0	0.551	6.120 (19.8)	65/198	[0.363, 0.739]	p < 0.001
Used fidelity measure b_1	-0.210	-1.820 (37.8)	65/198	[-0.445, 0.024]	p = 0.077

**** p < 0.001, ** p < 0.01, * p < 0.05.*

K = number of studies, N = number of effect sizes

Table 8. Multiple-predictor Meta-Regression with Treatment Characteristics as Covariates

	Estimate	t value (df)	K / N	95% CI	p value
Intercept b_0	0.729	1.135 (9.75)	41/111	[-0.707, 2.164]	p = 0.284
Outcome b_1	0.060	0.262 (15.12)	41/111	[-0.426, 0.545]	0.797
PCP involvement b_2	0.192	0.373 (6.86)	41/111	[-1.033, 1.418]	0.721
Tele-health intervention b_3	-0.414	-1.287 (4.71)	41/111	[-1.259, 0.429]	0.258
Minutes per session† b_4	0.026	1.573 (6.99)	41/111	[-0.013, 0.066]	0.160
Number of sessions† b_5	0.045	1.065 (13.17)	41/111	[-0.047, 0.137]	0.306
Treatment composition b_6	0.009	0.242 (6.86)	41/111	[-0.862, 0.879]	0.981
Treatment modality b_7	1.061	1.673 (5.18)	41/111	[-0.553, 2.674]	0.153
Setting b_8	-0.863	-2.547 (6.83)	41/111	[-1.668, -0.058]	0.039
Intervention (PST) b_9	-0.698	-1.423 (9.99)	41/111	[-1.791, 0.395]	0.185

† mean centered variables.

