A COGNITIVE BEHAVIORAL THERAPY (CBT) DEPRESSION INTERVENTION IN PERSONS WITH CO-OCCURRING CHRONIC MIGRAINES: A RANDOMIZED CLINICAL PILOT EFFICACY TRIAL

by

ASHLEY J. BRITTON

(Under the Direction of Bernadette D. Heckman)

ABSTRACT

Numerous epidemiological studies have shown a strong co-occurring relationship between chronic headache disorders and psychiatric disorders – particularly depression or anxiety. Epidemiological research has determined that headache disorders are the most prevalent neurological conditions, with significant psychosocial impacts on work, interpersonal well-being and recreational functioning. Prior headache research has repeatedly demonstrated that migraine is associated with significant negative impacts, including reduced quality of life, impaired functioning, and comorbid psychiatric disorders. Specifically, compared with migraine or a psychiatric condition alone, having migraine with co-occurring mental health disorders results in poorer health-related outcomes. Approximately 33 to 50% of chronic headache patients have mild to moderate depression; and traditional headache treatment was proven to be less effective in depressed patients. Antidepressants are well-documented for treatment of chronic daily headache disorders, including migraine and chronic tension headaches. Cognitive Behavioral Therapy was developed in response to changing conceptualizations of both pain and psychological change mechanisms. The psychology of chronic pain is extensive and ranges from

attention control and factors influencing performance of important social roles to aspects of identity construction. This study aimed to assess the effectiveness of a CBT intervention targeted to treat the depression in a community sample with co-occurring chronic headache disorders, with the goal of also improving the head pain severity and frequency. Relative to their counterparts in the Control Condition, individuals with frequent migraines and who also met diagnosis for a depressive disorder demonstrated significant reduction in depressive symptoms, headache days and headache-related disability immediately after undergoing a 4-session cognitive-behavioral intervention that targeted depression.

INDEX WORDS: cognitive behavioral treatment, behavioral therapies for chronic pain, migraine, chronic daily headache, comorbid depression

A COGNITIVE BEHAVIORAL THERAPY (CBT) DEPRESSION INTERVENTION IN PERSONS WITH CO-OCCURRING CHRONIC MIGRAINES: A RANDOMIZED CLINICAL PILOT EFFICACY TRIAL

by

ASHLEY JOI BRITTON

BA, University of Kentucky, 2009

MSEd, University of Miami, 2012

A Dissertation Submitted to the Graduate Faculty of The University of Georgia in Partial Fulfillment of the Requirements for the Degree

DOCTOR OF PHILOSOPHY

ATHENS, GEORGIA

2017

A COGNITIVE BEHAVIORAL THERAPY (CBT) DEPRESSION INTERVENTION IN PERSONS WITH CO-OCCURRING CHRONIC MIGRAINES: A RANDOMIZED CLINICAL PILOT TRIAL OF EFFICACY

by

ASHLEY JOI BRITTON

Major Professor:	Bernadette D. Heckman, PhD
Committee:	Ed Delgado-Romero, PhD
	Brian A. Glaser, PhD

Electronic Version Approved:

Suzanne Barbour
Dean of the Graduate School
The University of Georgia
August 2017

DEDICATION

Throughout my life, there have been two people who have unwaveringly and unconditionally loved, supported and championed for me. My father, John E. Britton, Jr., who frequently reminded me he loved me more than life itself; and my grandmother, Lucia Bacote James, a woman whose grace, diplomacy and generosity I have always aspired towards. I am very grateful for my mother and stepfather, Sheila and Reginald L. Pleasant, Jr., who have also supported me through the trials and tribulations of my journey into adulthood and pursuit of higher education. I would like to dedicate this dissertation to my three parents and grandmother, without whom I would not be the strong and resilient woman I am today. Lastly, I would also like to thank Melodie Turner for being my support away from home as I worked to complete this dissertation while on internship.

TABLE OF CONTENTS

Page

LIST OF TABLES

Page

LIST OF FIGURES

Page

CHAPTER 1

INTRODUCTION

Today, chronic pain is an extremely costly and a prevalent problem nationally and globally (Gatchel, 2013; Hooten et al., 2013). Affecting nearly 50 million adults annually, chronic pain is estimated to cost the US healthcare system $70 billion per year (Hooten et al., 2013). Indirect costs specific to migraine alone cost the US healthcare system an estimated $1.4-17 billion per year (Souza-e-Silva & Rocha-Filho, 2011), with over 80% of persons with migraine reporting experiencing some degree of disability (Merikangas, 2013). However, because tension-type headache (TTH) is much more prevalent than migraine, the societal burden of TTH-related disability is greater; and the corresponding healthcare costs of medications and medical services are 54% higher for TTH than migraine (Rains, Davis & Smitherman, 2015).

Epidemiological research has determined that headache disorders are the most prevalent of all neurological conditions and have a significant psychosocial impact on work, interpersonal well-being and recreational functioning (Shapiro & Goadsby, 2007; Smitherman, McDermott, & Buchanan, 2011). A 1999 epidemiological study of migraine prevalence with 29,727 respondents found twenty-three percent of respondent households had at least one member with migraine with a one-year prevalence rate of 13% in the United States (Lipton et al., 2001). Several US population-based studies determined the national prevalence of tension-type headaches, the most common type of non-migraine headache, was 78% (Heckman & Holroyd, 2006; Jensen, 2003; Rasmussen et al., 1991). A Canadian national population based-study (n= 36,984) estimated the lifetime prevalence of migraine to be 7 to 17% (Jette, Patten, Williams, Becker & Wiebe, 2008).

International studies have found the global percentage of the adult population with an active headache disorder is 46% for headache in general, 11% for migraine, 42% for tension-type headache, and 3% for chronic headache (Souza-e-Silva & Rocha-Filho, 2011); and yet are left without any specific treatment or remain untreated (Smitherman, Burch et al., 2013; Jensen, 2003).

Scope of Impact

Both headache and migraine are common and disabling neurological disorders on national and global scales (Katsarava, Buse, Manack & Lipton, 2012; Jensen & Stovner, 2008; Kalaydjian & Merikangas, 2008), and are considered to be one of the most common reasons for visits to neurologists (Falavigna et al., 2013). Globally, the prevalence of migraine in adults has recently been estimated to be approximately 10-12% (2-19% in men, 6-28% in women) (Falavigna et al., 2013). Similarly, Miller and Matharu (2014) estimated the one-year prevalence rate of migraine to be about 12% (6% male, 15% female) with a lifetime prevalence of approximately 15-18%. Nevertheless, many studies also state that migraine is the most prevalent disabling neurological condition because of both its disabling and transient nature, which disproportionately affects 17 to18% of women nationally – approximately 28 million, nearly three times higher than men; in addition to migraine prevalence rates peaking in the 25 to 55 age group, a period in the life considered to be most productive (Graves, 2006; Lipton et al., 2001; Merikangas et al., 1993; Migraine Research Foundation, 2017; Sammons, 2005; Shapiro & Goadsby, 2007).

Worldwide, headaches are ranked among the top 10 most disabling conditions affecting both men and women (Kalaydjian & Merikangas, 2008). The prevalence of headache disorders in adults is a public health concern, along with the personal and societal impact persons with

headache experience, and is highlighted by its high rate of approximately 46% (Kalaydjian & Merikangas, 2008). Similarly, Jensen and Stovner's 2008 epidemiological study of headache and its comorbidity found that globally, the percentage of the adult population suffering from a headache disorder was 47%. Headaches, especially those that meet criteria for chronic occurrence, are associated with loss of productivity (i.e. missed days of school or work and/or related activities (e.g. presenteeism and absenteeism) (Sammons, 2005; Stewart, Ricci, Chee, Morgan-stein, & Lipton, 2003), with TTH sufferers missing three times more workdays across the population than those with migraine (Rains et al., 2015). Furthermore, in a review of several national surveillance studies examining the impact, prevalence and treatment of migraine and severe headaches, headache (or head pain) was the fourth leading cause of emergency care visits, accounting for 3.1% (Burch, Loder, Loder, & Smitherman, 2015; Smitherman, Burch, Sheikh, & Loder, 2013), and among the top 20 reasons for primary care visits (Curry & Green, 2007; Smitherman et al., 2013).

Affecting nearly 1.5 billion people globally, tension-type headache (TTH) is the most common human health problem after tooth decay in permanent teeth, affecting 69% of men and 88% of women (Barbanti, Egeo, Aurilia & Fofi, 2014; Falavigna et al., 2013). Although prevalence varies according to gender, age and geographical location, earlier TTH epidemiological studies conducted in the US noted the incidence of TTH in the general population to be as follows: 24-37% has TTH attacks several times per month, 10% has TTH attacks weekly, and 2-3% has chronic TTH (CTTH) (Barbanti et al., 2014). Notably, a Danish population study cited by Barbanti and colleagues (2014) reported the productivity impact of TTH (i.e. number of lost workdays) was three times higher than days lost due to migraine.

Lifetime prevalence of headache is estimated to be 90% (93% in men, 99% in women) and is related to physical and emotional stress (Falavigna et al., 2013; Shapiro, 2013).

Both tension-type headache and migraine are broken into two major subtypes differentiated by the attack frequency of the migraine or headache – episodic and chronic (Katsarava et al., 2012). Episodic migraine is defined as headaches occurring less than 15 days per month, and typically lasting less than 24 hours (ICHD-3, 2013; Katsarava et al., 2012). Chronic migraine is defined as headache occurring on 15 or more days per month for greater than 3 months, with features of migraine headache on at least 8 days per month (ICHD-3, 2013; Tepper, 2013; Katsarava et al., 2012). The typical characteristics of migraine headaches are as follows: unilateral location, pulsating quality, aggravation by routine physical activity (e.g. climbing stairs, walking), association with nausea and/or photophobia and phonophobia, and moderate or severe intensity (ICHD-3, 2013).

Episodic tension-type headache (ETTH) may last 30 minutes to 1 week with infrequent attacks (<1 day per month or <12 days per year), and chronic (CTTH; >15 days per month or ≥180 days per year) – including some cases of high-frequency attacks which may be unremitting (ICHD-3, 2013; Rains et al., 2015). TTH is a headache diagnosis characterized by its bilateral location, non-pulsatile (e.g. pressing, band-like, or tightening) pain characteristics with mild to moderate intensity (ICHD-3, 2013; Rains et al., 2015). Rains and colleagues (2015) identified the following characteristics which distinguish TTH from migraine: (1) no association with significant nausea or vomiting (though mild nausea, phonophobia or photophobia may be present), and (2) physical activity does not exacerbate TTH.

There is also an umbrella term which describes headache conditions which occur frequently, daily or near daily with duration of greater than 4 hours (Saper, 2008). This group of

headache disorders is referred to as chronic daily headache (CDH) (i.e. chronic migraine, chronic TTH, new daily persistent headache and hemicranias continua) which occur on 15 or more days per month for at least 3 months (Katsarava et al., 2012; ICHD-3, 2013). An important diagnostic characteristic of CDH is that these headaches are typically a combination of TTH and migraine, with the less severe headaches resembling the TTH definition and the more severe headaches having traditional migraine features; and although some patients may have pure chronic migraine or pure chronic TTH without migraine features, much of those with CDH have the combined headache pattern (Couch, 2010). Saper (2008) noted an important sub-category of CDH, primary chronic daily headache, which indicates that the frequent attacks are not resultant of or associated with secondary causes (i.e. organic conditions). Prevalence rates for CDH in the general population are 3 to 5% (Couch, 2010; Scher, Midgette & Lipton, 2008), with new daily persistent headache consisting of 9-10% of the CDH – and is otherwise indistinguishable from CDH (Couch, 2010) – whereas primary chronic daily headache affects approximately 4 to 5% of the general population (Saper, 2008). Global rates of prevalence of the CDH group of headache disorders have been estimated to occur as follows: 38% for tension-type headache, 10% for migraine, and 3% for chronic headache (Jensen & Stovner, 2008).

Researchers investigating lost productivity in the United States (US) workforce because of common health and pain conditions found that the annual direct and indirect economic costs of headache disorders exceeded $31 billion (Alliance for Headache Disorders Advocacy, 2014). The direct costs consist of the medical costs and social services whereas the indirect costs cause production loss in the economy because of morbidity (Jensen, 2003), with US indirect costs accounting for $1.4-17 billion (Souza-e-Silva & Rocha-Filho, 2011). Migraine is the 12th most disabling disorder in the US, with headache disorders accounting for more than one percent of all

disability and nine percent of all lost labor productivity annually in the US (AHDA, 2014). Previous headache research has repeatedly demonstrated that migraine is associated with significant negative impacts, including reduced quality of life, impaired functioning, and comorbid psychiatric disorders (Hamelsky & Lipton, 2006; Shapiro & Goadsby, 2007; Smitherman, McDermott, & Buchanan, 2011). Specifically, compared with migraine or a psychiatric condition alone, the association of migraine with various mental health disorders results in poorer health-related outcomes (Jette et al., 2008).

Taking into consideration the consequences of recurring inability to work or attend school over a lifetime (e.g. reduced career opportunities, decreased probability of promotion, lower pay and impaired financial security), the cumulative burden of financial loss can be substantial (Stovner, Jumah, Birbeck et al., 2014). For instance, several epidemiological studies have found the following psychosocial impact of migraines: 53% of the study participants reported the severity of their headaches caused substantial impairment in activities and/or required bed rest; in the previous 3 months, 31% missed at least one day or school or work; and 51% reported a reduction by half in school or work productivity (Bigal, Lipton & Stewart, 2004; Lipton et al., 2001). In a Danish population-based study, the total loss of work days per annum due to tension-type headache was 820 days per 1000 employees as opposed to 270 days per 1000 employees because of migraines (Jensen, 2003).

Although migraines are associated with significant lost productivity, lower SES and reduced quality of life (Saper, 2008; Scher et al., 2008; Stewart, Roy & Lipton, 2013; Weeks, 2013), migraines are also associated with high rates of psychiatric comorbidity (Hamelsky & Lipton, 2006). Jensen and Stovner (2008) defined comorbidity as a medical condition existing independently yet simultaneously with another condition; however, other definitions have

implied causality between certain comorbid disorders. Research in community and clinical samples of headache patients suggests a bidirectional association between increased headache diagnosis and depression/anxiety (Lanteri-Minet, Radat, Chautard & Lucas, 2005; Smitherman, McDermott, & Buchanan, 2011). Frediani and Villani (2007) clarified that this 'bidirectional relationship' indicates an association which "seems to arise from the two conditions reciprocally affecting each other … rather than resulting from a one-way action (p. S163)" which strikes the possibility that mood disorders are secondary to chronic migraine attacks. Bidirectional and cross-sectional associations between migraine and various somatic and psychiatric conditions have been reported within migraine and headache literature.

For example, in a 2004 study, with participants from a mixed model health maintenance organization, the overall prevalence of major depression was 28.1% for migraine, 19.5% for probable migraine, 23.9% for migraine and probable migraine polled together, and 10.3% for the control group (Scher, Bigal & Lipton, 2005). Furthermore, compared with controls, the prevalence of major depression was elevated in all migraine groups on both crude and adjusted (by age, sex, education) prevalence ratios (Scher et al., 2005). Likewise, large-scale population based studies estimated that persons with migraines are approximately 2.2 to 4.0 times more likely to have a comorbid diagnosis of depression, an odds ratio of 3.5 to 5.3 for a comorbid diagnosis of generalized anxiety disorder, an odds ratio of 3.7 for comorbid panic disorder (Hamelsky & Lipton, 2006). Breslau et al.'s 2003 two-year longitudinal population-based study measured the bidirectional associations of migraine, severe non-migraine headache and depression considering the extent to which headache increased the incidence risk of depression and vice versa (Scher, Bigal & Lipton, 2005). Results found that participants with baseline depression increased their relative risk of migraine incidence (RR 3.4, 1.4-8.7), but not with

other severe non-migraine headaches; and the incidence risk of depression was higher for participants with baseline migraine (RR 5.8, 2.7-12.3) and marginally higher for those with severe non-migraine headache (RR 2.7, 0.9-8.1) (Scher, Bigal & Lipton, 2005).

Migraine has long been recognized as associated with a characteristic set of psychiatric disorders, as seen in clinical and general population studies with adults, the most relevant disorder for this study being major depression (Jette et al., 2008; Lanteri-Minet, Radat, Chautard & Lucas, 2005; Torelli, Lambru & Manzoni, 2006). Notably, research has found the risk of suicide to be higher in patients with migraine (Jette et al., 2008). The significant association between migraine and major depression has been also observed in several studies conducted on adolescents selected from community-based populations (Lanteri-Minet, et al., 2005).

While the literature often reports comorbidity of these psychiatric disorders and migraine, data from clinical populations has confirmed this same comorbidity to be present in patients with CTTH and CDH (Torelli, Lambru & Manzoni, 2006; Heckman & Holroyd, 2006). In fact, the rate of psychiatric disorders in patients with CTTH has been found to be equal to and sometimes greater than the rates found in migraine patients (Heckman & Holroyd, 2006). Specifically, studies with clinical samples reported psychiatric comorbidity in 40-90% of patients with primary CDH; and a reported 40-50% of patients with CTTH treated in primary care settings and up to 84% of patients with CTTH treated in specialty clinics (Lipchik & Penzien, 2004). The bi-directionality of this association has been demonstrated both when looking at the occurrence of affective symptoms in chronic headache and migraine patients and conversely when looking at migraine occurrence in subjects with depression (Lanteri-Minet et al., 2005).

Pathophysiology of Depression and Migraine

Research on the etiology of migraine and psychiatric disorders, particularly major depression, has found common underlying pathologic mechanisms (Nimnuan & Srikiatkhachorn, 2011; Frediani & Villani, 2007). Dysregulation of or imbalances in the neurotransmitters serotonin, norepinephrine, and dopamine are strongly associated with depression (Shulman, 2013). Serotonin is involved in regulating many important physiological functions – including sleep, sexual behavior, aggression, eating, and mood – with current research suggesting that a decrease in serotonin production can cause depression in some people (Nimnuan & Srikiatkhachorn, 2011; Nemade, Staats Reiss, & Dombeck, 2007). Another line of research investigated relationships between norepinephrine, stress, and depression suggested that the deficiency of norepinephrine in certain areas of the brain was responsible for creating depressed mood. Norepinephrine aids our bodies recognizing and responding to stressors. Research suggests that people who are susceptible to depression may have a norepinephrinergic system that does not efficiently handle the effects of stress. Finally, dopamine plays a key role in regulating our drive to seek out rewards and obtain a sense of pleasure from activities or people prior to becoming depressed. (Nemade, Staats Reiss, & Dombeck, 2007).

Graves (2006) noted that the vascular changes associated with migraines are actually responses to neural changes, rather than the cause as previously believed. The pathophysiology of migraine, involving the trigeminal nerve, can be described as the release of neuroinflammatory peptides in response to stressors with two consequences (Graves, 2006). First, serotonin is released, causing vasoconstriction and vasodilation (pain around the temples and eyes); second, is the sensitization of the trigeminal system (progressing from peripheral to central sensitization) resulting in cutaneous pain (of the skin or scalp) from an innocuous

stimulus which typically is not painful. (Graves, 2006). The trigeminal nerve is the largest of the cranial nerves, primarily responsible for sensation in the face, and because of this sensory information is processed via parallel pathways in the CNS, it is thought to be involved in the cause of migraine (NHS, 2010). In a clinical article reviewing the psychiatric comorbidities of migraine, Nimnuan and Srikiatkhachorn (2011) explained that the dysregulation of aminergic neurotransmitters in the central nervous system, specifically serotonin and dopamine, is the most likely hypothesis for explaining the comorbidities. Hamel (2007) cited evidence that suggests that a state of low serotonin "facilitates activation of the trigeminovascular nociceptive pathway, as induced by cortical spreading depression (p.1295)" – the cause of both migraine pain and migraine aura.

The clinical evidence of this bidirectional hypothesis is found in the medications that act on serotonin (i.e. SSRIs and SNRIs) which can be used effectively, both in the prophylaxis of migraine and in the treatment of depression (Nimnuan & Srikiatkhachorn, 2011; Frediani & Villani, 2007; Baskin et al., 2006). These antidepressants work for both conditions because they enhance norepinephrine or serotonin transport through the inhibiting reuptake in the synaptic cleft (Baskin et al., 2006).