K = number of studies, N = number of effect sizes

Figure 1. Tripartite Model of Depressive and Anxiety Disorders

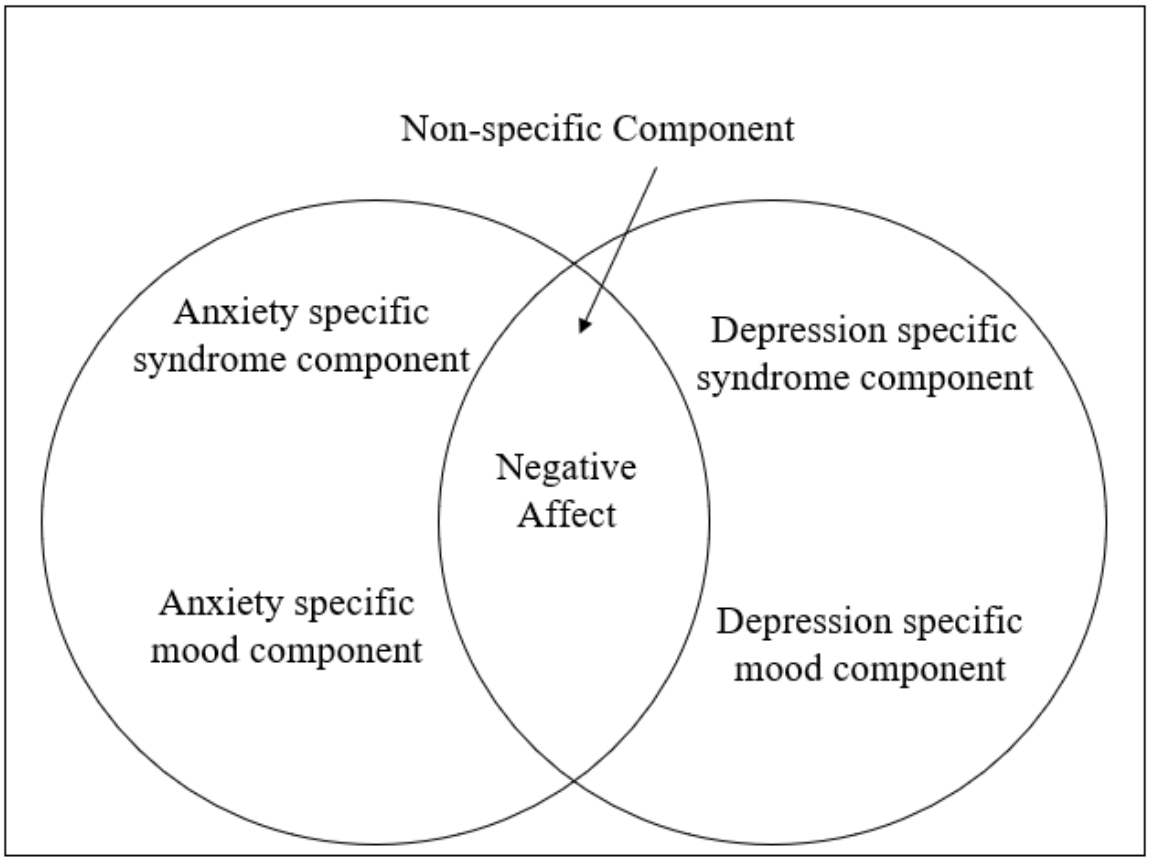


Figure 2. Hierarchical Model of Depressive and Anxiety Disorders