STATEMENT OF THE PROBLEM

The shared pathophysiology of depression with chronic headache and migraine sufferers has been found to be the rule rather than the exception. Psychiatric disorders, including bipolar and depression, have consistently been associated with migraines (Breslau, 1998; Breslau et al., 1994, Lipton et al., 2000; Merikangas et al., 1993). The comorbidity of these affective disorders and neurological conditions has been observed and studied extensively in various experiments including epidemiologic studies, clinical samples, family and twin studies, and longitudinal

studies (Beghi et al., 2007; Bigal & Lipton, 2004; Hamelsky & Lipton, 2006; Lanteri-Minet, et al., 2005). For instance, an earlier population-based study found greater disability was experienced among migraine sufferers with comorbid depression than among participants without comorbid headache disorders (Lanteri-Minet, Radat, Chautard & Lucas, 2005). Due to the high prevalence of comorbidity between neurological and psychiatric disorders, it is imperative that practitioners and physicians maintain diagnostic vigilance and consider both types of disorders when formulating treatment plans (Hamelsky & Lipton, 2006).

In a review of epidemiological migraine studies from the previous 15 years, Lipton and Bigal (2007) noted that 98% of patients in population studies take medications for their headaches. Of the 98%, 57% reported adequately self-treating their migraines with over-the-counter (OTC) medications while 41% reported taking prescription medications alone or in combination with OTCs (Lipton & Bigal, 2007). However, survey data indicated only 29% of migraine sufferers reported being "very satisfied with their usual acute treatment" (Lipton & Bigal, 2007). Moreover, migraine and tension-type headache have been found to be a clinically progressive disorder evolving from episodic to chronic (Lipton & Bigal, 2007).

A 2003 cross-sectional, population-based epidemiological study conducted by Scher and colleagues (2003) aimed to better understand the prognosis and etiology of chronic daily headache (CDH) and describe the factors predictive of onset or remission of CDH in an adult sample. The researchers interviewed 1,932 participants, 1,134 of which were potential CDH cases (180+ headache days per year) and 798 controls (2 to 104 headache days per year). Their results yielded the identification of specific and statistically significant risk factors associated with the development of CDH, including the following: attack frequency, medication overuse, low socioeconomic status, obesity, stressful life events, snoring and head injury (Scher et al.,

2003). Further, excessive use of OTCs in combination with caffeine, narcotics and barbiturates were associated with an increased risk of developing CDH; however, aspirin was determined protective (Lipton & Bigal, 2007; Scher et al., 2003).

While epidemiological studies have consistently confirmed headache patients are at a significantly higher rate of also suffering from a psychiatric disorder, a smaller but growing body of literature implicates psychiatric comorbidity as a risk factor for headache chronification (Smitherman, Maizels, & Penzien, 2008; Scher, Lipton & Stewart, 2002). The term "chronification" is used primarily in the pain literature in reference to the process by which episodic pain becomes chronic (Tepper, 2013; Scher et al., 2008). Specifically, many patients with chronic headache conditions endorse higher levels of depression than do their non-headache counterparts (Smitherman et al., 2008). In terms of societal cost, the comorbidity of chronification is associated with poorer headache prognosis and treatment satisfaction, increased headache-related disability and substantially higher medical costs and healthcare utilization (Smitherman et al., 2008; Jette et al., 2008; Shapiro & Goadsby, 2007). Although the empirical research about headache chronification recognition and management is quite limited, this evolved conceptualization of headache as a progressive disorder – with modifiable and identifiable risk factors – is consistent with other findings (Scher et al., 2008; Smitherman et al., 2008).

Although antidepressants are a common treatment of chronic migraine and chronic daily headache, why these medications are useful in treating head pain requires further explanation; however, Sammons' 2005 study indicated that the SRIs did not appear to "confer any specific benefit in the treatment of head pain, and indeed they may be less effective than older agents". Furthermore, in a meta-analysis conducted by Tomkins et al. (2001), the association between

improvement in head pain and depressive symptoms in headache patients treated with anti-depressants was present in some but not the majority of studies examined (Baskin, Lipchik & Smitherman, 2006; Sammons, 2005). Across studies validating the relationship between head pain and depression improvements, the strength of the relationship was deemed modest at best (Sammons, 2005). In an updated review of statistics from government health surveillance studies on the prevalence and burden of severe headache and migraine in the US, Burch and colleagues (2015) found in 2010 while triptans were administered in 1.5% of emergency department visits for headache, 35% of these visits administered opioids. In the earlier statistical review of national surveillance studies, Smitherman and colleagues (2013) found triptans accounted for nearly 80% of the prescribed migraine analgesics at primary care office visits in 2009.

Combined pharmacological and psychological treatment interventions have been poorly studied in head pain (Sammons, 2005). Although epidemiologic studies note that approximately 38% of migraineurs need preventive therapy, only 3% to 13% currently use it (Lipton, Bigal, Diamond, Freitag, Reed, Stewart, 2007; Smitherman et al., 2013). This gap between migraine prevalence and appropriate treatment highlights the impact of migraine (and severe headache) as a major public health problem that will persist until there is an improvement in recognition of headache burden and adequate provider assessment (Smitherman et al., 2013). In 2001, Holroyd and colleagues conducted one of few combined treatment trials comparing the efficacy of a tricyclic anti-depressant (TCA, either amitriptyline or nortriptyline), a home-based cognitive-behavioral therapy stress management regimen, or placebo in the management of chronic daily muscle tension headache. In patients with CTTH and comorbid mood disorders, Holroyd and colleagues (2001) found both tricyclic treatment and CBT demonstrated a greater treatment (Baskin et al., 2006).

PURPOSE OF THE STUDY

Strong epidemiological evidence suggests that headaches, particularly migraine, are associated with an increased risk of comorbidities including physical conditions (e.g. musculoskeletal conditions, immune and inflammatory diseases, asthma, rheumatoid arthritis, cardiovascular disease and stroke) (Merikangas, 2013; Kurth et al., 2011; Kalaydjian & Merikangas, 2008). A national US comorbidity survey replication study found the association with obesity to be attributable to headache in general rather than migraine, whereas hypertension was the only chronic physical condition specifically associated with migraine rather than general headache (Merikangas, 2013). Notably, among a group of the most disabling chronic disorders, a World Health Organization survey rated severe migraine equal to psychosis, dementia and quadriplegia; and profoundly interpreted this ranking to indicate that one-day living with severe migraines is considered to be as disabling as living a day with quadriplegia (Miller & Matharu, 2014).

Moreover, compared to their headache-free counterparts, adults with chronic headache conditions are more likely to rate their health as poor or fair, endorse physical and psychological limitations, and seek healthcare four or more times per year (Kalaydjian & Merikangas, 2008). Additionally, negative health perception and health utilization were more strongly influenced by comorbid psychological disorders rather than physical conditions – which underscore the importance of the implications for the clinical evaluation and treatment of headache in the general population (Kalaydjian & Merikangas, 2008). The importance of epidemiological studies in headache science has been increasingly recognized, thereby improving opportunities for treatment and prevention (Stovner et al., 2014).

Numerous community and population-based epidemiological studies have shown a strong relationship between migraine and chronic daily headache and psychiatric disorders, specifically depression (Beghi et al., 2007; Bigal & Lipton, 2004; Breslau et al., 1994; Falavigna et al., 2014; Fumal & Schoenen, 2008; Hamelsky & Lipton, 2006; Heckman & Holroyd, 2006; Jette et al., 2008; Lanteri-Minet et al., 2005; Lipton et al., 2000; Merikangas et al., 1993; Smitherman et al., 2011; & Torelli et al., 2006). Although depression is strongly associated with head pain (i.e. migraine, TTH, CDH), use of antidepressants in management of depressive symptoms has not predicted improvement in head pain (Sammons, 2005), particularly with TTH which is more complex and more difficult to treat (Barbanti et al., 2014; Fumal & Schoenen, 2008). Comprehensive treatment plans which address psychosocial stressors and related triggers are essential to effective treatment and management of chronic pain.

Cognitive Therapy (CT) and Cognitive Behavioral Therapy (CBT) are forms of psychotherapy pioneered by Dr. Aaron Beck while originally testing psychoanalytic concepts about the treatment of depression (Beck, n.d.). Compared to other forms of psychotherapy, cognitive therapy is usually more present oriented, time-limited, and solution focused (Goldberg, 2012; Beck, n.d.). CBT teaches patients specific skills that they can use for the rest of their lives – specifically, through identifying distorted thinking, modifying beliefs, relating to others in different ways, and changing behaviors (Ehde, Dillworth & Turner, 2014; Goldberg, 2012; Sun-Edelstein & Mauskop, 2012; Morley, 2011; Gatchel & Rollings, 2008; Beck, n.d.).

The aim of the study is to determine the effectiveness of an evidence-based intervention on headache frequency, severity and level of disability in a community sample with chronic headache disorders and depression, by targeting only the depression. The variables of interest are reduction of depressive symptoms and headache symptoms over a 4 to 8-week period. This study

aims to provide additional evidence for the utilization of multidisciplinary treatment interventions for chronic pain conditions. Finally, this study will contribute to the literature on the efficacy of behavioral treatment of chronic head pain, specifically with the psychosocial intervention of cognitive behavioral therapy.

THEORETICAL FRAMEWORK

The framework this study will operate from is the biopsychosocial model (figure 1), and serves as the replacement of the earlier and outdated biomedical approach to pain (Gatchel, 2004 & 2013; Gatchel & Howard, 2015). This model addresses the person holistically in all of their complexity – which includes conceptualizing physical and biological factors, psychological state and beliefs, in addition to the influence of family, social and work/school environment (Hooten et al., 2013). Despite the domination of the biomedical model in the conceptualization and treatment of health over the past three centuries, it has failed to explain why some people develop an illness under the same stressors and exposure while others do not and it does not account for diseases with genetic markers not being able to predict or guarantee the onset of that disease (Test & Test, 2014; Gatchel, Peng, Peters, Fuchs & Turk, 2007).

The biopsychosocial model informs the process of diagnosis, management and intervention decisions, and the measurement of outcomes (Gatchel & Howard, 2015; Powers et al., 2006), particularly in treating chronic pain and headache disorders. Resultant of significant advances in understanding the etiology of chronic pain over the past decade, the biopsychosocial model has proved to be the most widely accepted and most heuristic perspective to the understanding and treatment of chronic pain (Gatchel & Howard, 2015; Gatchel et al., 2007). Where disease is conceptualized as an "objective biological event" and defined as an altered condition caused by the disruption of normal physiological systems, illness is conceptualized as

the "subjective experience" associated with the disease state – and represented by the unique interaction among the biological, psychological and social factors (Gatchel & Howard, 2015; Turk & Monarch, 2002). Thus, chronic pain must be viewed as an illness that can only be managed not cured, as with most chronic illnesses (i.e. diabetes mellitus, essential hyptertension, asthma, etc.) (Gatchel & Howard, 2015). Consequently, this heuristic approach is directed not at the disease but rather towards the illness, while focusing on the diversity of the individual pain differences in the overall pain experience (Gatchel, 2015).

The hallmark of this model is the emphasis on the dynamic and complex interactions among physiological, psychological and social factors that often perpetuate and may worsen the clinical presentation (Ehde et al., 2014; Gatchel, 2013; Test & Test, 2014). Given this advancement in the conceptualization and treatment of the human condition, the biopsychosocial model has become regarded as a more effective means of evaluating and treating chronic pain patients (Gatchel, 2013; Powers et al., 2006). Each individual experiences pain uniquely; and as such an evaluation of such interactions needs to be conducted on an individual basis to allow for the tailoring of treatment plans which meet the specific needs of each patient (Gatchel, 2013; Hooten et al., 2013, Powers et al., 2006).

RESEARCH QUESTION & HYPOTHESES

The research question of interest is: If the depression is treated with a psychosocial intervention, in patients with comorbid chronic headaches, does the head pain also improve? Consistent with the literature on the use of CBT for chronic pain, and more specifically chronic headache disorders, this study will implement empirically supported techniques including relaxation, self-instructions (e.g. imagery, motivational self-talk), changing maladaptive beliefs (about depression and pain), development of coping strategies (e.g. minimizing catastrophizing

and self-defeating thoughts, behavioral activation, increasing self-efficacy & assertiveness), with the goal being to effectively reduce depressive symptoms. Specifically, using a pilot randomized clinical trial design, the following hypotheses were examined:

Hypothesis 1: Relative to their counterparts in the Control Condition, individuals with frequent migraines (based on ICHD II criteria, 2004) and who also met diagnosis for a depressive disorder (based on DSM-5, 2013) will demonstrate significant reductions in depressive symptoms, headache days and headache-related disability immediately after receiving a cognitive-behavioral intervention for depression. Hypothesis 2: Relative to their counterparts in the Control Condition, the psychosocial intervention will positively and significantly impact participants' pain management self-efficacy and improve participants' quality of life. Specifically, the intervention participants will demonstrate significantly increased self-efficacy and greater internal locus of control (as opposed to external LOC) along with the depressive and headache symptom reduction – which would continue to help participants with symptom management after conclusion of the study.

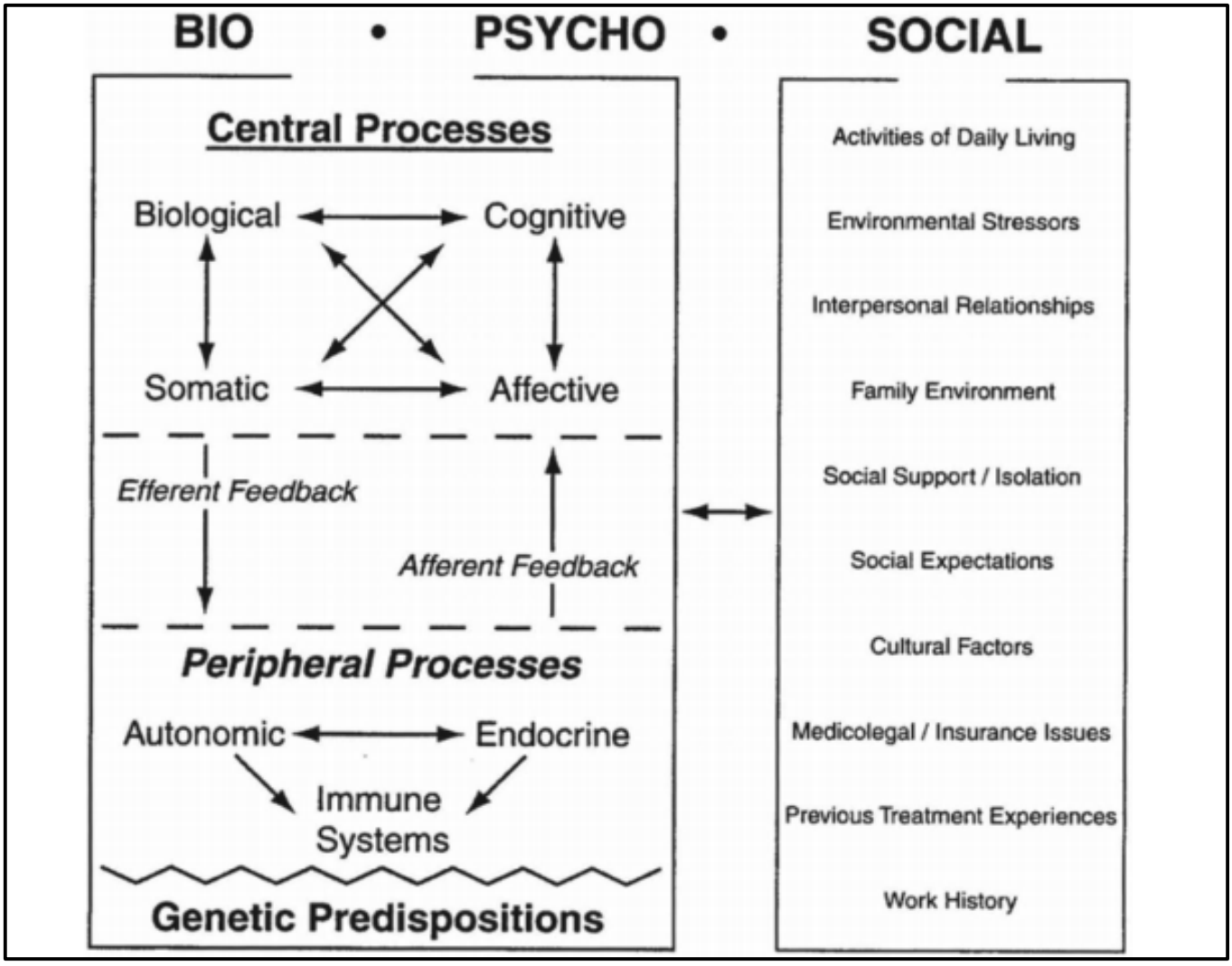


Figure 1. Conceptual model of the biopsychosocial theoretical framework (Gatchel, 2004).

CHAPTER 2

REVIEW OF THE LITERATURE

CBT and Treatment of Depression

Cognitive behavioral therapy is a well-documented empirically supported treatment for depression and other psychiatric disorders. The goal of CBT is to replace patients' maladaptive coping skills, cognitions, behaviors and emotions with more adaptive coping mechanisms (Ehde, Dillworth & Turner, 2014; Gatchel & Rollings, 2008; Sun-Edelstein & Mauskop, 2012). Primary strategies for managing depression are (1) behavioral activation (promoting increases in productive and enjoyable activities) or (2) cognitive restructuring (modifying negative and self-defeating automatic thought patterns) (Sørensen Høifoøt et al., 2011; Smitherman et al., 2008).

A 2006 review of meta-analyses on the empirical status of CBT found CBT to be one of the most extensively researched forms of psychotherapy (Butler, Chapman, Forman & Beck, 2006). Hundreds of studies have demonstrated the efficacy and effectiveness of CBT for treatment of psychiatric disorders, psychological problems and medical problems with a psychiatric component (Butler et al., 2006; Beck, n.d.). According to the World Health Organization (WHO), depression affects at least 350 million people worldwide and is the leading cause of disability worldwide; and although depression is treatable, many people do not receive the support or treatment they need (WHO, 2012). Similarly, Sørensen Høifoøt and colleagues (2011) noted several investigations found 21-65% of patients treated in primary care for depression received psychosocial guideline-concordant treatment.

In a selective review of CBT in primary care settings, CBT was found to be an effective treatment for depression, whether it was supported by general physicians, nurses, social workers or mental/ behavioral health providers (Sørensen Høifoøt et al., 2011). Sørensen Høifoøt and colleagues (2011) also reported psychological presentations to account for 30% of primary care consultations and that patients generally prefer psychological treatment to medication. Sørensen Høifoøt and colleagues (2011) further noted the projected lifetime risk of depression to be up to 31%. European epidemiological studies demonstrated a 1-year prevalence for depression (7% in men, 11% in women); and highlight the substantial impairment these persons experience in multiple functional domains within daily life (e.g. increased medical service utilization and reduced quality of life) (Sørensen Høifoøt et al., 2011).

CBT and Treatment of Chronic Pain

Chronic pain is a condition influenced by a combination of biopsychosocial factors, the consequences of which must be assessed to attain optimal treatment, and is estimated to affect 100 million US adults (Ehde et al., 2014). CBT is a class of treatments that have developed in response to changing conceptualizations of both pain and of psychological change mechanisms (Morley, 2011). The psychology of chronic pain is extensive and ranges from attention control and factors influencing performance of important social roles to aspects of identity construction (Morley, 2011). Empirical research on the treatment of chronic pain with CBT emphasizes the role and impact of cognitive factors on pain and pain management (e.g. appraisal & beliefs, catastrophizing and fear-avoidance beliefs, perceived control & self-efficacy, and vulnerability & resilience) (Gatchel et al., 2007). Gatchel and colleagues (2007) defined CBT, within the parameters of pain management, as a widely varying treatment approach that offers varying selections of the following strategies: relaxation or biofeedback, self-instructions (e.g. imagery,

motivational self-talk), changing maladaptive beliefs about pain, development of coping strategies (e.g. minimizing catastrophizing and self-defeating thoughts, increasing assertiveness), and goal setting.

Morley, Eccleston and Williams (1999), in an influential meta-analysis and systematic review of 25 RCTs of cognitive behavioral therapy for the treatment of pain (excluding headache), concluded CBT to be an effective treatment for a variety of chronic pain conditions. The underlying proponent for CBT in pain management programs was that CBT provides an additional option, aside from medication, for limiting the impact of pain the patient experiences while assisting them to resume normal functional activities (Morley et al., 1999). Similarly, Gatchel et al. (2007) described CBT techniques as embedded within more "comprehensive pain management programs that include functional restoration, pharmacotherapy, and general medical management" (Gatchel et al., 2007, p. 606). For example, in a 2006 study cited in Gatchel and Rollings (2008), the effectiveness of CBT was evaluated in treatment of a cohort of chronic pain patients (75% of whom had chronic low back pain). At the long-term follow-up, they found the combination of CBT with a traditional spinal medical procedure yielded significant improvement in disability, self-efficacy, affective stress and catastrophizing in this cohort which previously showed a suboptimal response to either treatment when administered alone (Gatchel & Rollings, 2008).

In an updated review, Williams, Eccleston and Morley (2012) provided a reaffirmation that "psychological interventions can reduce pain, disability, psychological distress and catastrophic ways of thinking about pain" (p.15). Williams and colleagues (2012) aimed to evaluate the effectiveness of psychological therapies for chronic pain (excluding headache) in adults, compared with treatment as usual (TAU), waiting list control, or placebo control, for

pain, disability, mood and catastrophic thinking conducted a review of 42 RCTs meeting criteria, of which 35 provided data. This review concluded that at post-treatment, compared with TAU or waitlist controls, CBT demonstrated statistically significant though small effects on pain and disability and moderate effects on mood and catastrophizing; at the 6 and 12 month follow-up, mood was the only significant effect (Williams et al., 2012). However, compared with active controls CBT was not superior for pain for mood outcomes, despite showing small, statistically significant benefits for disability and catastrophizing post-treatment; at the 6 and 12 month follow-up, benefits were found only for disability (Williams et al., 2012).

Current Treatment of Headache and Implications

The two approaches to pharmacological treatment are acute pharmacotherapy and prophylactic pharmacotherapy, and are further delineated based upon frequency of headache attacks (i.e. episodic vs. chronic). The first line of pharmacologic treatments for episodic TTH or migraine are typically simple analgesics and non-steroidal anti-inflammatory drugs (NSAIDs), with the second line consisting of combination analgesics containing caffeine (e.g. excedrine) (Freitag, Lyss & Nissan, 2013; Lipton, Serrano et al., 2013; Couch, 2011; Bendtsen et al., 2010; Fumal & Schoenen, 2008; Saper, 2008). The common dosage for these abortive, non-specific treatments are as follows: Paracetamol (500-1000mg), Aspirin (500-100mg), Ibuprofen (800mg), Naproxen sodium (825mg), and Caffeine combination (65-200mg). In most trials, NSAIDs have been found to be superior to aspirin, with Ibuprofen as the first choice of treatment for acute TTH (Freitag et al., 2013; Couch, 2011; Bendtsen et al., 2010; Fumal & Schoenen, 2008; Saper, 2008).

However, frequent and excessive use of these acute pharmacologic treatments has become a widespread problem which leads to medication overuse (Miller & Matharu, 2014;

Lipton, Serrano et al., 2013;Tepper, 2013; Bendtsen et al., 2010; Fumal & Schoenen, 2008; Saper, 2008; Lipton & Bigal, 2007; Scher et al., 2003). Castien et al. (2009) aptly noted patients with chronic TTH (CTTH) report functional and emotional impairments (e.g. loss of workdays, sleep disturbances, emotional well-being) and are at risk for medication overuse. Medication overuse headache (MOH) occurs when typically effective abortive agents are overused, and the over-consumption results in decreased efficacy for headache relief (Miller & Matharu, 2014; Saper, 2008; Weeks, 2013). Tepper (2013) further noted that medication overuse headache (MOH), or rebound headache, often sneaks up on headache patients because the medications work initially, but as they are continually used, they become less effective until they stop working altogether.

For preventative treatment of chronic TTH, amitriptyline (a tricyclic antidepressant) is the first-line choice, while mirtazapine (a noradrenergic and specific serotonergic antidepressant) and venlafaxine (a serotonin and noradrenaline reuptake inhibitory) are second-line medications (Bendtsen & Jensen, 2011; Bendtsen et al., 2010; Fumal & Schoenen, 2008; Saper, 2008; Lipton & Bigal, 2007; Scher et al., 2003). Many studies note the first-line preventative treatment for migraines to be triptans (e.g. sumatriptan), beta-blockers (e.g. propranolol) or tricyclic anti-depressants (e.g. amitriptyline) (Miller & Matharu, 2014; Lipton, Serrano et al., 2013; Saper, 2008; Rampello et al., 2004). Though increasingly used for pain relief of chronic migraine and CDH, treatment via narcotics (opioids, hydrocodone, and oxycodone being the most common) or butalbital (barbiturate) combinations should be avoided to prevent MOH (Miller & Matharu, 2014; Tepper, 2013). Shapiro (2012) noted that while opioids are a mainstay of therapy for acute pain conditions, they actually have a very limited role in recurrent headache conditions (i.e. migraine). Moreover, the use of opioids characteristically renders other medications less

effective, which significantly increases the risk for worsened frequency and severity of headache and migraine attacks (Shapiro, 2012).

While MOH is a real problem in the treatment of TTH and migraine, particularly for chronic presentations and CDH, the efficacy of many prophylactic drug treatments is often hampered by side effects (Weeks, 2013; Bendtsen et al., 2010; Saper, 2008; Sammons, 2005; Campbell, Penzien, & Wall, 2000). This fact again stresses the importance of collaboration between patients and providers (Miller & Matharu, 2014; Barbanti et al., 2014; Nicholson et al., 2011), while also providing another explanation for the gap between migraine prevalence and appropriate treatment (Smitherman et al., 2013). Additionally, Hughes, Wu and colleagues (2013) cited prior research which indicated that only 42.5% of patients with headache conditions receive treatment or care consistent with current guidelines. Furthermore, in their study of healthcare workers with headaches, more than 30% of their sample was not using any medications while 60% reported unsatisfactory treatment (Hughes et al., 2013). They also noted the relationship between headache severity, increased medication and poorer treatment outcomes which present ongoing challenges for their provider (e.g. monitoring symptoms, individualizing treatment, and continually assessing for symptom reduction) (Hughes et al., 2013).

CBT for Treatment of Headache and Migraine

Smitherman and colleagues (2008) accurately assessed that during assessment and treatment of headache, the majority of headache patients would benefit from attention to psychological factors. Because of the complex relationship between the various pathophysiological aspects of TTH, elucidating the difficulty in treating this disorder, multimodal approaches should be implemented through a stepped-care approach, either sequentially or in combination (Fumal &Schoenen, 2008). However, because the majority of

headache patients are treated in primary care or emergency department settings (Burch et al., 2015; Falavigna et al., 2013; Smitherman et al., 2013) with brief physician interaction, the successful implementation of comprehensive bio-behavioral programs is not always feasible (Smitherman et al., 2008).

Weeks (2013) emphasized the fact that behavioral and non-pharmacological treatments for chronic migraine should not be considered anti-pharmacological but rather as an alternative. Haque et al. (2012) similarly found that most people with headache disorders utilize many non-pharmacological measures as a means of alleviating headache pain, especially since the majority of the precipitating and relieving factors of migraine and TTH are both similar and common. A 2007 meta-analytic review echoed this finding that these common non-pharmacological treatments (e.g. relaxation training, EMG biofeedback, CBT, and thermal biofeedback combined with relaxation training) are effective for both TTH and migraine headaches (cited in Weeks, 2013).

Reasons listed for why migraine patients seek these non-pharmacological treatments include the following: poor tolerance/response to preventive medications, medical contraindications to medications, history of overuse of acute care medications, (planned) pregnancy or nursing, to acquire coping strategies for significant stress or pain, and patient preference (Weeks, 2013; Campbell, Penzien, & Wall, 2000). The US Headache Consortium stated that in most instances, behavioral and physical interventions were used as preventative treatment of migraine rather than as a means of alleviation of pain after the fact (Campbell et al., 2000). Campbell and colleagues (2000) also noted five long-term goals for non-pharmacological treatment of migraine as part of the evidence-based guidelines for migraine headache: (1) reduced frequency and severity of headache, (2) reduced headache-related disability, (3) reduced

reliance on poorly tolerated or unwanted pharmacotherapies, (4) enhanced personal control of migraine, and (5) reduced headache-related distress and psychological symptoms (Weeks, 2013).

Lipchik and Nash (2002) noted that in the treatment and management of chronic daily headache, CBT focuses on preventing mild pain from transforming into disabling pain, thereby improving headache-related disability, reducing medication overuse, improving affective distress and improving quality of life. Additionally, CBT is implemented to reduce responses to stress which may trigger, exacerbate and/or sustain headaches, thereby increasing disability and distress (Lipchik & Nash, 2002). Furthermore, consistent with the biopsychosocial model, cognitive-behavioral treatments (e.g. biofeedback and relaxation training) are introduced as methods that reduce physical arousal involved in the onset of headache (i.e. muscle tension, vascular responses) (Lipchik & Nash, 2002).

Holroyd (2002) reviewed the behavioral and psychological aspects of the pathophysiology and management of frequent TTH, and noted behavioral treatments (including CBT) were efficacious when used as an alternative or as an adjunct to medications. Holroyd (2002) further noted that while clinical trials comparing tricyclic antidepressant medications and CBT for CTTH yielded similar treatment outcomes, the combination of the two would likely enhance outcomes as was discovered in a large study conducted the previous year (Holroyd, O'Donnell, et al., 2001).

More recently, Fumal and Schoenen (2008) reviewed current knowledge about treatment and management of TTH and cited solid scientific support for use and effectiveness of non-pharmacological behavioral treatments such as relaxation and electromyography (EMG) biofeedback therapies. In fact, the combination of relaxation and EMG behavioral feedback training led to a nearly 50% reduction in TTH headache activity (Fumal & Schoenen, 2008).

CBT interventions (e.g. stress management) were also presented as an effective means of reducing TTH activity; however, the researchers stated CBT was most effective in combination with relaxation and EMG biofeedback. A remarkable finding was that although sole use of these behavioral interventions were yielded improvements in headache activity more slowly compared to pharmacological treatments, headache improvement was maintained for longer periods of time, up to several years, without monthly follow-up (through either the therapist or monthly sessions) (Fumal & Schoenen, 2008).

Treating Pain with Comorbid Depression

While the literature is replete with evidence for the bidirectional relationship between headache and psychiatric disorders, few other pain disorders (except back pain, fibromyalgia and IBS) have been studied for the presence of this same relationship (Ligthart, Gerrits, Boomsma, & Pennix, 2013). Despite the combination of pain and depression seen in clinical practice and reported by patients, few studies focus on the influence of pain and psychiatric comorbidity and its intersectionality (Gerrits, Vogelzangs, van Oppen, van Marwijk, van der Horst & Penninx, 2012). A 2003 literature review did however note 14 studies on patients assessed for pain with comorbid depression which found the mean prevalence of pain to be 65%, within a range of 15-100% (Gerrits et al., 2012).

In the investigation of the interrelationship between depression, migraine and pain (n=2,981) Ligthart et al. (2013) observed the following: (1) the strongest associations were between depression and chest pain, neck pain, and strict migraine, (2) probable migraine and mild non-migrainous headache were more weakly associated with depression, (3) association with pain was strongest in patients with combined psychiatric disorders, (4) combined psychiatric disorders were associated with a higher number of pain sites (compared to depression

alone). Consistent association among migraine and pain in other sites was observed; however, these observations were significantly weaker after depression was added to this model (Ligthart et al., 2013). Finally, associations with neck pain, orofacial pain and abdominal pain were most evident after adjusting for the effects of comorbid depression (Ligthart et al., 2013). Ligthart et al. (2013) also noted that several of these important findings are consistent with previous findings. While CBT alone may not address all of the important factors which contribute to the chronic pain (i.e. biological factors), research has shown that CBT may improve care for patients with psychiatric comorbidities (Gatchel & Rollings, 2008; Sun-Edelstein & Mauskop, 2012).

Gerrits et al. (2012) studied the impact of pain on depressive disorders and found pain to be associated with a worse prognosis for comorbid depressive disorders; and the chronic pain variables were especially strong predictors of chronicity of depression. The review of prior research found increasing attention being paid to collaborative care studies for patients with depression, several of which showed positive results with combined psychological and antidepressant therapies on the reduction of various pain symptoms; however, several found either poorer treatment response or worse depression (Gerrits et al., 2012).

CBT Treatment for Chronic Headache with Depression

Approximately 33 to 50% of chronic headache patients have mild to moderate depression; and traditional headache treatment has been proven to be less effective in depressed patients (Martin, Meadows et al., 2013). Barbanti and colleagues (2014) reviewed various studies evaluating the effectiveness of pharmacologic, non-pharmacological and other treatments for patients with chronic TTH and chronic TTH with psychiatric comorbidities. They found that CBT decreased TTH activity by 50% or more in 40-50% of the patients and for patients with higher stress levels and psychiatric comorbidities, CBT combined with relaxation training was

found to be most effective (Barbanti et al., 2014). The same study did however note that the most beneficial prophylactic treatment of unremitting TTH or concurrent mood disorders was the combination of CBT with antidepressants (Barbanti et al., 2014). The review, though brief and with additional focus of manual therapy in the treatment of TTH in primary care settings, underscored the benefits TTH patients may receive from treatments aside from simple analgesics (Barbanti et al., 2014).

In the 2010 European Federation of Neurological Societies (EFNS) guidelines on the treatment of TTH with psycho-behavioral methods, Bendtsen, Evers et al. (2010) remarked that the common psychosocial treatment methods of EMG biofeedback, CBT and relaxation training have been the most investigated, yet stated only a few trials have provided sufficient power and clear outcomes on their effectiveness; and hypnotherapy did not have enough convincing evidence for the few reported effects it had in treating TTH. As did other studies encountered within this literature review, Bendtsen et al. (2010) cited the conflicting results of the Holroyd et al. studies of 2001 and 2002 which studied the efficacy of headache treatment with either CBT , tricyclic antidepressants, placebo or combined treatment; and determined that while CBT may be effective treating TTH, there was not any convincing evidence.

Similarly, Verhagen and colleagues (2014) reviewed RCT behavioral treatments of CTTH in adults, compared to no treatment, waiting list, or other treatment. Of the 44 RCTs selected, only 29 had sufficient outcome data, eight of which examined the effectiveness of CBT (Verhagen et al., 2014). No significant differences were found between CBT and placebo or a tricyclic (amitriptyline) on headache improvement in four of the RCTs (n=430), and no significant headache outcomes were found when CBT was used as an adjunct to relaxation therapy in the remaining four RCTs (n=186) (Verhagen et al., 2014). Overall, this review found

no indications that these commonly used behavioral therapies were better than waiting list or attentional controls in improving TTH outcomes (Verhagen et al., 2014). Bendtsen and Jensen (2011) found evidence of EMG biofeedback having an effect on TTH but did not find convincing evidence of CBT or relaxation training on TTH.

Conversely, a 2011 review of non-pharmacologic treatment for both migraine and TTH not only found behavioral treatment to possess the most evidence for successful headache management, but also endorsed the recommendation that these behavioral therapies should be first-line options for prophylactic treatment (Nicholson, Buse, Andrasik & Lipton, 2011). Nicholson and colleagues (2011) further clarified that the patients most likely to benefit from non-pharmacologic treatment have any of the following: comorbid mood or psychiatric disorders, difficulties coping with headache/significant headache-related disability, medication overuse, trauma history, patient preference for a specific treatment modality or significant problems managing stress. As with any treatment, physical or psychological, active collaboration between patient and provider is required when choosing the optimal interventions which both meet the patient's needs and preferences (Miller & Matharu, 2014; Barbanti et al., 2014; Nicholson et al., 2011). This review found the same results as Barbanti and colleagues (2014) as did the US Headache Consortium (2000), that CBT yielded reduced TTH activity by 50% or more in 40-50% of the patients; however, this review suggested CBT to be a class I, indicating it exhibits the highest level of evidence for headache prevention (Nicholson et al., 2011).

Finally, Test and Test (2014) conducted a literature review to discern the efficacy of mindfulness-based cognitive therapy (MBCT) in treating depression and found MBCT protected participants from depressive episode recurrence and relapse who had been previously treated with antidepressants. The findings of this review provided evidence that MBCT is effective as a

preventative treatment for depression (Test & Test, 2014). Their implications for further research align with the purpose of this proposed pilot study because perceived stress and self-efficacy were negatively correlated with higher depression and lower dispositional hope (Test & Test, 2014); however, cognitive therapies are shown to be effective in helping individuals cope with their situation (i.e. chronic headache/migraine, chronic pain, comorbid psychiatric disorders).

CHAPTER 3

METHODOLOGY

Participants

Participants of this pilot study were primarily recruited from Mercy Health Center, a primary care center in a rural Southeastern town and from the Henry Ford Health System in a large, urban Midwestern city. Participants were also recruited from the clinicaltrials.gov online posting requisite for all IRB approved clinical intervention studies. Prospective participants contacted the student researcher, either by phone or email, in response to flyers posted at Mercy Health Center, from provider referrals within the Henry Ford Health System and directly from the clinicaltrials.gov online study posting. After they were provided with additional information about the study, prospective participants who desired to participate completed an eligibility screening by phone. During eligibility screening interviews, provision of informed consent in addition to possible benefits and risks associated with the study were discussed with all participants.

This clinical sample was comprised of patients who met both a chronic headache diagnosis (International Classification of Headache Disorders 2nd Edition, ICHD-II, 2004) and psychiatric diagnosis of a depression disorder (emotion formal diagnosis) DSM 5, 2013). Of the 22 enrolled participants, 9 were recruited from the Southeastern primary care center and 13 were recruited from the Midwest health system. Enrolled participants met the following inclusion criteria: (1) age 18 to 75 (the peak period for headache activity), (2) have depressive symptoms (PHQ-9 score of 5 or higher), and (3) frequent to chronic headache condition (i.e. chronic daily

headache or migraine) characterized as approximately 10 or more headache days per month for the past 3 months.

A common technique used in clinical trials to both achieve balance in the allocation of participants to treatment arms and reduce bias, particularly with small sample sizes, is blocked randomization. The advantages of this restricted randomization method are that it prevents imbalances that are more likely with simple randomization and, more importantly, the sample tends to be uniformly distributed by key outcome-related characteristics (Efird, 2011; Kang, Ragan & Park, 2008; Lachin, 1988). Randomization was conducted once an even number of successfully screened prospective participants was reached (i.e. blocks of 2 or 4) to ensure that each group had an equal number of participants and met the minimal proposed number of participants per study arm. The block sizes and allocation ratio were specified and entered into an online tool (Sealed Envelope, 2016) that generated blocked randomization lists.

Within each block the allocation of participants was random. For example, a block size of 4 and an allocation ratio of 1:1 would lead to random assignment of 2 subjects to one group and 2 to the other (Wikipedia, 2016). See Figure 2: NIH CONSORT (Consolidated Standards of Reporting Trials) of enrollment, allocation and follow-up in a 2-group parallel randomized trial (Hopewell, Hirst, Collins, Mallett, Yu, & Altman, 2011). After eligibility criteria was met and randomization was completed, participants in both study arms completed the pre-, post-, and follow up measures online through the Qualtrics survey platform via an anonymous link that was emailed to them. Paper/ hard copies were available upon request.

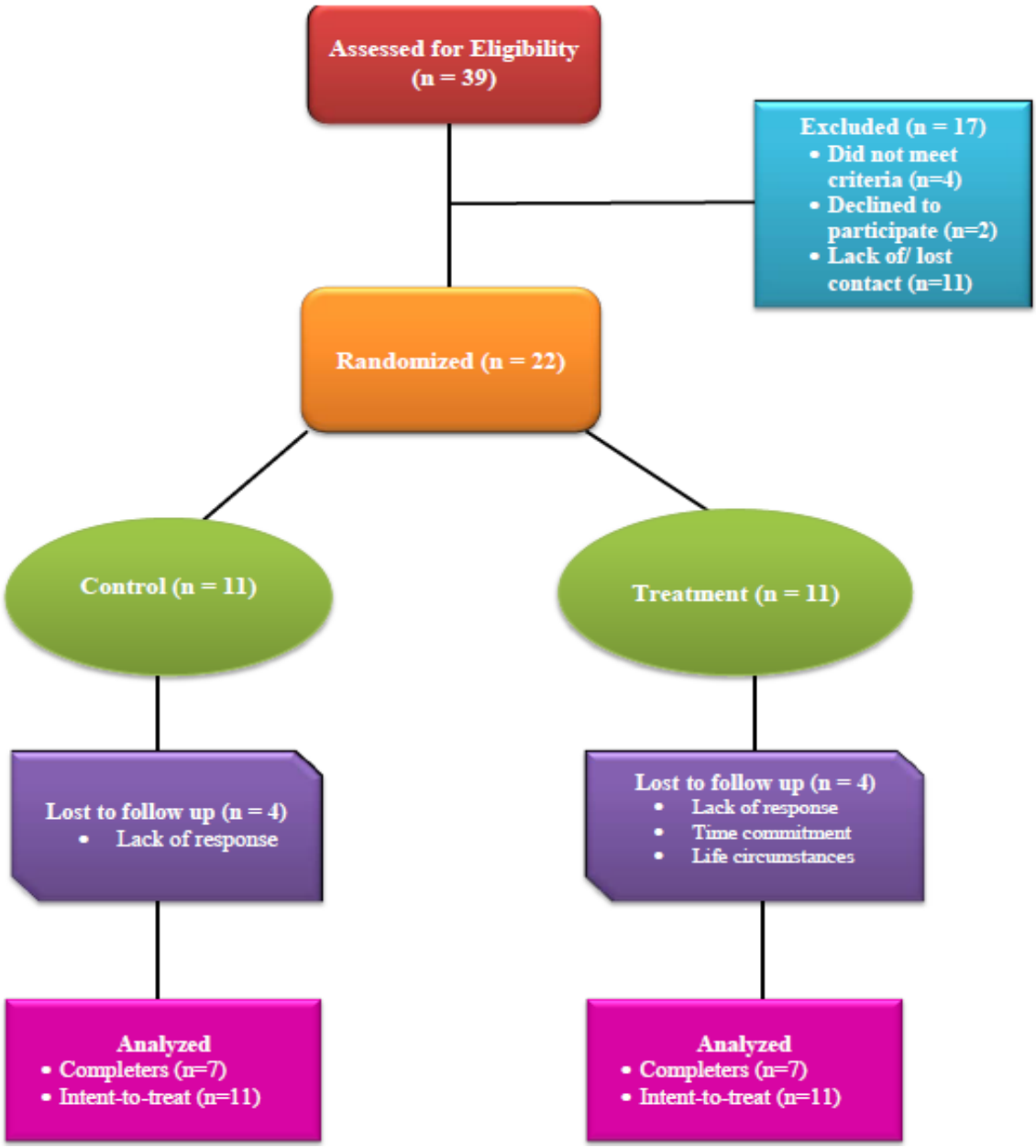


Figure 2. NIH CONSORT (Consolidated Standards of Reporting Trials) flow diagram of enrollment, allocation and follow-up in a 2-group parallel randomized trial.