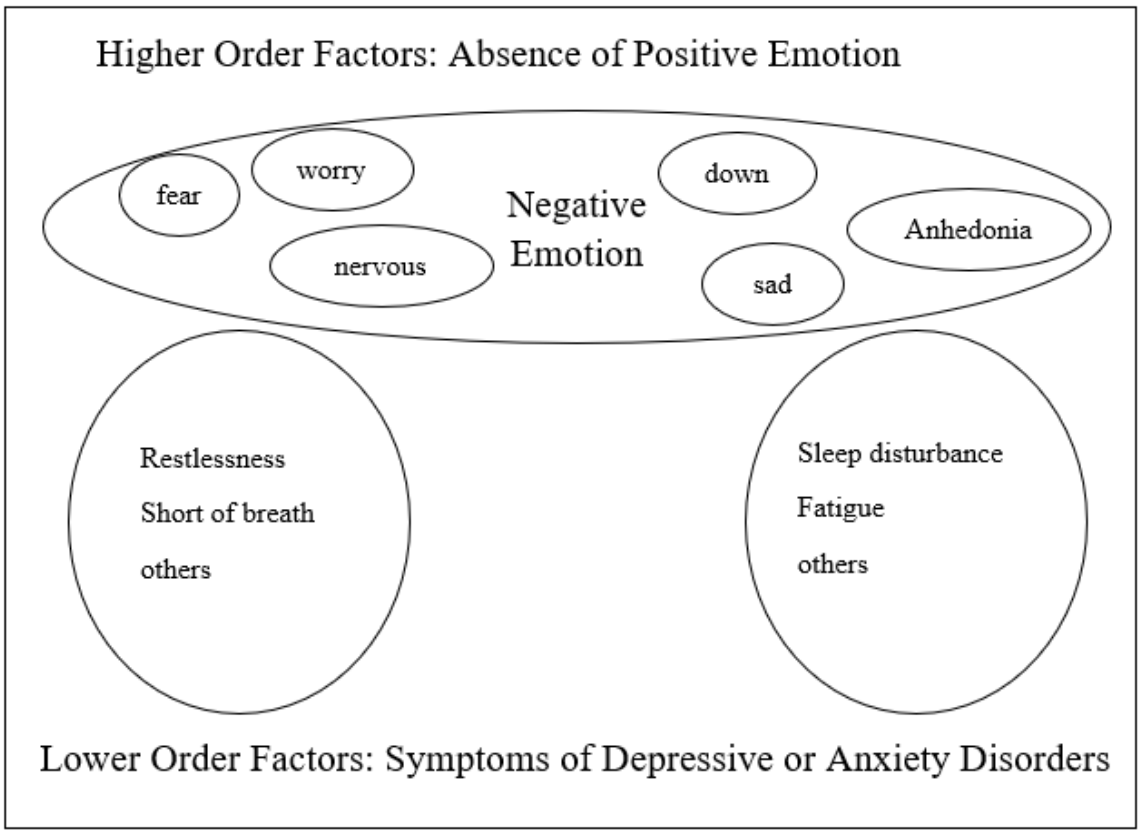


Figure 3. Integrative Hierarchical Model of Depressive and Anxiety Disorders

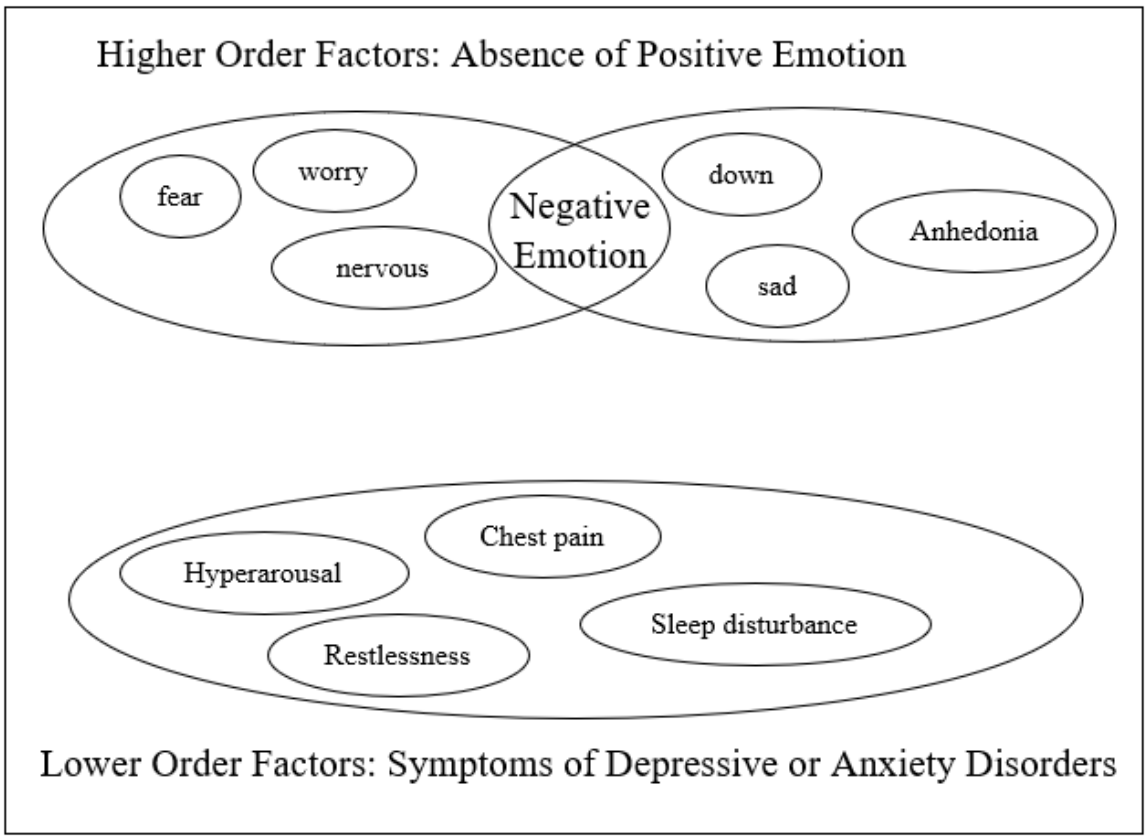


Figure 4. Beck's Generic Cognitive Model

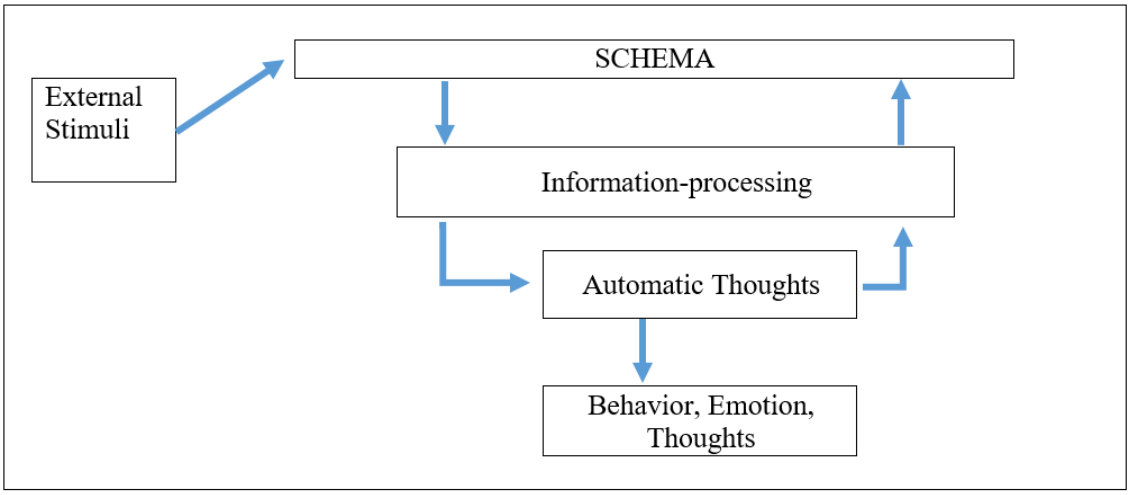