Design

This pilot study was presented as a pretest-posttest randomized clinical trial evaluating the effectiveness of Cognitive Behavioral Therapy targeted to treat depression in persons with co-occurring chronic headache pain compared to treatment as usual. Participants who satisfied all eligibility criteria were randomized into either the control or treatment group via an online

random coin flip program. Randomization was used to isolate and nullify any confounding variables and noise.

Pre-tests, post-tests and 4-week follow-up tests were given to participants in study arms to assess for reduction in both depressive symptoms and of headache characteristics (e.g. frequency and severity). Upon completion of the online assessment (pre, post or follow-up), participants were compensated with a $10 gift card to a retailer of their choice for each completed assessment for a total of $30 – retailer selections included Kroger, Meijer, Walmart or Amazon. Additionally, the treatment group received $10 for each attended intervention session, to offset the travel expense, up to a total of $40. The treatment condition received a 4-week manualized cognitive-behavioral intervention to treat the depression symptoms and teach adaptive coping strategies that could then later be generalized to headache management.

Design rationale (i.e. sample size and number of intervention sessions) was supported by a review of several meta-analyses of studies conducted with similar aims, designs and intervention methods. A search of similar study designs yielded several meta-analyses, systematic reviews and stand-alone case studies of CBT interventions treating depression – face-to-face, computer-based CBT with or without therapist support, and CBT v. control comparison – that recruited from community samples. Antoniades, Mazza & Brijnath (2014) conducted a systematic review of 15 studies on the efficacy of depression treatment for immigrant patients (living in the US). Of the 15 studies, 6 described a similar design of a pilot RCT with pre- and post-intervention measures, some of which also included a subsequent post-study follow up, with sample N sizes ranging from 5 to 38 – though half were in the 10-19 range. Similarly, 3 of the 4 face-to-face studies in Richards & Richardson's (2012) meta-analysis reviewed recorded sample

N sizes ranging from 20 to 27. Moreover, a power analysis conducted at α= .05, power = .8, effect size = .4 for a multiple linear regression indicated N=22.

Regarding the length of the intervention, the majority of systematically reviewed studies that conducted face-to-face interventions implemented an average of 8 sessions (Twomey, O'Reilly, & Byrne, 2015; Cujipers, Berking, Andersson, Quigley, Kleiboer, & Dobson, 2013). However, some studies implemented as few as 5-6, though most fell in the 8-12 or 8-16 range with very few studies implementing greater than 16 sessions (Antoniades et al., 2014; Quigley et al., 2013).

Procedure

Participants assigned to the treatment group scheduled their 4 intervention sessions with the student researcher upon completion of the pre-test measures. The objective of the CBT intervention sessions was to teach the participants to recognize, cope with and manage their stress and depression, identify cognitive distortions, provide psychoeducation about behavioral activation and the role of mood and activity in the cycle of depression, long-term depression management and to address wellness activities (sleep, exercise and dietary hygiene). The intervention CBT techniques implemented outlined below in each session (refer to Appendix A for the complete treatment manual) and the interventionist instructed the participants on how to continue to use these techniques at home after the study conclusion. Each session lasted approximately 45-60 minutes except for session 1, which lasted approximately 60-75 minutes. Session 1 included a pre-session intervention overview in which participants were taught the basics about clinical depression, provided an overview about the CBT approach, and the circumstances of the participant's depression were briefly ascertained to partially tailor the intervention activities and examples to each participant. This additional time allotted for the pre-

session served as a "getting-to-know-you" period that allowed the participant and student researcher tobetter establish rapport, and ideally enhance the participant's intrinsic motivation to complete the intervention. Prior to beginning sessions 2 through 4, homework from the previous module was briefly reviewed to assess comprehension and applicability of module activities.

After the pre-session overview of session 1, participants were led through Module 1: How Thoughts Affect Mood-Part 1 (identified depressed v. non-depressed thoughts; learned to distract themselves from/disrupt negative automatic thoughts) and were assigned related homework activities. In session 2, participants completed the second half of Module 1: How Thoughts Affect Mood-Part 2 (identified, recorded and challenged cognitive distortions via the ABCD method) with related homework activities. In session 3, participants were led through Module 2: How Activities Affect Mood (defined & recalled enjoyable activities; learned to make SMART goals) with related homework activities. In the fourth and final session, participants completed Module 3: How Relationships Affect Mood (learned the importance of a strong support network & how to expand it; learned how to establish healthy relationships; learned to understand thoughts/feelings/expectations of self and others; reviewed assertiveness training for boundary setting/toxic relationships) with take home worksheets and an opportunity to provide feedback about the intervention (e.g. what they learned, what did and did not work as it applied to their specific situations).

A total of 6 interventionists (doctoral psychology students in different points of the graduate program) administered the CBT intervention. Among the interventionists were 2 females (the primary student investigator and a 3rd year doctoral student) and 3 males (a 3rd year doctoral student, a 4th year doctoral student and a pre-doctoral intern). All interventionists were supervised by a licensed clinical psychologist. Therapists completed a 2-hour training session to

maximize comprehension of the study's purposes and procedures, the theoretical background of the CBT intervention, and the importance of fidelity to intervention protocol. A standardized (i.e., briefer) adaptation of a CBT group intervention manual was used by all interventionists to facilitate intervention delivery efforts and increase fidelity to intervention protocol. The intervention training also reviewed the study's protocol to be enacted in the event of mental health emergencies (e.g., participants being actively suicidal or expressing homicidal intent). Current information outlining local mental health organizations in the geographic areas from which participants were recruited and telephone numbers of national suicide hotlines were provided to therapists during training, and included in the informed consent provided to all participants.

Supervision was conducted vertically, with the primary student investigator speaking with each interventionist following each session to assess protocol fidelity and discuss any potential clinical issues. No issues arose while this intervention was conducted. However, had there been any clinical issues, per the established protocol, the student researcher would have discussed them with either site supervisor – Dr. Bernadette Heckman for the Southeastern primary care site and Dr. Lisa Matero at the Midwest healthcare system site – as appropriate based on the participant's respective site.

Measures

Eligibility screening for all prospective participants was conducted via administration of the Patient Health Questionnaire-9 (PHQ-9; Spitzer, Williams & Kroenke, 1999), an abbreviated headache diagnostic interview to assess headache frequency and severity, in addition to current and past treatment for both their depression and headaches. Upon completion of the eligibility screening, the study participants were contacted to complete pre-test, post-test and 4-week follow

up surveys comprised of the following measures: self-report of number of headache days for the past 30 days, the Beck Depression Inventory-II (BDI-II; Beck et al., 1961), the Migraine Disability Assessment (MIDAS; Lipton et al., 2000), the Headache Disability Inventory (HDI; Jacobson et al., 1994), the Headache Self-Efficacy Scale: Adaption to Recurrent Headaches (HSE; Martin et al., 1993), and the Headache Specific Locus of Control: Adaption to Recurrent Headaches (HSLC; Martin et al., 1990). Participants also provided information on the following sociodemographic variables: gender, race/ethnicity, sexual orientation, level of education, employment status, relationship status, health insurance, duration of headaches in years, type of treatment they received (i.e. medication, therapy, alternative, etc.) – past or present – for their headaches and depression. The pre-test survey responses established a baseline for perceived disability of the participants' headache and depressive symptoms. See Table 4 in the appendix for a comparison of the Cronbach's alphas from this study compared to a larger, clinical sample with similar diversity.

The PHQ-9 is the 9-item depression module from the full Patient Health Questionnaire measure, with each of the 9 items scored from 0 (not at all) to 3 (nearly every day). The total score is derived from the sum of the items and may range from 0 to 27. Diagnoses of major depression and other depression are diagnoses depending on the number of depressive symptoms endorsed and the duration (i.e. "more than half the days" in the past 2 weeks). If present at all, regardless of duration, the criteria "thoughts that you would be better off dead or of hurting yourself in some way" counts. The additional item added to the end of the diagnostic portion of the questionnaire asked patients who endorsed any problems, "How difficult have these problems made it for you to do your work, take care of things at home, or get along with other people?" The internal reliability of the PHQ-9 was considered excellent (Cronbach's $\alpha = 0.89$) in

the PHQ Primary Care Study (N=3,000) and (α = 0.86) in the replicated PHQ Ob-Gyn Study (N=3,000) – through which external validity was achieved with excellent test-retest reliability. There was a strong correlation (r=0.84) between the patient self-report PHQ-9 and the PHQ-9 administered over the phone by a mental health provider within 48 hours. A PHQ-9 score ≥10 had a sensitivity of 88% and a specificity of 88% for major depression, using the MHP re-interview as the criterion standard. (Kroenke, Spitzer & Williams, 2001).

The Beck Depression Inventory-II (BDI-II) was originally developed to assess the intensity of depression and is one of the most widely accepted and used depression inventories (Richter, Werner et al., 1998). This instrument is a 21 item self-report Likert-scale inventory which allows the respondent to endorse four items from 0 to 3, reflecting a continuum of depressive severity (French et al., 2000). The total score is derived from the sum of the items (French et al. 2000). Richter et al. (1998) noted a strength of the BDI in that it was "designed to reflect the depth of depression, monitor changes over time, provides an objective measure for judging improvement and the effectiveness or otherwise of treatment methods. The BDI has been used in over 2000 empirical studies and remains widely used in research (Richter, Werner et al., 1998). The instrument was shown to have high internal consistency (α=.91) in addition to a high one-week test–retest reliability (Pearson r =0.93), indicating this measure is not "overly sensitive to daily variations in mood" (Beck, Steer et al., 1996). Internal consistency for this study sample was poor (α=.68).

In 1999, Stewart and Lipton developed the Migraine Disability Assessment Questionnaire (MIDAS) to assess the severity of disability related to migraine with the past 3 months (Lipton, Stewart et al., 2001). The first 5 items of this 7-item questionnaire focuses on disability in three domains of (1) school or paid work, (2) household chores, and (3) family,

social, or leisure; and serves as a simple tool to improve physician-patient communication (Stewart, Lipton et al., 2001). Stewart, Lipton, et al. (2001) determined the MIDAS Questionnaire to be internally consistent, highly reliable, valid, and correlated with physicians' clinical judgment. Internal consistency for this study sample was excellent (α=.96).

The Henry Ford Hospital Headache Disability Inventory (HDI) assesses the impact of headache on daily living, headache treatment, and to periodically evaluate the level of disability experienced by a patient with headache (Jacobson et al., 1994). Additionally, the HDI is used to determine the effectiveness of a management strategy over time with the higher the patient's score on the inventory, the greater the disability caused by the headache (Jacobson et al., 1994). The level of disability is interpreted as follows (scores are expressed as a percentage): 10-28% = mild disability, 30-48% = moderate disability, 50-68% = severe disability, and 72% or more = complete disability. The inventory has demonstrated strong internal consistency, reliability and construct validity with a 1 week total score test-retest reliability of 0.76, and a 6-week test-retest reliability of 0.83 (Jacobson et al., 1994). Internal consistency for this study sample was acceptable (α=.79) for the total scale, good for the Emotional disability subscale (α=.82) and acceptable for the Functional disability subscale (α=.70).

The Headache Self-Efficacy Scale: Adaption to Recurrent Headaches (HSES) is a 51 item Likert-scale designed specifically for recurrent headache sufferers. Items rated on a 5-point scale that ranges from 1 (very confident) to 5 (no confidence). This measure was designed to assess the individuals' confidence in their abilities to prevent headache episodes (e.g. "I can prevent headaches by recognizing headache triggers") and to manage head pain (e.g. "I can reduce the intensity of headache by relaxing") when confronted with personally relevant

headache precipitants (Martin et al., 1993). Internal consistency for the current pilot study sample was poor (α=.69).

The Headache Specific Locus of Control: Adaption to Recurrent Headaches (HSLC) is 33 item Likert-scale designed specifically for recurrent headache sufferers (Martin, Holroyd & Penzien, 1990). The HSLC has responses ranging from 1 (strongly disagree) to 5 (strongly agree). There are 3 subscales: Health Care Professionals Locus of Control, Internal Locus of Control, and Chance Locus of Control. These subscales assess the individual's perceptions (i.e. locus of control) about their headache problems and headache relief on three dimensions: the individual's behavior (internal factors), healthcare professionals, or chance factors (Martin et al., 1990). It should be noted that to create a total Internal/External LOC score, items on either the Internal subscale or on the two External subscales (Health Care Professionals & Chance) must be reverse scored. In this study, consistent with previous reports using this scale, items on the Internal subscale have been reverse scored so that higher scores indicate a greater external LOC and greater perceived disability. Internal consistency for this study sample was good (α=.82).

Data Analytic Plan

Data Preparation. Analyses were conducted on aggregated data by treatment condition (i.e. control vs. intervention), to compare the effectiveness of the proposed intervention to usual care and other available treatment approaches. All statistical analyses were conducted with the latest version of Statistical Package for the Social Sciences. Descriptive statistics and exploratory analyses were conducted for the variables of depression, headache severity, headache frequency, headache-related disability, headache-related quality of life, and psychiatric comorbidities. The data was screened for outliers via measures of skewness, kurtosis and by comparisons of means – and no outliers were found. Assumptions of normality were examined via probability plots,

Levene's Test for Equality of Variances, and Box's M Test of Equality of Covariance Matrices. Because the baseline scores for Midas-A and MGrade were skewed, and thereby violated the assumptioms of normality, the data was subjected to a square root transformation to stabilize the variance for the factorial repeated measures ANOVA and meet the assumptioms of normality. Descriptive statistics, independent samples t-tests, and chi-squared analyses examined if randomization was successful.

Outcome Analyses. Chi-square tests of association and independent samples t-tests were initially conducted to identify associations among demographics assessed at baseline, intervention condition, and intervention completion status (completed, dropped). The primary intervention outcome analysis compared CBT intervention and control/standard care participants using a 2 (condition) x 2 (time) repeated measures ANOVA on the primary outcome measures (BDI-2, HDI, and 30-day headache self-report) and the psychosocial measures (HSES and HSLC). Clinical change in the primary outcome measures was defined as the relative change in symptom frequency and/or severity post-intervention compared to baseline, with the formula (Xpre-Xpost)/Xpre. Clinically meaningful change was assessed using Bengtson, et al.'s (2015) methodology that categorizes clinically meaningful responses to therapy as: (1) full response = ≥50% reduction in depressive or headache symptoms from pre- to post-intervention; (2) partial response = 25-49% reduction; and (3) no response = <25% reduction.

Intention-to-treat (ITT) analyses, the standard for controlled clinical trials (particularly drug trials), consists of the inclusion of all participants in the analysis according to the group determined at randomization (Molnar, Hutton & Fergusson, 2008). One ITT method is the "Last Observation Carried Forward" (LOCF). This analysis method assumes that missing values are "missing completely at random" (i.e., the probability of dropout is not related to variables such

as disease severity, symptoms, group assignment or side effects of treatment) and that the participant's responses (e.g., outcome measures) would have been stable from the point of dropout to study completion, rather than improving or declining further (Molnar et al., 2008). With this method, the participant's missing values after dropout are replaced with the last available measurement (Molnar et al., 2008; Streiner & Geddes, 2001). Advantages to this analytical approach include the ability for analyses to examine trends over time and that it minimizes the number of participants eliminated from the analysis.

CHAPTER 4

RESULTS

Sample Characteristics

The sample (N=22) consisted of 19 females and 3 males. Sample characteristics at baseline are shown in Table 1. Most participants (*M* age=41.09 years, min=19, max=63) were White (54.5%) or Black (45.5%) and female (86.4%). The majority of the sample completed two years of college or less (59.1%), were employed part-time or less (i.e. Unemployed, unable to work/on social security disability; 59.1%), had some type of health insurance (68.2%), self-identified as heterosexual (81.8%), and were single (68.2%). Half were chronic migraineurs for 3 to 9.5 years and 36.4% were chronic migraineurs for 10 years or more.

At baseline, participants' overall mean BDI-II value was 28.55 (sd = 10.35), with 95.5% reporting scores greater than 13, the cut-off value recommended to identify mild depression (Beck et al., 1996); 50% reported scores in the severe range. Chi-square measures of association found no associations among intervention condition and gender, ethnicity, employment status, partner status, sexual orientation, health insurance status, or level of education (all ps > .10). A series of independent t-tests found no significant differences between intervention condition and age or number of headache years (all ps > .44; see Table 2). However, CBT participants reported marginally higher number of headache days, in the past 90 days, as measured by the MIDAS at baseline (M = 61.45, SD=17.62) relative to Standard Care Control (SCC) group, [(M = 43.45, SD=25.48); t(17.78) = -1.93, p=.07].

Table 1. Characteristics of participants at baseline

Characteristic	*Statistic*	*Overall n = 22*	*Control n = 11*	*Treatment n = 11*	*P-value**
Age	Mean (SD)	41.09 (13.10)	42.36 (12.39)	39.82 (14.27)	.660
Sex:					
Female	N (%)	19 (86.4%)	10	9	.534
Male		3 (13.6%)	1	2	
Race/Ethnicity:					
Black	N (%)	10 (45.5%)	5	5	1.00
White		12 (54.5%)	6	6	
Sexual Orientation:					
Heterosexual	N (%)	19 (86.4%)	10	9	.943
Questioning		2 (9.1%)	1	1	
Missing Value		1 (4.5%)	0	1	
Partner Support:					
Partnered	N (%)	7 (31.8%)	5	2	.170
No Partner		15 (68.2%)	6	9	
Education:					
Kinder – 2yr degree	N (%)	13 (59.1%)	6	7	.665
College or beyond		9 (40.9%)	5	4	
Employment:					
Full-Time	N (%)	9 (40.9%)	6	3	.193
Part-Time or Less		13 (59.1%)	5	8	
Health Insurance:					
None/Unsure	N (%)	7 (31.8%)	4	3	.647
Any Insurance		15 (68.2%)	7	8	

*P-values from independent samples t-tests and chi-square tests, for continuous and categorical variables, respectively.

Attrition Rates

Eight participants were lost to follow-up, resulting in an overall study attrition rate of 36.4%. An equal number from each study arm discontinued study involvement. Most participants who discontinued their participation could not be contacted by phone or e-mail (n=6), one discontinued participation due to lack of time, and , and one due to loss of a family member (n = 1). Independent-group t-tests and chi-square analyses revealed no significant

associations between completion status (completed, dropped) and any demographic or psychosocial variable assessed at baseline (see Tables 1and 2).

Table 2. Significance test values of outcome measures at baseline

Measure	*Statistic*	*n = 22*	*χ2 or t p-value**
BDI Total:	Mean (SD)	28.55 (10.35)	.84
HDI Total:	Mean (SD)	73 (19.88)	.60
HDI-Emotional:	Mean (SD)	35.91 (10.54)	.37
HDI-Functional:	Mean (SD)	37.09 (10.51)	.94
HSES Total:	Mean (SD)	188.95 (49.35)	.46
HSLC Total:	Mean (SD)	94.86 (17.84)	.67
MIDAS-A:	Mean (SD)	52.45 (23.28)	.05
MGrade Level:	Mean (SD)	3.55 (.96)	.38
Headache Years	Mean (SD)	11.21 (12.06)	.44

*P-values from independent samples t-tests and chi-square tests, for continuous and categorical variables, respectively.

Treatment Outcomes and Completer versus Non-completer Analyses

A series of 2 (condition) x 2 (time) repeated measures factorial ANOVAs were conducted to assess the "Condition x Time" interaction on the primary outcome measures (BDI-2 Total, HDI Total plus the emotional and functional disability subscales, MGRADE: level of disability and the 30-day headache self-report) and the psychosocial measures (HSLC Total and HSES Total) from baseline to post-intervention.

Outcomes were assessed between the two conditions using intention-to-treat (N=22) and completer-only (N=14) approaches for participants with baseline values. Intention-to-treat analyses used data from all 22 participants regardless of number of intervention sessions attended. Completer-only analyses used data from the 14 CBT participants who attended all 4

intervention sessions, and completed the post-intervention measures, as well as all Standard Care Control (SCC) group. In intention to treat analyses, a Last Observation Carried Forward (LOCF; Gupta, 2011; Molnar et al., 2008, Streiner & Geddes, 2001) approach was used to impute missing post-intervention values on measures depressive symptoms, headache disability, perceived headach-related self-efficacy and locus of control.

ITT analyses with LOCF

Beck Depression Inventory. A 2 x 2 RM ANOVA found no main effect for Time, $F(1,20)=0.57$, $p=.46$ and no main effect for Condition, $F(1,20)=0.21$, $p=.66$. The "Time x Condition" interaction was also not statistically significant, $F(1,20)=0.20$, $p=.66$.

Migraine Disability Assessment (90-day headache report). A 2 x 2 RM ANOVA found a statistically significant main effect for Time, $F(1,20)=5.705$, $p=.027$, but no main effect for Condition, $F(1,20)=0.62$, $p=.44$. However, the main effect for "Time" was complicated by a "Time x Condition" interaction that was statistically significant $F(1,20)=4.807$, $p=.04$.

Headache Disability Inventory. A 2 x 2 RM ANOVA found no main effect for Time, $F(1,20)=.71$, $p=.41$ and no main effect for Condition, $F(1,20)=.39$, $p=.54$. The "Time x Condition" interaction was also not statistically significant, $F(1,20)=0.94$, $p=.76$.

HDI-Emotional disability subscale. A 2 x 2 RM ANOVA found no main effect for Time, $F(1,20) = .020$, $p=.89$ and no main effect for Condition, $F(1,20)=.65$, $p=.43$. The "Time x Condition" interaction was also not statistically significant, $F(1,20)=0.18$, $p=.68$.

HDI-Functional disability subscale. A 2 x 2 RM ANOVA found no main effect for Time, $F(1,20)=2.95$, $p=.10$ and no main effect for Condition, $F(1,20)=.16$, $p=.69$. The "Time x Condition" interaction was also not statistically significant, $F(1,20)=1.87$, $p=.19$.

MGRADE (MIDAS level of disability). A 2 x 2 RM ANOVA found no main effect for Time, F(1,20)=3.34, p=.08 and no main effect for Condition, F(1,20)=.03, p=.86. The "Time x Condition" interaction was also not statistically significant, F(1,20)=.03, p=.87.

Headache Self-Efficacy Scale. A 2 x 2 RM ANOVA found no main effect for Time, F(1,20)=.11, p=.74 and no main effect for Condition, F(1,20)=.27, p=.61. The "Time x Condition" interaction was also not statistically significant, F(1,20)=.48, p=.50.

Headache Specific Locus of Control. A 2 x 2 RM ANOVA found no main effect for Time, F(1,20)=.02, p=.90 and no main effect for Condition, F(1,20)=.19, p=.67. The "Time x Condition" interaction was also not statistically significant, F(1,20)=. 02, p=.90.

Completer Analyses

Beck Depression Inventory. A 2 x 2 RM ANOVA found no main effect for Time, F(1,12)=.55, p=.48 and no main effect for Condition, F(1,12)=0.00, p=.99. The "Time x Condition" interaction was also not statistically significant, F(1,12)=.20, p=.67.

Migraine Disability Assessment (90-day headache report). A 2 x 2 RM ANOVA found a statistically significant main effect for Time, F(1,12)=6.99, p=.02, but no main effect for Condition, F(1,12)=0.01, p=.92. However, the main effect for "Time" was complicated by a significant "Time x Condition" interaction, F(1,12)=6.28, p=.03.

Headache Disability Inventory. A 2 x 2 RM ANOVA found no main effect for Time, F(1,12)=.69, p=.42 and no main effect for Condition, F(1,12)=.03, p=.87. The "Time x Condition" interaction was also not statistically significant, F(1,12)=0.91, p=.77.

HDI-Emotional disability subscale. A 2 x 2 RM ANOVA found no main effect for Time, F(1,12) = .020, p=.89 and no main effect for Condition, F(1,12)=.21, p=.65. The "Time x Condition" interaction was also not statistically significant, F(1,12)=0.17, p=.69.

HDI-Functional disability subscale. A 2 x 2 RM ANOVA found no main effect for Time, F(1,12)=3.23, p=.10 and no main effect for Condition, F(1,12) =.02, p=.91. The "Time x Condition" interaction was also not statistically significant, F(1,12) =2.04, p=.18.

MGRADE (MIDAS level of disability). A 2 x 2 RM ANOVA found no main effect for Time, F(1,12)=3.68, p=.08 and no main effect for Condition, F(1,12) =.03, p=.86. The "Time x Condition" interaction was also not statistically significant, F(1,12) =.08, p=.79.

Headache Self-Efficacy Scale. A 2 x 2 RM ANOVA found no main effect for Time, F(1,12) =.11, p=.75 but there was a statistically significant main effect for Condition, F(1,12) =4.96, p=.05. The "Time x Condition" interaction was also not statistically significant, F(1,12) =.46, p=.51.

Headache Specific Locus of Control. A 2 x 2 RM ANOVA found no main effect for Time, F(1,12) =.02, p=.90 and no main effect for Condition, F(1,12) =.00, p=.99. The "Time x Condition" interaction was also not statistically significant, F(1,12) =. 02, p=.90.

Clinically Meaningful Change among Study Completers

Clinical change in the primary outcome measures of headache frequency (30-Day headache self-report), HDI, and BDI was defined as the relative change in symptom frequency and/or severity post-intervention compared to baseline. Clinically meaningful change was assessed using methodology that categorizes clinically meaningful responses to therapy as: (1) full response = ≥50% reduction in depressive or headache symptoms from pre- to post-intervention; (2) partial response = 25-49% reduction; and (3) no response = <25% reduction (Bengtson et al., 2015; Heckman, Heckman et al., 2016). This methodology was used for the completer-only group. (See Table 3 for pre- and post-intervention scores of outcome measures by study arm. See Figures 3, 4 and 5 for clinically meaningful change by outcome measure).

30-Day Headache Self-Report Clinically Meaningful Change

In the CBT group, 3 participants evidenced partial headache frequency responses of 26.3%, 33.3%, and 48% reduction in the number of headache days on the 30-day headache self-report; 1 participant experienced a full headache frequency response with a 56.3% reduction of headache days; and the remaining 3 CBT participants evidenced no headache frequency response. In the SCC group, 1 participant evidenced a partial headache frequency response of 28% reduction of headache days on the 30-day headache self-report. The remaining 6 SCC participants evidenced no headache frequency response or increase in headache frequency.

HDI Clinically Meaningful Change

In the CBT group, 1 participant evidenced a partial disability response of 45% symptom reduction. The remaining 6 CBT participants evidenced no disability response or deterioration of symptoms. In the SCC group, no participants evidenced a disability response or deterioration.

BDI-2 Clinically Meaningful Change

Of the 7 participants who completed the 4-session CBT intervention, 2 participants experienced a partial depression response (38.5% and 42% symptom reduction) and 1 participant experienced a full depression response (77.4% symptom reduction). Three CBT participants did not respond to treatment and 1 CBT participant reported significant symptom deterioration (192% increase in symptoms). Three SCC (n=7) participants experienced a partial depression response over the 4-week study period, 1 experienced no response and 1 reported significant symptom deterioration (113.3% increase symptoms).

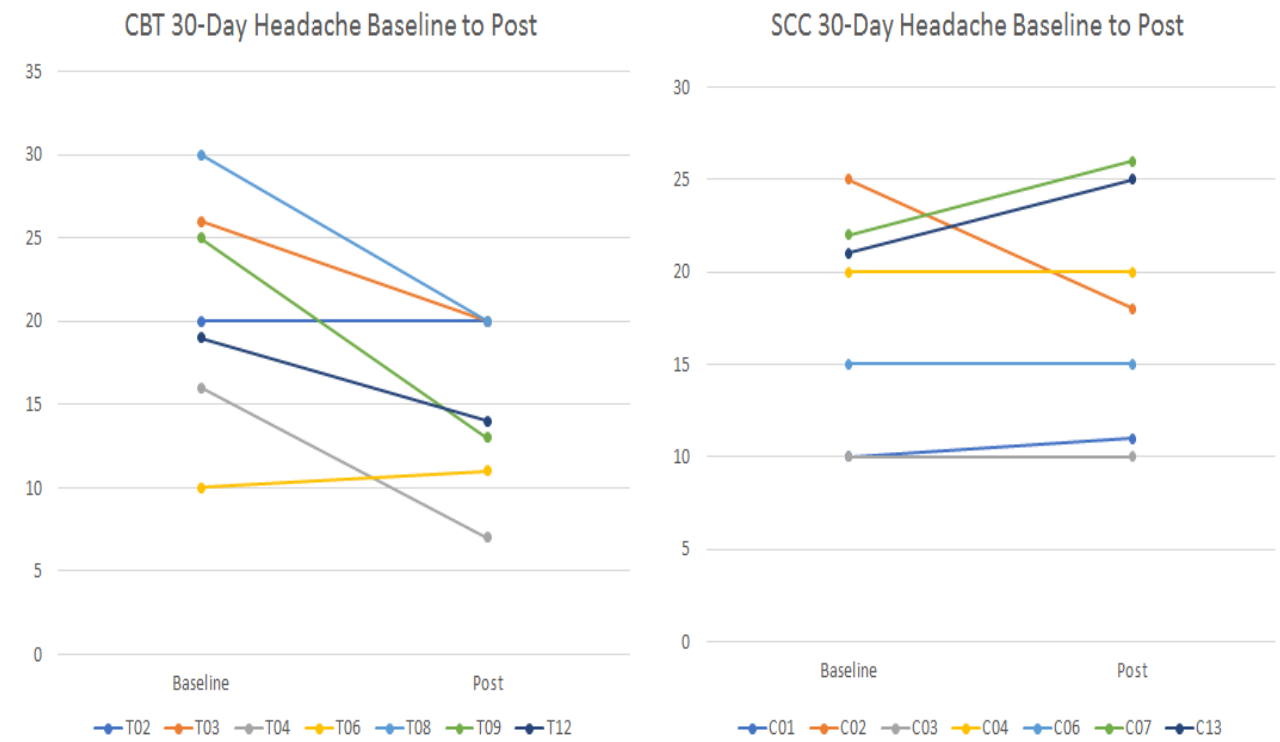


Figure 3. Clinically Meaningful Change in Headache Days from Baseline to Post.

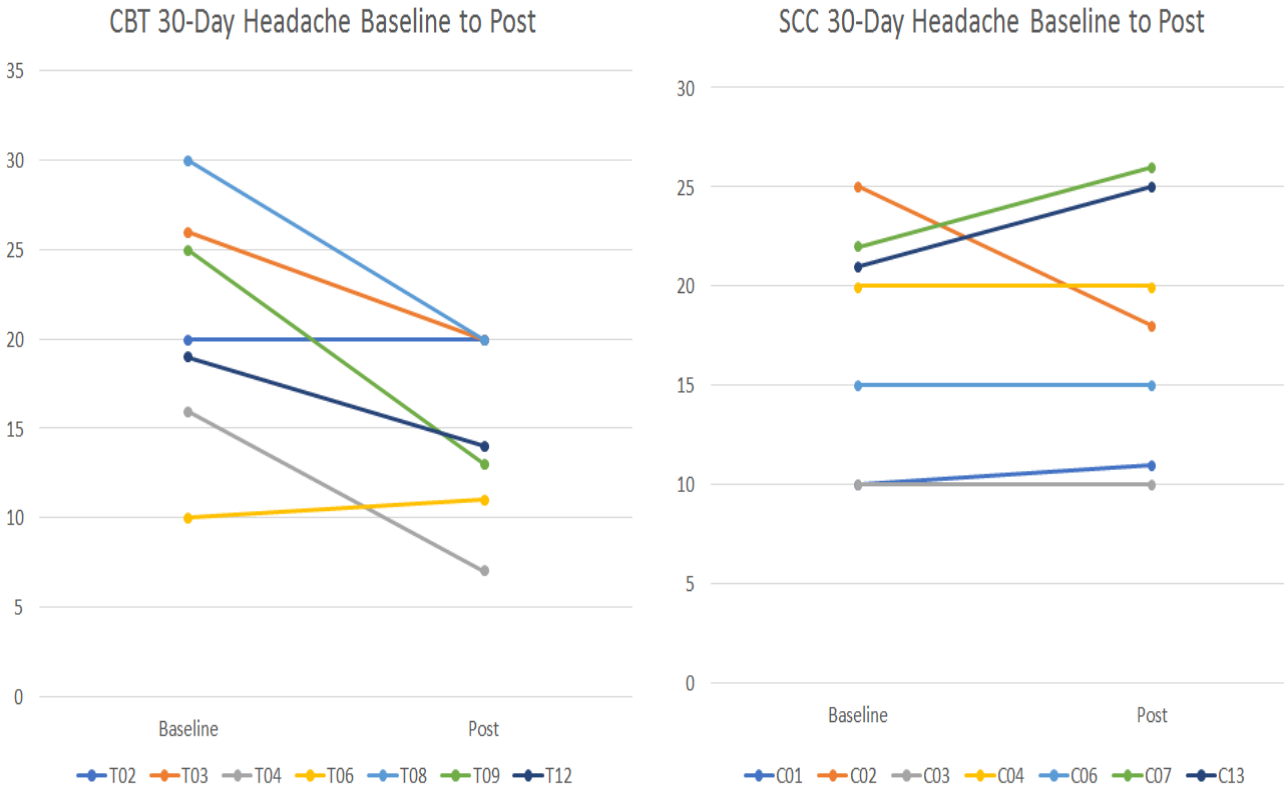


Figure 4. Clinically Meaningful Change in Headache Disability from Baseline to Post.

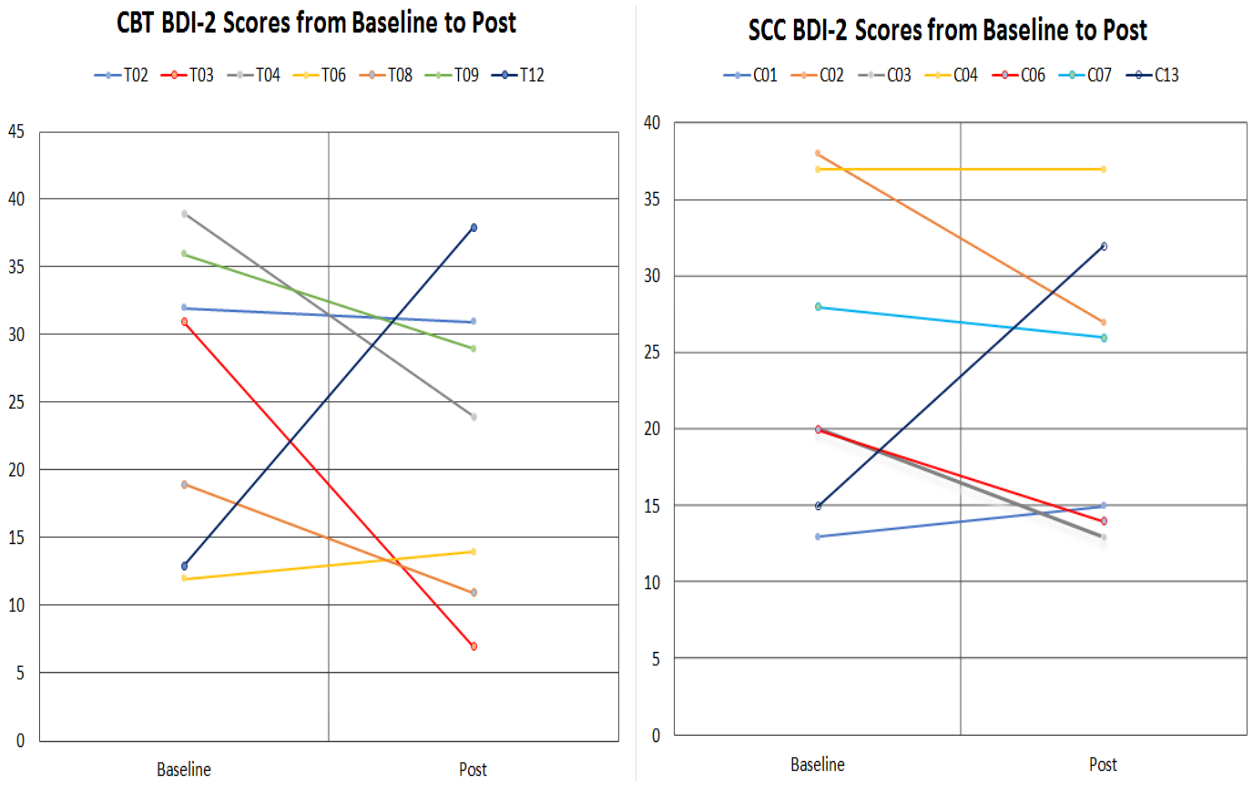


Figure 5. Clinically Meaningful Change in Depression from Baseline to Post.

Table 3. Headache and Psychosocial scores at Baseline and Post-Treatment by study arm.

		Total	Control Group		Treatment Group	
		Post-treatment	Baseline	Post-treatment	Baseline	Post-treatment
Measure	*Total n*	*M (SD)*	*M (SD)*	*M (SD)*	*M (SD)*	*M (SD)*
BDI Total:						
LOCF Imputation	22	26.95 (10.92)	29 (11.25)	28.36 (11.25)	28.09 (9.90)	25.55 (10.93)
Completer-Only	14	22.71 (10.20)	24.43 (10.11)	23.43 (9.54)	26 (11.14)	22 (11.55)
HDI Total:						
LOCF Imputation	22	71 (20.47)	75.27 (20.30)	74 (22.31)	70.73 (20.17)	68 (19.04)
Completer-Only	14	73.43 (19.91)	76.86 (20.52)	74.86 (23.86)	76.29 (17.64)	72 (16.89)
HDI-Emotional:						
LOCF Imputation	22	35.68 (11.03)	38 (11.06)	37.09 (12.88)	33.82 (10.06)	34.27 (9.23)
Completer-Only	14	37.07 (10.85)	39.14 (9.99)	37.71 (13.24)	35.71 (10.42)	36.43 (8.87)
HDI-Functional:						
LOCF Imputation	22	35.32 (10.46)	37.27 (10.44)	36.91 (10.60)	36.91 (11.08)	33.73 (10.58)
Completer-Only	14	36.36 (10.12)	37.71 (11.28)	37.14 (11.54)	40.57 (8.54)	35.57 (9.34)
HSES Total:						
LOCF Imputation	22	191.86 (48.91)	180.91 (53.81)	189.82 (53.22)	197 (45.57)	193.91 (46.70)
Completer-Only	14	185.14 (48.37)	154.43 (43.64)	168.43 (50.81)	206.71 (39.53)	201.86 (42.85)
HSLC Total:						
LOCF Imputation	22	95.09 (20.77)	96.55 (18.57)	97 (22.37)	93.18 (17.81)	93.18 (19.93)
Completer-Only	14	95.36 (22.45)	94.86 (19.63)	95.57 (25.44)	95.14 (17.63)	95.14 (21.08)
MIDAS-A:						
LOCF Imputation[a]	22	40.32 (24.65)	43.45 (25.49)	42.82 (27.12)	61.45 (17.62)	37.82 (22.95)
Completer-Only	14	35.36 (23.01)	45.86 (20.98)	44.86 (24.25)	63 (20.97)	25.86 (18.68)
MGrade Level:						
LOCF Imputation	22	3.86 (.35)	3.55 (.93)	3.91 (.30)	3.55 (1.04)	3.82 (.41)
Completer-Only	14	3.79 (.43)	3.29 (1.11)	3.86 (.38)	3.29 (1.25)	3.71 (.49)
Headache Years						
LOCF Imputation	22	11.21 (12.06)	9.15 (8.00)	–	13.27 (15.24)	–
Completer-Only	14	10.82 (12.41)	8.14 (9.85)	–	13.50 (14.83)	–

Note: M = mean, SD = standard deviation, BDI = Beck Depression Inventory-2, HDI = Headache Disability Inventory, HSES = Headache Self-Efficacy Scale for recurrent headaches, HDI-Emotional = HDI Emotional disability subscale, HDI-Functional = HDI Functional disability subscale, HSLC = Headache Specific Locus of Control for recurrent headaches, MIDAS-A = # of headache days in past 3 months, MGRADE = MIDAS disability level).
[a]MIDAS-A LOCF Imputation data was subjected to a square root transformation to stabilize the variance and meet distribution assumptions of normality.