Figure 5. Diagram of the relationship between five dimensions of the revised problem-solving model.

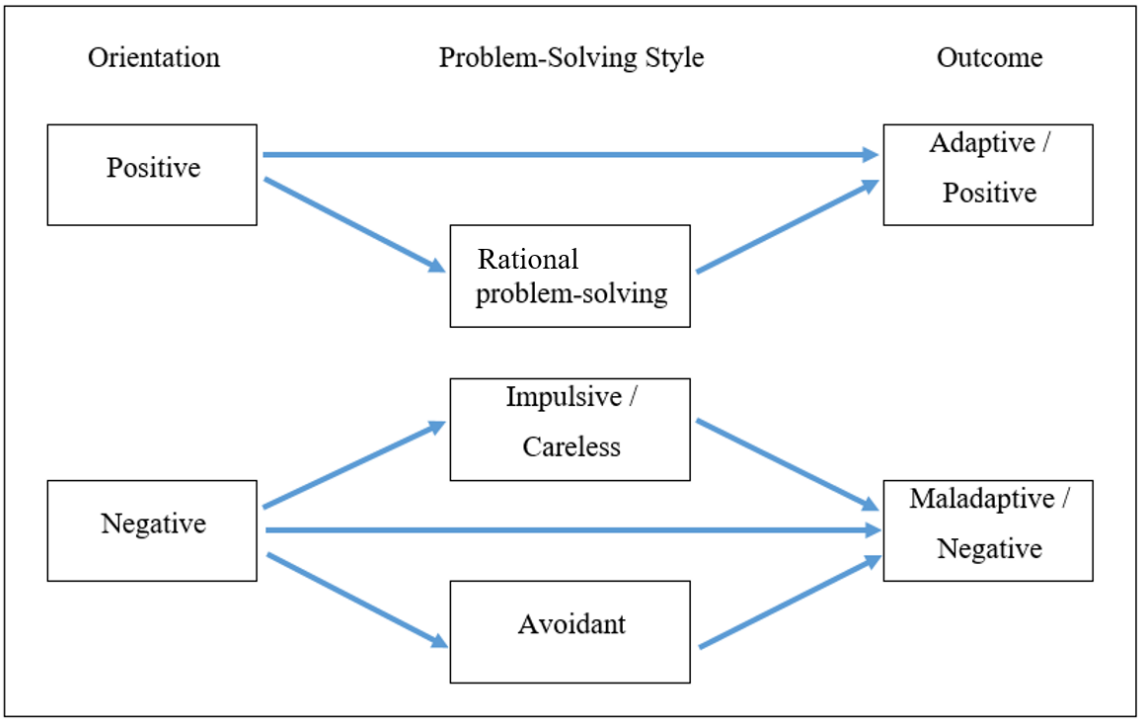


Figure 6. Relational model of life events and individual well-being.

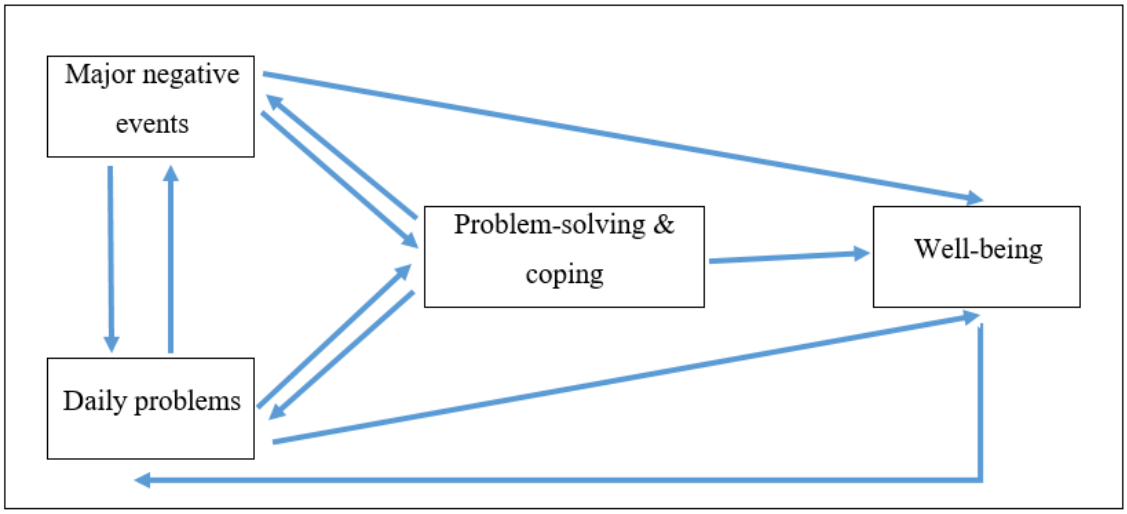


Figure 7. The Transtheoretical Model of Change

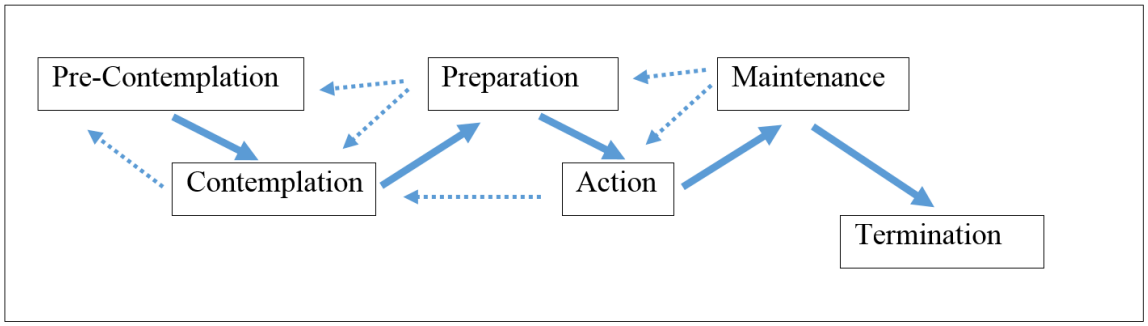


Figure 8. The Change Process of Solution-Focused Brief Therapy (Kim & Franklin, 2015)