CHAPTER 5

DISCUSSION

The primary aim of this research was to determine the efficacy of an alternative treatment approach for an underserved and undertreated population with co-occurring depression and chronic headache disorders; and who are at an increased risk for impaired functioning, comorbid psychiatric disorders and reduced quality of life. Another primary aim of this project was to examine the common pathophysiological and bidirectional link between headache disorders and depression—a clinically significant issue in chronic headache management and treatment.

Epidemiological research has determined that headache disorders are the most prevalent of all neurological conditions with significant psychosocial and interpersonal impacts predominantly due to the clinically progressive nature of the conditions, with the literature implicating psychiatric comorbidity as a risk factor for headache chronification (Lipton & Bigal, 2007; Smitherman et al., 2008). Strong epidemiological evidence has established that individuals with a depressive disorder have an increased relative risk for migraine incidence. Bidirectional and cross-sectional associations between migraine and various somatic and psychiatric conditions have been reported within migraine and headache literature (Nimnuan & Srikiatkhachorn, 2011; Frediani & Villani, 2007; Hamel, 2007).

Migraine has long been recognized as associated with a characteristic set of psychiatric disorders, as seen in clinical and general population studies with adults, the most relevant disorder for this study being major depression (Jette et al., 2008; Lanteri-Minet et al., 2005; Torelli et al., 2006). Depression and migraine continue to be treated primarily via

pharmacologics, despite research to the contrary that has shown that traditional headache treatments (medications) have been proven to be less effective in depressed patients (Martin et al., 2013). Despite this finding, integrated pharmacological and psychological treatment interventions continue to be poorly studied in head pain; and few researchers have attempted alternative behavioral treatments through the bidirectional neurological relationship.

Study Findings

Statistical Findings

For both the ITT analyses and the Completer analyses, there was a statistically significant "Time by Condition" interaction on the MIDAS (90-day headache self-report). These findings indicate that the depression intervention had a significant effect on number of headache days reported post-intervention on the MIDAS compared to headache days reported at baseline, over a 90-day period, suggesting the intervention had a positive impact over time (4 weeks). There were no other statistically significant effects for the HDI for either the ITT or Completer analyses. The researchers also hypothesized that relative to their Control counterparts, Treatment participants will experience significantly greater confidence in their ability to manage their headaches.

There was a statistically significant main effect for the HSES in the ITT analyses, but no statistically significant interaction effects for the HSES from the ITT or Completer analyses. This finding shows that there was a significant difference between the CBT group participants and their SCC counterparts regarding reported headache-related self-efficacy from baseline to end-of-study assessment. This indicates that the participants of the intervention arm reported greater self-efficacy towards managing and coping with their headaches compared to their control counterparts, from baseline to end-of-study assessment. Finally, there were no statistically

significant main effects or interaction effects for the BDI-2, HSLC among either the ITT or Completer analyses.

The researchers anticipated there would not be any statistical findings given the sample size – primarily because the focus was on clinically meaningful change among the completers. Overall, CBT completers demonstrated clinically significant reductions in headache days, headache-related disability & depressive symptoms immediately after receiving a 4-session cognitive-behavioral intervention that solely targeted their depression compared to their SCC completer counterparts. (See Table 5 for clinically meaningful changes by outcome measure from baseline to post, and Table 7 for clinically meaningful changes by outcome from baseline to follow up).

Clinically Meaningful Change in Headache Characteristics

With regard to headache frequency, an impressive 4 of 7 CBT completers (57%) experienced a clinically significant reduction on the 30-day self-report from baseline to post-intervention – many of whom went from the chronic headache range (>15 days/month) to episodic range (<14 days/month). Participant T04 demonstrated a full treatment response with a 56.3% reduction in headache days from 16 to 7 days per month. Participant T12 demonstrated a partial treatment response with a 26.3% reduction in headache days from 19 to 14 per month. Participant T08 demonstrated a partial treatment response with a 33.3% reduction in headache days from 30 to 20 days, indicating the frequency remained in the chronic range. Participant T09 demonstrated a partial treatment response with a 48% reduction in headache days from 25 to 13 days per month. Comparatively, only 1 of 7 SCC completers experienced a reduction in headache frequency from the 30-day self-report from baseline to post-intervention – with a

partial response (28%) from 25 to 18 days per month, indicating the frequency remained in the chronic range.

Regarding clinically meaningful change in headache-related disability, 1 CBT participant reported a partial treatment response with 45% symptom reduction on the HDI – which translates to an improvement in headache disability from the range of complete disability to moderate disability. Comparatively, no participants from the Control group experienced a meaningful reduction in headache disability. This indicates that the intervention had a small but positive effect on headache disability across the 4-week intervention compared to no treatment at all.

Looking at reduction in headache characteristics in general, 5 of 7 individuals from the CBT group (71%) reported reduction in frequency and/or disability. In comparison, only 1 participant of the SCC group (14%) achieved any reduction in headache characteristics (frequency only); however, despite this partial reduction in headache frequency for the SCC participant, the number of reported headache days remained in the chronic range. Thus, it can be inferred that the intervention had a small, but positive effect on headache frequency and disability for treatment participants across the 4-week study period compared to the control counterparts.

Clinically Meaningful Change in Depressive Symptoms

Nearly half of the CBT completers (3 of 7) reported a clinically significant reduction (greater than 25%) in depressive symptoms that demonstrated partial to full treatment responses. The degree depressive symptom reduction for CBT participants was remarkable considering the brief nature of the intervention. Participant T03 experienced a full treatment response of 77.4% symptom reduction, which means they moved from the severe range (32) to minimal range (7). Participant T04 experienced a partial treatment response of 38.5% symptom reduction, which

means they moved from the severe range (39) to moderate range (24). Participant T08 experienced a partial treatment response of 42% symptom reduction, which means they moved from the mild range (19) to minimal range (11).

Similarly, 3 of 7 SCC completers also reported partial depressive symptom reductions from baseline to post. Participant C02 reported a partial response in depressive symptom reduction of 28.9% and moved from the severe (38) to moderate (27) range. Participant C06 reported a partial response in depressive symptom reduction of 30% and moved from the moderate (20) to minimal (13) range. Participant C03 reported a partial response in depressive symptom reduction of 35% and moved from the moderate (20) to mild (14) range. Interestingly, two participants reported a significant symptom deterioration in depressive symptoms, on the BDI-2, from baseline to post – indicating a significant depressive relapse. The CBT participant (T12; 192.3% worse) reported symptoms moving from the minimal to severe range and the SCC participant (C13; 113.3% worse) reported symptoms moving from the mild to severe range, from baseline to post.

These finding indicate that the 4-week depression intervention was at least partially effective for nearly half of the CBT completers. However, other factors may be partially responsible for the reduction in depressive symptoms since Control group members also reported partial symptom reduction. Moreover, two participants experienced significant depressive relapses that, although they were unexpected, were not entirely surprising given the characteristics of the clinical population sampled. In retrospect, there are several somatic symptoms associated with depression that also overlap with and/or mimic chronic health conditions, including migraine. This may also help explain the findings related to reported changes in depressive symptoms from baseline to post.

Clinical Implications

These small but promising study findings suggest a feasible alternative to pharmacological migraine treatment by treating the co-occurring depression. Furthermore, this feasible alternative is an easily administered brief intervention that can be conducted at the patients' own local healthcare organization. The ability to provide a free service to a clinical sample such as this, many of whom belong to several marginalized groups, begins to address social issues such as healthcare disparities (i.e. barriers of access to or availability of healthcare facilities and treatment services) – in alignment with my values as a Counseling Health Psychologist. Additionally, the brief duration of the intervention can aid in buy-in for patient's unable to commit to longer behavioral interventions.

More importantly, these findings support the bidirectional hypothesis of a shared pathophysiology of migraine and psychiatric disorders, specifically depression. Research on the burden and prevalence of migraine clearly elucidates migraine as a public heath issue with serious social and economic consequences. Because of its complex nature and etiology, migraine continues to be a poorly understood disease that, consequently, is often undiagnosed, misdiagnosed, and/or undertreated. Subsequently, migraineurs generally do not seek medical treatment; and of those who do, only 4% of migraine sufferers who seek medical treatment seek out headache and pain specialists (MRF, 2017). This alternative treatment is particularly noteworthy in light of the fact that the efficacy of many prophylactic drug treatments is often hampered by side effects (Weeks, 2013; Bendtsen et al., 2010). The baseline characteristics of this study's clinical sample clearly illustrates that migraine is not just a "bad headache" but rather an incapacitating neurological condition that significantly diminishes quality of life.

Study Strengths

Although the study sample was small there are also several notable strengths. The study successfully recruited participants from two geographically and ethnically diverse sites – the first being a small, primary care clinic for uninsured members of a rural Southeastern town and the second was a large healthcare system in an urban Midwestern city. Despite the small sample size, there was still representation of men and members of the LGBT community. The sample consisted primarily of women (86.4%), which is consistent with migraine prevalence since adult women are disproportionately affected, nearly three times higher than men.

Additionally, the study arms demonstrated equal racial diversity among White and Black Americans. Notwithstanding the difficult nature of working with a clinical sample, that is often arduous to retain and subsequently track, the study successfully screened and enrolled 54% of prospective participants. The study completion rate of 63.6% (attrition rate of 36.4%) demonstrates a rate of assessment follow-up at post-intervention that is consistent with trends for depression interventions and dropout rates (e.g. approximately 20% follow up on therapy referrals with a 50% dropout rate; Mohr, 2010). Moreover, the study incorporated multiple intervention outcome data analyses approaches to ascertain differences among completers versus non-completers. Furthermore, all participants were recruited from the community through local healthcare organizations, either a primary care clinic or an urban hospital system.

Lastly, the study interventionists were all advanced psychology doctoral students with a significant amount of training in CBT work to allow tailoring of the intervention for individual circumstances without the intervention devolving into traditional psychotherapy. Interventionists also consulted with the primary student researcher after the initial protocol and intervention delivery training to better anticipate how to address potential issues prior to them arising in

session (e.g. working with highly emotional participants, boundary setting and redirection for participants trying to engage in therapy, etc.). Some of the post-study feedback from interventionists included the following: easy administration of the manual with the ability to tailor the intervention to individuals because of succinct and standardized nature of the intervention; participants appeared to benefit from the intervention, despite its brevity, and provided a good foundation for participants to begin making changes and carry forward the skills and coping strategies they learned in session; the homework was relevant and allowed for efficient use of the session time since participants were prepared.

Study Limitations

The current study had several limitations. The sample size of this pilot study was very small (n =22) especially when considering the size of the completer group (n=14) with respect to the brief duration of the intervention. Although the data collected from participants was self-report, which may have been subject to recall bias (e.g. inaccuracy of the recollections retrieved by study participants regarding events or experiences from the past), social desirability, and demand characteristics (i.e. good-participant role and apprehensive-participant role), pain is subjective and there is no objective means of assessing this construct.

Although data was collected on other services that patients were receiving during the current intervention (e.g. therapeutic and neurological/pharmacologic treatment), this study did not examine the potential group differences or interaction effects these other services may have had on participant outcomes. Of note, there was a clinically significant deterioration of two study participants, one from each study arm, that the researchers had not anticipated and the study measures were unable to explain or address – aside from this occurrence demonstrating a depression relapse. Finally, although the interventionists were well-trained in CBT to deliver the

intervention, the only means of assessing protocol fidelity was through conversational reviews of each module following each session but this was not checked against a more rigourous fidelity checklist.

Future Directions

The goal of this pilot study was to serve as part of the preliminary results to apply for an R21 NIH grant and further explore this issue to advance health research. The R21 is an Exploratory/Development NIH grant that is designed to provide funding for exploratory research that has the potential to lead to advances in health research. Future investigations with a similar clinical population should also endeavor to obtain additional types of data from a medical chart review data (when possible), and collateral information via observer or partner reports of psychological and somatic symptoms, to allow for more robust analyses. Moreover, use of a better depression measure with less somatic symptom overlap and a more rigourous fidelity protocol assessment will strengthen the study's overall rigor and aid with a more accurate account of study findings.

Additionally, there are increasingly more studies demonstrating the effectiveness of tele-health in the treatment of depression for diverse clinical populations. An examination of the effects of a tele-health intervention on this bidirectional relationship would make a meaningful contribution to the literature on alternative treatments. More importantly, a tele-health intervention may help with attrition and better address some of the social justice and health disparity issues such as access to treatment for such an underserved population. Finally, as a Counseling Health Psychologist, I intend for my research to continue to study such health disparities and address related multicultural issues as part of my commitment to being an agent of social justice.

REFERENCES

Alliance for Headache Disorders Advocacy. (2013). Headache disorders fact sheet. Retrieved March 20, 2014 from http://www.allianceforheadacheadvocacy.org/

American Headache Society. (2014). Epidemiology and impact of headaches and migraines pdf. Retrieved July 21, 2014 from http://www.americanheadachesociety.org/assets/1/7/NAP_for_Web_-_Epidemiology___Impact_of_Headache___Migraine.pdf

Antoniades, J., Mazza, D. & Brijnath, B. (2014). Efficacy of depression treatments for immigrant patients: results from a systematic review. *BMC Psychiatry, 14,* 176-187.

Baskin, S., Lipchik, G., & Smitherman, T. (2006). Mood and anxiety disorders in chronic headache. Headache, 46, Suppl 3:S76-87.

Beck, A. (n.d.). History of cognitive therapy. Beck Institute for Cognitive Behavior Therapy. Retrieved January 2, 2015, from http://www.beckinstitute.org/history-of-cbt/

Beck, A., Epstein, N., Brown, G., & Steer, R. (1988). An inventory for measuring clinical anxiety: Psychometric properties. Journal of Consulting and Clinical Psychology, 56, 893–897. doi:10.1037/0022-006x.56.6.893.

Beck, A., Steer, R., Ball, R., &Ranieri, W. (1996). Comparison of Beck Depression Inventories - IA and -II in psychiatric outpatients. *Journal of Personality Assessment, 67(3),* 588–597. doi:10.1207/s15327752jpa6703_13.

Beck, A., Ward, C., Mendelson, M. (1961). Beck Depression Inventory (BDI). Archives of General Psychiatry, 4(6), 561–571. doi:10.1001/archpsyc.1961.01710120031004.

Beck, A., Ward, C., Mendelson, M. (1961). Beck Depression Inventory (BDI). *Archives of General Psychiatry, 4(6),* 561–571. doi:10.1001/archpsyc.1961.01710120031004

Bendtsen, L., Evers, S., Linde, M., Mitsikostas, D., Sandrini, G., & Schoenen, J. (2010). EFNS guideline on the treatment of tension-type headache – report of an EFNS task force. *European Journal of Neurology, 17(11),* 1318-1325. doi:10.1111/j.1468-1331.2010.03070.x.

Bendtsen, L. & Jensen, R. (2011). Treating tension-type headache – an expert opinion. *Expert Opinion on Pharmacotherapy, 12(7)*, 1099-1109. doi: 10.1517/14656566.2011.548806

Bengtson, A., Pence, B. W., O'Donnell, J., Thielman, N., Heine, A., Zinski, A., Modi, R., McGuinness, T., & Gaynes, B. (2015). Improvements in depression and changes in quality of life among HIV-infected adults. AIDS Care, 27(1), 47–53. http://doi.org/10.1080/09540121.2014.946386

Bigal, M., Bigal, J., Betti, M., Bordini, C., & Speciali, J. (2001). Evaluation of the impact of migraine and episodic tension-type headache on the quality of life and performance of a university student population. *Headache, 41*, 710-719.

Bigal, M., Lipton, R., & Stewart, W. (2004). The epidemiology and impact of migraine. *Current Neurology and Neuroscience Reports, 4*, 98–104.

Breslau, N. (1998). Psychiatric comorbidity in migraine. *Cephalalgia, 18*, Suppl 22:56-61.

Breslau, N., Davis, G., Schultz, L., & Peterson, E. (1993). Migraine and major depression: A longitudinal study. *Headache, 34,* 387-393.

Breslau, N., Schultz, L., Stuart, W., Lipton, R., Lucia, V., & Welch, K. (2000). Headache and major depression: Is the association specific to migraine? *Neurology, 54*, 308-313.

Breslau, N., Lipton, R., Stewart, W., Schultz, L., Welch, K. (2003). Comorbidity of migraine and depression: investigating potential etiology and prognosis. *Neurology, 60*, 1308-1312.

Butler, A., Chapman, J., Forman, E., & Beck, A. (2006). The empirical status of cognitive-behavioral therapy: A review of meta-analyses. *Clinical Psychology Review, 26,* 17-31. doi: 10.1016/j.cpr.2005.07.003

Campbell, J., Penzien, D., & Wall, E. (2000). Evidence-based guidelines for migraine headaches: behavioral and physical treatments. The US Headache Consortium. Retrieved December 20, 2014 from http://www.aan.com/professionals/practice/pdfs/g10089.pdf

Castien, R., van der Windt, D., Dekker, J., Mutsaers, B. & Grooten, A. (2009). Effectiveness of manual therapy compared to usual care by the general practitioner for chronic tension-type headache: Design of a randomised clinical trial. *BMC Musculoskeletal Disorders, 10(21),* 1-7. doi:10.1186/1471-2474-10-21

Cathcart, S., Galatis, N., Immink, M., Proeve, M., & Petkov, J. (2014). Brief mindfulness-based therapy for chronic tension-type headache: A randomized controlled pilot study. *Behavioral and Cognitive Psychotherapy, 42(1)*, 1-15. doi: 10.1017/S1352465813000234.

Cujipers, P., Berking, M., Andersson, G., Quigley, L., Kleiboer, A., & Dobson, K. (2013). A meta-analysis of cognitive-behavioural therapy for adult depression, alone and in comparison with other treatments. *The Canadian Journal of Psychiatry, 58(7),* 376-385.

Curry, K. & Green, R. (2007). Prevalence and management of headache in a university undergraduate population. *Journal of the American Academy of Nurse Practitioners, 19*, 378-382. doi:10.1111/j.1745-7599.2007.00237.x

Efird, J. (2011). Blocked randomization with randomly selected block sizes. *International Journal of Environmental Research and Public Health, 8(1)*, 15–20. http://doi.org/10.3390/ijerph8010015.

Ehde, D., Dillworth, T., & Turner, J. (2014). Cognitive-behavioral therapy for individuals with chronic pain: Efficacy, innovations and directions for research. *American Psychologist, 69(2),* 153-166. doi.org/10.1037/a0035747.

Falavigna, A., Teles, A., Braga, G., Conzatti, L., Ruschel, L., & Silva, P. (2013). Association between primary headaches and depression in young adults in southern Brazil. *Revista da Associação Médica Brasileira (Journal of the Brazilian Medical Association), 59(6),* 589-593. doi:10.1016/j.ramb.2013.06.014.

Freitag, F., Lyss, H., & Nissan, G. (2013). Migraine disability, healthcare utilization, and expenditures following treatment in a tertiary headache center. *Baylor University Medical Center Proceedings, 26(4),* 363-367.

French, D., Holroyd, K., Pinell, C., Malinoski, P., O'Donnell, F., & Hill, K. (2000). Perceived Self-efficacy and Headache-Related Disability. *Headache, 40(8)*, 647–656.

Fumal, A. & Schoenen, J. (2008). Tension-type headache: Current research and clinical management. *The Lancet Neurology, 7(1),* 70-83.

Gatchel, R. (2004). Comorbidity of chronic mental and physical health disorders: The biopsychosocial perspective. *American Psychologist, 59,* 792-805.

Gatchel, R. (2013). The biopsychosocial model of chronic pain. In: Clinical Insights: Chronic Pain Unitec House, 2 Albert Place, London N3 1QB, UK (Future Medicine Ltd), p.5-17. eBook ISBN: 978-1-78084-348-3. Accessed January 5, 2015 from http://www.futuremedicine.com/doi/abs/10.2217/ebo.13.469

Gatchel, R. J., & Howard, K. J. (2015, November 9). The Biopsychosocial Approach. Retrieved July 11, 2017, from https://www.practicalpainmanagement.com/treatments/psychological/biopsychosocial-approach?page=0%2C1

Gatchel, R., Peng, Y., Peters, M., Fuchs, P., & Turk, D. (2007). The biopsychosocial approach to chronic pain: Scientific advances and future directions. *Psychological Bulletin, 133(4),* 581– 624. doi:10.1037/0033-2909.133.4.58

Gatchel, R., & Rollings, K. (2008). Evidence-informed management of chronic low back pain with cognitive behavioral therapy. *The Spine Journal, 8(1),* 40–44. doi:10.1016/j.spinee.2007.10.007

Gerrits, M., Vogelzangs, N., van Oppen, P., van Marwijk, H., van der Horst, H., Pennix, B. (2012). Impact of pain on the course of depressive and anxiety disorders. The Journal of Pain,153(2), 429-36. doi: 10.1016/j.pain.2011.11.001. Epub 2011 Dec 10.

Goldberg, J. (2012). Cognitive Behavioral Therapy for Depression: Techniques, Sessions, and More. Retrieved January 2, 2015, from http://www.webmd.com/depression/guide/cognitive-behavioral-therapy-for-depression?page=4

Gupta, S. K. (2011). Intention-to-treat concept: A review. Perspectives in Clinical Research, 2(3), 109–112. http://doi.org/10.4103/2229-3485.83221

Haque, B., Rahman, K., Hoque, A., Hasan, A., Chowdhury, R., Khan, S., Alam, M., Habib, M., & Mohammad, Q. (2012). Precipitating and relieving factors of migraine versus tension type headache. *BMC Neurology, 12(82)*, 1-4. doi:10.1186/1471-2377-12-82

Headache Classification Subcommittee of the International Headache Society. (2004). The International Classification of Headache Disorder--2nd Edition. Cephalagia, 24(Suppl. 1), 1-151. Retrieved August 21, 2014, from https://www.ichd-3.org/wp-content/uploads/2016/08/ihc_II_main_no_print.pdf.

Heckman, B. & Holroyd, K. (2006). Tension-type headache and psychiatric comorbidity. *Current Pain and Headache Reports, 10*, 439–447.

Heckman, T., Heckman, B., Andeson, T., Lovejoy, T., Markowitz, J. Shen, Y. & Sutton, M. (2016). Tele-interpersonal psychotherapy acutely reduces depressive symptoms in depressed HIV-infected rural persons: A randomized clinical trial. Behavioral Medicine, 1-11. doi: 10.1080/08964289.2016.1160025. [Epub ahead of print].

Holroyd, K., O'Donnell, F., Stensland, M., Lipchik, G., Cordingley, G., & Carlson, B. (2001). Management of chronic tension-type headache with tricyclic antidepressant medication, stress management therapy, and their combination: A randomized controlled trial. *Journal of the American Medical Association, 285*, 2208–2215.

Hooten, W., Timming, R., Belgrade, M., Gaul, J., Goertz, M., Haake, B., Myers, C., Noonan, M., Owens, J., Saeger, L., Schweim, K., Shteyman, G., & Walker, N. *Institute for Clinical Systems Improvement.* Assessment and Management of Chronic Pain. Updated November 2013.

Hopewell, S., Hirst, A., Collins, G. S., Mallett, S., Yu, L.-M., & Altman, D. G. (2011). Reporting of participant flow diagrams in published reports of randomized trials. Trials, 12, 253. http://doi.org/10.1186/1745-6215-12-253.

Jacobson, G, Ramadan, N., Aggarwal, S., & Newman, C. (1994). The Henry Ford Hospital Headache Disability Inventory (HDI). Neurology. 44(5), 837-842.

Jensen, R. (2003). Diagnosis, epidemiology, and impact of tension-type headache. *Current Headache Reports, 2*, 455-459.

Jette, N., Patten, S., Williams, J., Becker, W., & Wiebe, S. (2008). Comorbidity of migraine and psychiatric disorders – A national population-based study. *Headache, 48*, 501-516.

Kang, M., Ragan, B., & Park, J. (2008). Issues in outcomes research: An overview of randomization techniques for clinical trials. *Journal of Athletic Training, 43(2),* 215–221.

Katsarava, Z., Buse, D., Manack, A., & Lipton, R. (2012). Defining the Differences Between Episodic Migraine and Chronic Migraine. Current Pain and Headache Reports, 16(1), 86–92. doi:10.1007/s11916-011-0233-z

Kroenke, K., Spitzer, R., & Williams, J. (2001). The PHQ-9: Validity of a brief depression severity measure. *Journal of General Internal Medicine, 16*, 606-613. doi:10.1046/j.1525-1497.2001.016009606.x

Kwong, W., Landy, S., Braverman-Panza, J., Rosen, J., Hutchinson, S., & Burch, S. (2007). A migraine disease management program in the primary care setting: Impact on patient quality of life and productivity loss. *Journal of Clinical Outcomes Management, 14(6),* 332-338.

Lachin, J., Matts, J., Wei, L. (1988). Randomization in clinical trials: Conclusions and recommendations, *Controlled Clinical Trials, 9(4)*, 365-374. ISSN 0197-2456, http://dx.doi.org/10.1016/0197-2456(88)90049-9. Retrieved February 2016 at http://www.sciencedirect.com/science/article/pii/0197245688900499.

Lanteri-Minet, M., Radat, F., Chautard, M., & Lucas, C. (2005). Anxiety and depression associated with migraine: influence on migraine subjects' disability and quality of life, and acute migraine management. *Journal of Pain, 118*, 319–326.

Ligthart, L., Gerrits, M., Boomsma, D., & Pennix, B. (2013). Anxiety and depression are associated with migraine and pain in general: An investigation of the interrelationships. The Journal of Pain, 14(4), 363-370

Lipton, R. & Bigal, M. (2007). Ten lessons on the epidemiology of migraine. *Headache, 47*(Suppl 1), S2-S9.

Lipton, R., Bigal, M., Diamond, M., Freitag, F., Reed, M., & Stewart, W. (2007). The American Migraine Prevalence and Prevention Advisory Group. Migraine prevalence, disease burden, and the need for preventive therapy. *Neurology, 68,* 343–349.

Lipton, R., Hamelsky, S., Kolodner, K., Steiner, T., & Stewart, W. (2000). Migraine, quality of life, and depression: A population-based case–control study. *Neurology, 55*, 629–635.

Lipton, R., Stewart, W., Sawyer, J., Westhead, E., & Edmeads, J. (2001). Clinical utility of a new instrument assessing migraine disability: the Migraine Disability Assessment (MIDAS) questionnaire. *Headache, 41(9*), 854–861.

Lipton, R., Stewart, W., Diamond, S., Diamond, M., & Reed, M. (2001). Prevalence and burden of migraine in the United States: data from the American Migraine Study II. *Headache*, *41(7)*, 646-657.

Loerch, B., Szegedi, A., Kohnen, R., & Benkert, O. (2000). The primary care evaluation of mental disorders (PRIMEMD) German version: a comparison with the CIDI. *Journal of Psychiatric Research, 34*, 211-220. doi:10.1016/S0022-3956(00)00005-4.

Martin, N., Holroyd, K., Penzien, D. (1990). The headache-specific locus of control scale: Adaption to recurrent headaches. *Headache, 30,* 729-734.

Martin, N. Holroyd, K. & Rokiki, L. (1993). The headache self-efficacy scale: : Adaption to recurrent headaches. *Headache, 33,* 244-248.

Martin, P., Meadows, G., Piterman, L., Sharman, M., Reece, J., & Milgrom, J. (2013). Cognitive Behavioral Therapy Effective for Comorbid Chronic Headache, Depression. Retrieved December 23, 2014, from http://www.psychcongress.com/article/cognitive-behavioral-therapy-effective-comorbid-chronic-headache-depression-12514

Merikangas, K., Merikangas, J., & Angst, J. (1993). Headache syndromes and psychiatric disorders: association and familial transmission. Journal of Psychiatric Research, 27, 197–210.

Miller, S. & Matharu, M. (2014). Migraine is underdiagnosed and undertreated. *The Practitioner, 258(1774),* 19–24, 2-3.

Mohr, D. C., Ho, J., Duffecy, J., Baron, K. G., Lehman, K. A., Jin, L., & Reifler, D. (2010). Perceived Barriers to Psychological Treatments and Their Relationship to Depression. Journal of Clinical Psychology, 66(4), 394–409. http://doi.org/10.1002/jclp.20659

Molnar, F. J., Hutton, B., & Fergusson, D. (2008). Does analysis using "last observation carried forward" introduce bias in dementia research? CMAJ : Canadian Medical Association Journal, 179(8), 751–753. http://doi.org/10.1503/cmaj.080820

Morley, S. (2011). Efficacy and effectiveness of cognitive behavior therapy for chronic pain: Progress and some challenges. *The Journal of Pain, 152(3, Suppl)*, S99-S106. doi:10.1016/j.pain.2010.10.042

Morley, S., Eccleston, C., & Williams, A. (1999). Systematic review and meta-analysis of randomized controlled trials of cognitive behavior therapy and behavior therapy for chronic pain in adults, excluding headache. *The Journal of Pain, 80(1-2)*, 1-13. doi:10.1016/S0304-3959(98)00255-3

National Health Service: Migraine Action. (2010). Migraine and the trigeminal nerve. Retrieved February 1, 2015, from http://www.nhs.uk/ipgmedia/national/migraine action/assets/migraineandthetrigeminalnerve.pdf

Nemade, R., Staats Reiss, N., & Dombeck, M. (2007). Biology of Depression - Neurotransmitters. Retrieved January 31, 2015, from http://www.mentalhelp.net/poc/view_doc.php?type=doc&id=12999&cn=5

Nimnuan, C., & Srikiatkhachorn, A. (2011). Migraine: Psychiatric comorbidities. Retrieved January 25, 2015, from http://www.medmerits.com/index.php/article/migraine_psychiatric_comorbidities

Powers, S., Gilman, D., & Hershey, A. (2006). Suggestions for a biopsychosocial approach to treating children and adolescents who present with headache. *Headache, 46*[Suppl 3], S149-S150.

Rampello, L., Alvano, A., Chiechio, S., Malaguarnera, M., Raffelle, R., Vecchio, I., & Nicoletti, F. (2004). Evaluation of the prophylactic efficacy of amitriptyline and citalopram, alone or in combination, in patients with comorbidity of depression, migraine, and tension-type headache. *Neuropsychobiology, 50(4)*, 322-328.

Rasmussen, B. (2001). Epidemiology of headache. *Cephalagia, 21,* 774-777.

Richards, D. & Richardson, T. (2012). Computer-based psychological treatments for depression: A systematic review and meta-analysis. *Clinical Psychology Review, 32*, 329-342.

Richter, P., Werner, J., Heerlein, A., Kraus, A., & Sauer, H. (1998). On the validity of the Beck Depression Inventory: A review. Psychopathology, 31(3), 160–168. doi:10.1159/000066239

Sammons, M. (2005). Treatment of head pain with psychotropics. *Professional Psychology: Research and Practice, 36(6)*, 611-614.

Saper, J. (2008). Chronic daily headache: transformational migraine, chronic migraine, and related disorders. *Current Neurology and Neuroscience Reports, 8(2),* 100-107.

Scher, A., Bigal, M., & Lipton, R. (2005). Comorbidity of migraine. *Current Opinion in Neurology, 18*, 305-310.

Scher, A., Lipton, R., & Stewart, W. (2002). Risk factors for chronic daily headache. *Current Pain Headache Report, 6,* 486-491.

Scher, A., Midgette, L., & Lipton, R. (2008). Risk factors for headache chronification. *Headache, 48,* 16-25. doi: 10.1111/j.1526-4610.2007.00970.x

Scher, A., Stewart, W., Ricci, J., & Lipton, R. (2003). Factors associated with the onset and remission of chronic daily headache in a population-based study. *The Journal of Pain*, *106(1-2),* 81 -89.

Sealed Envelope Ltd. (2016). Create a blocked randomisation list. [Online] Available from: https://www.sealedenvelope.com/simple-randomiser/v1/lists [Accessed 17 Feb 2016].

Shapiro, R. & Goadsby, P. (2007). The long drought: The dearth of public funding for headache research. *Cephalalgia, 27*, 991–994.

Shulman, R. (2013, May 17). Psychiatric Aspects of Headache, Pain and Depression [Webinar]. In *National Headache Foundation* Webinar Series. Retrieved January 30, 2015 from https://www.youtube.com/watch?v=4n3BTs6-fFU&list=PL6bpjkbYtk-MBnNjRF7pAxzBJI52_XdPd.

Silberstein, S. (1998). Comprehensive management of headache and depression. *Cephalaia, 18*, 50-55.

Silberstein, S., Holland, S., Freitag, F., Dodick, D., Argoff, C., & Ashman, E. (2012). Evidence-based Guidelines Update: Pharmacologic Treatments and NSAIDs and Other Complementary Treatments for Episodic Migraine Prevention in Adults. Report of the Quality Standards Subcommittee of the American Academy of Neurology and the American Headache Society. Accessed January 5, 2015.

Smitherman, T., Burch, R., Sheikh, H., & Loder, E. (2013). The prevalence, impact, and treatment of migraine and severe headaches in the united states: A review of statistics from national surveillance studies. *Headache, 53(3),* 427–436. doi: 10.1111/head.12074.

Smitherman, T., McDermott, M. & Buchanan, E. (2011). Negative impact of episodic migraine on a university population: Quality of life, functional impairment, and comorbid psychiatric symptoms. *Headache, 51*(4), 581-589. doi:10.1111/j.1526-4610.2011.01857.x

Smitherman, T., Maizels, M., & Penzien, D. (2008). Headache chronification: Screening and behavioral management of comorbid depressive and anxiety disorders. *Headache, 48,* 45-50.

Sørensen Høifoøt, R., Strøm, C., Kolstrup, N., Eisemann, M., & Waterloo, K. (2011). Effectiveness of cognitive behavioural therapy in primary health care: a review. Family Practice, 28(5), 489-504. doi: 10.1093/fampra/cmr017

Souza-e-Silva, H., Rocha-Filho, P. (2011). Headaches and academic performance in university students: A cross-sectional study. *Headache, 51,* 1493-1502. doi:10.1111/j.1526-4610.2011.02012.x

Spitzer, R., Kroenke, K., Williams, J., and the Patient Health Questionnaire Primary Care Study Group (1999). Validation and Utility of a Self-report Version of PRIME-MD: The PHQ

Primary Care Study. *Journal of the American Medical Association, 282*(18), 1737-1744. doi:10.1001/jama.282.18.1737

Spitzer, R., Williams, J., Kroenke, K., et al. (1994). Utility of a new procedure for diagnosing mental disorders in primary care: the PRIME-MD 1000 study. *Journal of the National Medical Association*, *272*, 1749-1756.

Stewart, W., Lipton, R., Dowson, A., & Sawyer, J. (2001). Development and testing of the Migraine Disability Assessment (MIDAS) Questionnaire to assess headache-related disability. *Neurology, 56(6 Suppl 1)*, S20–S28.

Stewart, W., Roy, J., & Lipton, R. (2013). Migraine prevalence, socioeconomic status, and social causation. Neurology, 81(11), 948–955. doi:10.1212/WNL.0b013e3182a43b32

Streiner, D. & Geddes, J. (2001). Intention to treat analysis in clinical trials when there are missing data. Evidence-Based Mental Health, (4), 70-71.

Sun-Edelstein, C., & Mauskop, A. (2012). Complementary and alternative approaches to the treatment of tension-type headache. *Current Pain and Headache Reports, 16(6),* 539-544. doi: 10.1007/s11916-012-0295-6.

Szumilas, M. (2010). Explaining Odds Ratios. *Journal of the Canadian Academy of Child and Adolescent Psychiatry*, *19*(3), 227–229.

Tepper, D. (2013). Chronic migraine. *Headache, 53(2),* 423-424. doi:10.1111/head.12036

Test, T., & Test C. (2014). The biopsychosocial model, stress, and the efficacy of mindfulness therapy. *WebmedCentral PSYCHOLOGY, 5(11),* 1-6. Accessed January 5, 2015 from http://www.webmedcentral.com/wmcpdf/Article_WMC004765.pdf

Tobin, D. L., Holroyd, K. A., Baker, A., Reynolds, R. V., & Holm, J. E. (1988). Development and clinical trial of a minimal contact, cognitive-behavioral treatment of tension headache. *Cognitive Therapy and Research, 12(4)*, 325-339. doi:10.1007/BF01173301

Torelli, P., Lambru, G., Manzoni, G. (2006). Psychiatric comorbidity and headache: clinical and therapeutical aspects. *Journal of the Neurological Sciences, 27*, Suppl 2:S73-76.

Turk, D., & Monarch, E. (2002). Biopsychosocial perspective on chronic pain. In D. Turk & R. Gatchel (Eds.), Psychological Approaches to Pain Management: A Practitoner's Handbook (2nd ed., pp. 3-29). New York, NY: Guilford Press.

Twomey, C., O'Reilly, G., & Byrne, M. (2015). Effectiveness of cognitive behavioural therapy for anxiety and depression in primary care: A meta-analysis. *Family Practice, 32(1),* 3-15.

Verhagen, A., Damen, L., Berger, M., Passchier, J., & Koes, B. (2009). Behavioral treatments of chronic tension-type headache in adults: Are they beneficial? *CNS Neuroscience and Therapeutics, 15(2)*, 183-205.

Weeks, R. (2013). Application of behavioral therapies in adult and adolescent patients with chronic migraine. Neurological Sciences, 3411-17.

Williams, A., Eccleston, C., Morley, S. (2012). Psychological therapies for the management of chronic pain (excluding headache) in adults. *Cochrane Database of Systematic Reviews, 11,* 1-111. DOI: 10.1002/14651858.CD007407.pub3.

World Health Organization. (2012). *Depression.* http://www.who.int/mental_health/management/depression/flyer_depression_2012.pdf?ua=1 (accessed on 21 January 2015).

Table 4. Psychometric attributes of outcome measures compared to a larger, diverse, clinical sample

Scale	Britton CBT Study (N = 22)	Heckman et al. (2011)* (N = 230)
Internal Consistency (α)		
Headache Disability Inventory (HDI) Total	0.79	W = 0.94, B = 0.95
HDI Emotional disability subscale	0.70	W = 0.89, B = 0.92
HDI Functional disability subscale	0.82	W = 0.88, B = 0.91
Headache Specific Locus of Control (HSLC) Total	0.82	—
Health Care Provider subscale	—	W = 0.82, B = 0.84
Internal Locus of Control subscale	—	W = 0.84, B = 0.86
Chance Locus of Control subscale	—	W = 0.82, B = 0.81
Headache Self-Efficacy Scale (HSES) Total	0.70	W = 0.85, B = 0.88
Migraine Disability Assessment (MIDAS) Total	0.96	—
Beck Depression Inventory-II (BDI-2) Total	0.68	—

*Heckman et al. (2011) psychometric data reported by race. W=White (N=173), B=Black (N=114).

Table 5. Clinically Meaningful Change by Outcome Measure, from baseline to post

30-Day headache	Baseline	Post	Pre-Post Change
C01	10	11	10% worse
C02	25	18	**28% improvement***
C03	10	10	0% change
C04	20	20	0% change
C06	15	15	0% change
C07	22	26	18.2% worse
C13	21	25	19.1% worse
T02	20	20	0% change
T03	26	20	23.1% improvement
T04	16	7	**56.3% improvement****
T06	10	11	10% worse
T08	30	20	**33.3% improvement***
T09	25	13	**48% improvement***
T12	19	14	**26.3% improvement***
BDI-2 Total	**Baseline**	**Post**	**BDI-2 Pre-Post Change**
C01	13	15	15.4% worse
C02	38	27	**28.9% improvement***
C03	20	13	**35% improvement***
C04	37	37	0% change
C06	20	14	**30% improvement***
C07	28	26	7.1% improvement
C13	15	32	**113.3% worse*****
T02	32	31	3.1% improvement
T03	31	7	**77.4% improvement****
T04	39	24	**38.5% improvement***
T06	12	14	16.7% worse
T08	19	11	**42% improvement***
T09	36	29	19.4% improvement
T12	13	38	**192.3% worse*****
HDI Total	**Baseline**	**Post**	**HDI Pre-Post Change**
C01	46	36	21.7% improvement
C02	82	64	21.95% improvement
C03	54	54	0% change
C04	94	94	0% change
C06	90	90	0% change
C07	100	100	0% change
C13	72	86	19% worse
T02	96	92	4% improvement
T03	88	48	**45% improvement***
T04	72	82	13.8% worse
T06	62	74	19% worse
T08	90	84	6% improvement
T09	80	74	7.5% improvement
T12	46	50	8.7% worse

Clinically meaningful baseline to post change scores were calculated by (Xpre-Xpost)/Pre.
* = partial response (25-49% reduction of symptoms/headache days)
** = full response (50%+ reduction of symptoms/headache days)
*** = full negative/inverse response (100%+ worsening of symptoms/headache days)

Table 6. Headache and Psychosocial scores at Baseline, Post-Treatment and 4-week Follow-Up by study arm.