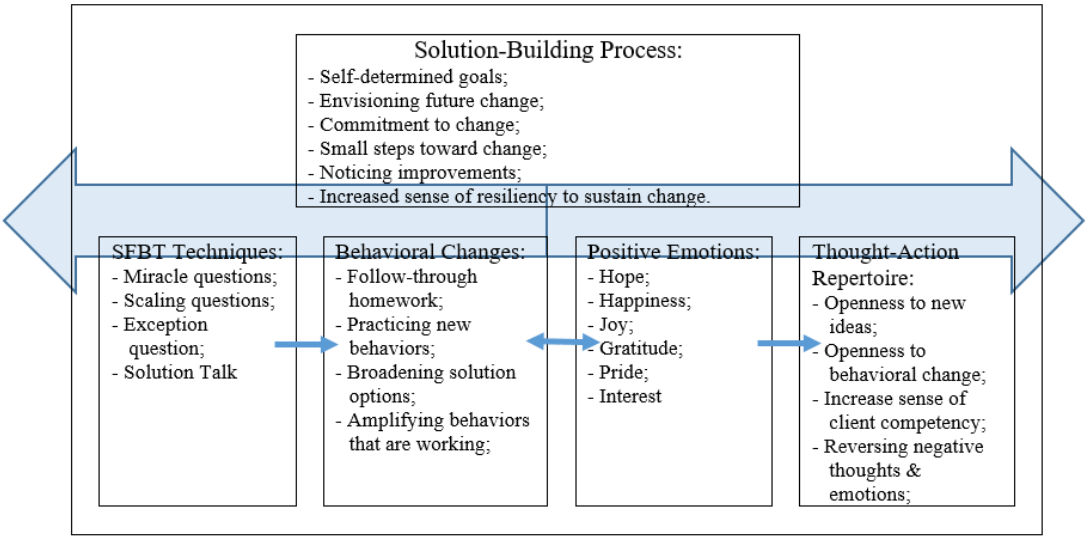


Figure 9. PRISMA Chart of Literature Search Process

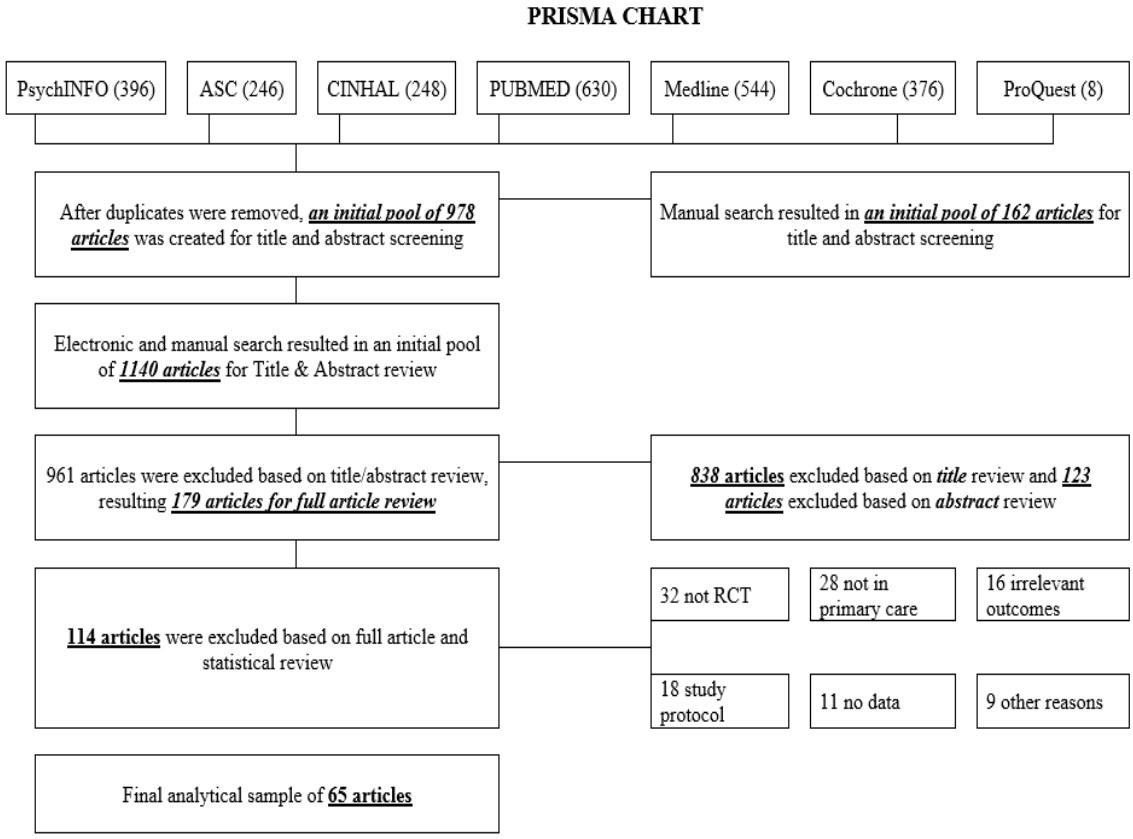
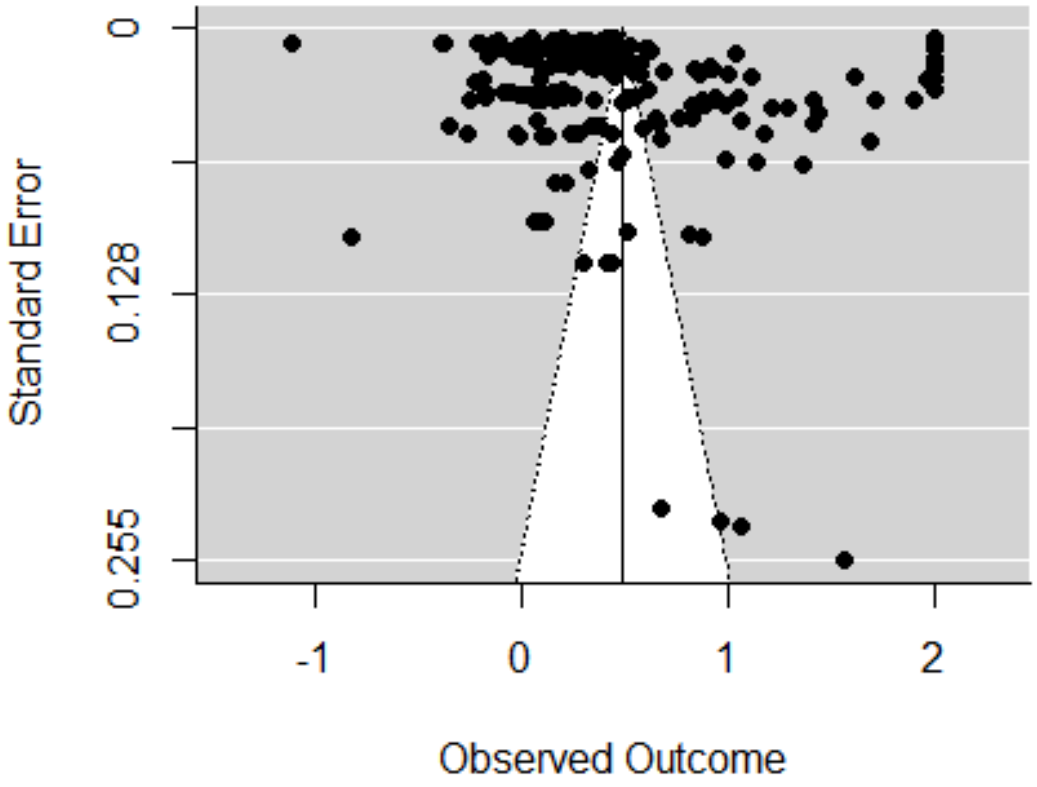


Figure 10. Funnel Plot of Observed Outcome by Standard Error (to Assess Publication Bias)



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