		Total	Control Group			Treatment Group		
		Follow-Up	*Baseline*	*Post*	*Follow-Up*	*Baseline*	*Post*	*Follow-Up*
Measure	*Total n*	*M (SD)*	*M (SD)*	*M (SD*	*M (SD)*	*M (SD)*	*M (SD)*	*M (SD)*
BDI Total:								
LOCF Imputation	22	28.05 (10.45)	29 (11.25)	28.36 (11.25)	28.45 (11.7)	28.09 (9.90)	25.55 (10.93)	27.64 (9.6)
Completer-Only	14	23.25 (10.51)	24.43 (10.11)	23.43 (9.54)	22.17 (10.74)	26 (11.14)	22 (11.55)	24.33 (11.17)
HDI Total:								
LOCF Imputation	22	71.09 (21.75)	75.27 (20.30)	74 (22.31)	72.55 (23.92)	70.73 (20.17)	68 (19.04)	69.64 (20.41)
Completer-Only	14	71 (22.78)	76.86 (20.52)	74.86 (23.86)	70.33 (28.15)	76.29 (17.64)	72 (16.89)	71.67 (18.65)
HDI-Emotional:								
LOCF Imputation	22	35.55 (11.69)	38 (11.06)	37.09 (12.88)	36.18 (13.01)	33.82 (10.06)	34.27 (9.23)	34.91 (10.82)
Completer-Only	14	35.33 (12.25)	39.14 (9.99)	37.71 (13.24)	35 (14.3)	35.71 (10.42)	36.43 (8.87)	35.67 (11.20)
HDI-Functional:								
LOCF Imputation	22	35.55 (10.69)	37.27 (10.44)	36.91 (10.60)	36.36 (11.66)	36.91 (11.08)	33.73 (10.58)	34.73 (10.13)
Completer-Only	14	35.67 (10.98)	37.71 (11.28)	37.14 (11.54)	35.33 (14.12)	40.57 (8.54)	35.57 (9.34)	36 (8.1)
HSES Total:								
LOCF Imputation	22	180.73 (57.49)	180.91 (53.81)	189.82 (53.22)	167.36 (66.19)	197 (45.57)	193.91 (46.70)	194.09 (46.56)
Completer-Only	14	171.33 (55.55)	154.43 (43.64)	168.43 (50.81)	142.67(51.81)	206.71 (39.53)	201.86 (42.85)	200 (46.18)
HSLC Total:								
LOCF Imputation	22	96.18 (17.93)	96.55 (18.57)	97 (22.37)	99.91 (16.1)	93.18 (17.81)	93.18 (19.93)	92.45 (19.63)
Completer-Only	14	94.67 (17.96)	94.86 (19.63)	95.57 (25.44)	96.67 (14.15)	95.14 (17.63)	95.14 (21.08)	92.67 (22.36)
MIDAS-A:								
LOCF Imputation[a]	22	41.23 (23.71)	43.45 (25.49)	42.82 (27.12)	35.45 (25.65)	61.45 (17.62)	37.82 (22.95)	47 (23.71)
Completer-Only	14	40 (21.63)	46 (22.98)	48.17 (24.77)	34.67 (23.07)	61 (22.23)	28.5 (18.97)	45.33(20.7)
MGrade Level:								
LOCF Imputation[a]	22	3.82 (.50)	3.55 (.93)	3.91 (.30)	3.82 (.41)	3.55 (1.04)	3.82 (.41)	3.82 (.60)
Completer-Only	14	3.75 (.62)	3.29 (1.11)	3.86 (.38)	3.83 (.41)	3.29 (1.25)	3.71 (.49)	3.67 (.82)
Headache Years								
LOCF Imputation	22	11.21 (12.06)	9.15 (8.00)	–	–	13.27 (15.24)	–	–
Completer-Only	14	10.82 (12.41)	8.14 (9.85)	–	–	13.50 (14.83)	–	–

Note: M = mean, SD = standard deviation, BDI = Beck Depression Inventory-2, HDI = Headache Disability Inventory, HSES = Headache Self-Efficacy Scale for recurrent headaches, HDI-Emotional = HDI Emotional disability subscale, HDI-Functional = HDI Functional disability subscale, HSLC = Headache Specific Locus of Control for recurrent headaches, MIDAS-A = # of headache days in past 3 months, MGRADE = MIDAS disability level.

[a]MIDAS-A and MGrade LOCF Imputation data was subjected to a square root transformation to stabilize the variance and meet distribution assumptions of normality; input values are the original descriptive values within the 90-day context.

Table 7. Clinically Meaningful Change by Outcome Measure, from Baseline to 4-week Follow-Up

30-Day headache	Baseline	Post	4-week F/U	Change from Baseline
C01	10	11	8	20% improvement
C02	25	18	18	**28% improvement***
C03	10	10	9	10% improvement
C04	20	20	16	20% improvement
C06	15	15	10	**50% improvement****
C07	22	26	14	**36% improvement***
C13	21	25	—	19.1% worse
T02	20	20	—	0% change
T03	26	20	25	3.85% improvement
T04	16	7	17	6.25% worse
T06	10	11	17	**70% worse*****
T08	30	20	14	**53.3% improvement****
T09	25	13	20	20% improvement
T12	19	14	16	15.8% improvement
BDI-2 Total	**Baseline**	**Post**	**4-week F/U**	**BDI-2 Pre-Post Change**
C01	13	15	10	23.1% improvement
C02	38	27	18	**52.6% improvement****
C03	20	13	14	**30% improvement***
C04	37	37	40	8.11% worse
C06	20	14	24	20% worse
C07	28	26	27	3.57% improvement
C13	15	32	—	**113.3% worse*****
T02	32	31	—	3.1% improvement
T03	31	7	37	19.4% worse
T04	39	24	35	10.3% improvement
T06	12	14	13	8.3% worse
T08	19	11	16	15.8% improvement
T09	36	29	31	13.9% improvement
T12	13	38	14	3.3% worse
HDI Total	**Baseline**	**Post**	**4-week F/U**	**HDI Pre-Post Change**
C01	46	36	32	**30.4% improvement***
C02	82	64	74	9.8% improvement
C03	54	54	40	**25.9% improvement***
C04	94	94	82	12.8% improvement
C06	90	90	96	6.7% worse
C07	100	100	98	2% improvement
C13	72	86	—	19% worse
T02	96	92	—	4% improvement
T03	88	48	96	9.1% worse
T04	72	82	78	8.3% worse
T06	62	74	58	6.5% improvement
T08	90	84	84	6.7% improvement
T09	80	74	70	12.5% improvement
T12	46	50	44	4.3% improvement

Clinically meaningful baseline to post change scores were calculated by (Xpre-Xpost)/Pre.

* = partial response (25-49% reduction of symptoms/headache days)

** = full response (50%+ reduction of symptoms/headache days)

*** = full negative/inverse response (50%+ worsening of symptoms/headache days)

— = no follow up data reported; change from baseline is calculated with post data only.

TREATMENT MANUAL FOR BRIEF COGNITIVE-BEHAVIORAL THERAPY FOR DEPRESSION

Interventionist's Manual

Individual Format

Ashley J. Britton, MSEd

Bernadette D. Heckman, PhD

Adapted from the Group Therapy Manual for Cognitive-behavioral Treatment of Depression
Ricardo F. Muñoz, Ph.D.
Jeanne Miranda, Ph.D.

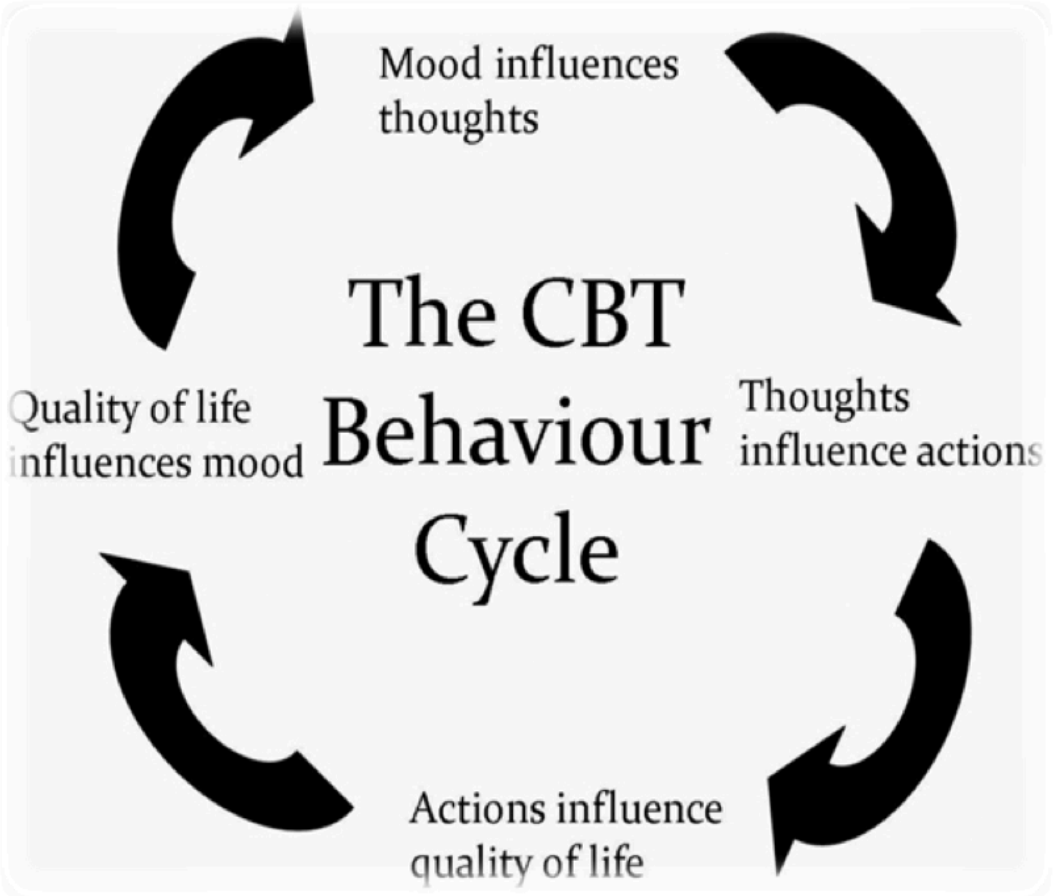


TABLE OF CONTENTS	PAGE

Overview: Cognitive-Behavioral Therapy for the Treatment of Depression

Research studies and meta-analyses conducted over the past two decades have consistently shown that CBT is efficacious in treating psychological disorders such as depression and anxiety, among others (Butler, Chapman, Forman & Beck, 2006). Cognitive-behavioral therapy is based on the interrelationship of thoughts, actions, and feelings. To work with feelings of depression, this model establishes the importance of identifying the thoughts and actions that influences mood. In this manner the participant learns to gain control of his/her feelings.

CBT is a collaborative process that builds a skill-set which enables an individual to (1) become aware of their thoughts and emotions, (2) identify how thoughts, behaviors, and situations influence their emotions, and (3) change distorted thinking and dysfunctional behaviors to improve their feelings. Homework assignments and skill acquisition are the fundamental components of CBT which differentiates it from other talk therapies.

Brief CBT compresses the material, thereby reducing the number of sessions from 12-20 into 4-8 sessions. This brief model is concentrated with specific interventions within a limited number of sessions. This model offers the incentive for the participant and interventionist to work effectively and efficiently. While some variability may exist, the following intervention outline with session-by-session examples should be adhered to as closely as possible, for the sake of treatment standardization. (Cully & Teten, 2008).

The intervention is divided into four modules that will address several aspects of depression. The first two modules address how thoughts influence mood. The third module addresses how daily activities that affect mood. The last module addresses how interactions or relationships with others affect our mood. A description of each module is provided on the corresponding pages within the table of contents.

Sample Session Outline:

A. Briefly review the participants' mood and/or physical functioning (5 min)

- To gauge progress when eliciting responses, consider any discrepancies and ask for explanations for mood improvement or decline.

B. Review homework from the previous session (10 min)

- Reviewing HW allows for reinforcement and troubleshooting between-session learning. It also allows for assessment of participant skill acquisition.

C. Bridge the discussion from the previous session with the current session (5-10 min)

- Check for understanding of previous content while reinforcing what was learned – this is essential to the participants' improvement beyond the intervention.

D. Set the agenda for the current session and prioritize items (5 min)

- Consult the agenda items for the current session and prioritize the session with anything the participant may also wish to discuss.

E. Discuss agenda items and assign homework (35-40 min)

- Start with the first and most important item and assign HW that is directly related to the content that was discussed.
    - **The mood gauge should be assigned each session**, to help the participant track any changes, along with any other assignments.
    - If you are running short on time, inform the participant that you will discuss the other items at the next session.

F. Summarize the current session and exchange feedback (5-10 min)

- Clarify and remind the participant of the thoughts he/she presented and how they have changed as a result of the in-session exercise. Then summarize the main points of the entire session. For session(s) 3 and/or 4, you may ask the participant to summarize instead.
- Lastly, encourage and motivate the participant to continue working towards change by exchanging feedback about the sessions, skills, or progress.

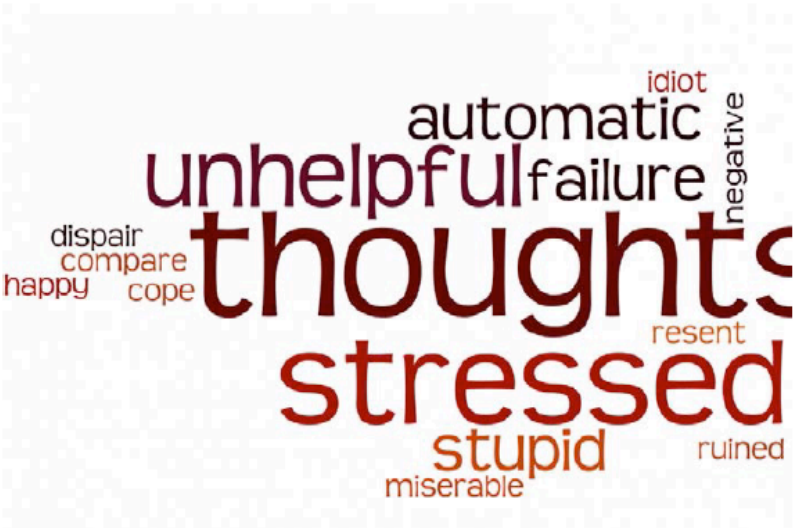
Module I: Thoughts → *How our THOUGHTS affect our mood*

(Pre-Session Introduction & Sessions 1-2)

The purpose of this module is to present information about how our thoughts influence our mood. The pre-session introduction can either be conducted by phone prior to the first scheduled session or can be added 15-30 minutes prior to the start of the first session.

For participants in the intervention condition, the pre-session introduction establishes the structure and purpose of the intervention sessions. The time and day of the sessions will be established, as well as rules for therapy and limits of confidentiality. It is important that participants are clear on the limits and scope of confidentiality since this can have an effect on the type and quality of the therapeutic relationship. This pre-session introduction also begins a dialogue on depression: what it is and how the participant experiences it. The interventionist also presents the purpose of the first module, which is to understand how our thoughts influence our mood. Thoughts are defined in this session.

The first 2 sessions will work with different types of thinking errors and dysfunctional thoughts associated with depression, as well as how they can be disputed and modified to improve our mood. In-session exercises are used to identify thinking errors. The design of these sessions provide the participant with strategies for increasing positive thoughts and decreasing unhealthy or dysfunctional negative thoughts, and thus, decreasing depressive symptoms.



PRE-SESSION MEETING – *INTRODUCTION*

I. *Provide an overview of the day's session.*

Today's session has several goals:

A. Get to know each other better
B. Discuss the rules for the sessions
C. Learn what depression is
D. Learn how your thoughts affect the way you feel
E. Introduce Mood Gauge and Assign Homework #1

II. *Introduce yourself.* If the participant doesn't respond you can share information similar to information you want the participant to share by modeling.

III. *Encourage the participant to share personal information such as:*

- Where he/she was born, information about his/her history or development
- Things about his/her family
- The school he/she attends
- What his/her principal interests are (goals, likes and dislikes, hobbies)
- Things about him/herself that they consider important

IV. *Ask about their main problems* (e.g. primary difficulties). You can also ask: "What would you like to change or improve about your life?"

RULES FOR TREATMENT SESSIONS

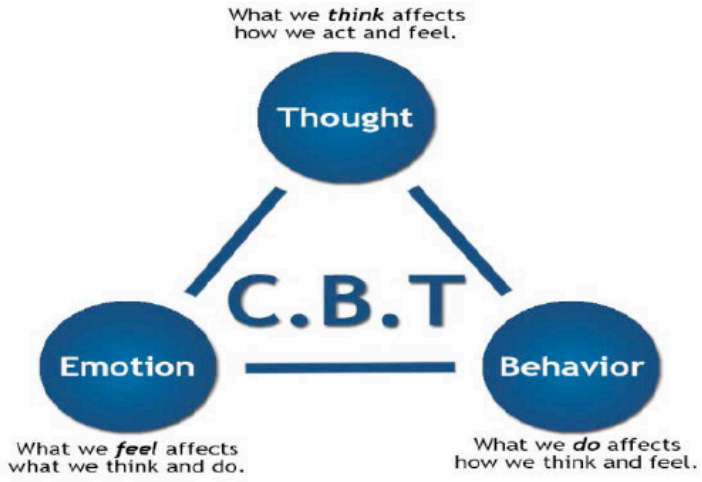
1. Be punctual and arrive a few minutes early to each session.
2. Come every week! This treatment is a commitment you make with yourself and your interventionist.
3. Keep a positive attitude.
4. Do the Homework! By practicing what you will learn in these sessions, you can determine whether these skills can help you control your symptoms of depression once the treatment sessions have ended.
5. What you talk about in session is confidential. However, it is ok if you what to share what you have learned with other people.
6. Try to be as honest as possible, and express yourself just as you are and how you feel.
7. Turn off your phone or put it on "silent" once you come into the therapy so it does not interrupt the session.

Understanding Depression

V. The purpose of this session is to introduce you to the therapy in which you are going to participate. The kind of skills we provide are called "*Cognitive-Behavioral Therapy*"
   - "Cognitive" refers to our thoughts.
   - "Behavioral" refers to our actions.
   - Depression has most to do with our feelings.

We can learn to gain more control over them and improve our mood (feel better) by identifying thoughts and actions that affect our feelings.

Use this diagram **Understanding Depression** (also in Appendix A) to explain CBT.



   - This treatment for depression consists of 8 therapy sessions.
   - We will focus on what is going on in your life right now, in the present.
   - Therapy is focused on how to control depression in practical ways that can be used now and in the future.

The four sessions are divided into three modules or parts:

- How your thoughts affect your mood. (2 sessions)
- How your actions affect your mood. (1 session)
- How your relationships affect your mood. (1 session)

In this type of treatment, we try to teach people practical things they can use in their daily lives. We expect that the most important effect of this treatment will be to learn to understand and manage the things that affect your mood (how you feel).

VI. What is depression? What does it mean to be depressed?

Ask this question in a manner that will encourage the participant to share about their personal experience. Then integrate the following information with what they have shared.

"Depression" is a word that is use in many ways, and can mean:

- A *feeling* lasting a few minutes
- A *mood* lasting a few hours or a few days
- A *clinical condition* that:
  - Lasts for at least 2 weeks
  - Causes strong emotional distress/suffering
  - Makes it difficult to function & carry out daily activities

The treatment for this intervention will focus on treating clinical depression.

VII. *Using the participants' answers from the previous section, present the following symptoms of clinical depression while integrating the information they have shared.*

Persons with clinical depression have **5 or more** of the following symptoms:

- Feeling depressed or down nearly every day
- Not being interested in or unable to enjoy activities you used to enjoy
- Change in appetite and/or weight (more or less than is usual for you)
- Noticeable changes in sleep (trouble falling asleep, sleeping too much/little, waking often/too early, unable to fall back asleep)
- Changes in your movement (either too fidgety/restless or slowed down)
- Feeling tired all the time or becoming easily fatigued
- Feeling worthless or guilty
- Difficulty concentrating, thinking and/or making decisions
- Frequent thoughts of death or hurting yourself (either suicide or non-suicidal injury)

VIII. *The following questions can be used to guide a discussion on how CBT can be applied to the participants' particular experience or situation:*

- What types of thoughts do you have when you feel depressed or sad?
- What do you do when you feel depressed?
- How do you get along with others when you feel depressed?
- What do you think causes your depression?

IX. *Introduce the concept of how our thoughts affect our mood (how we feel):*

- Having certain kinds of thoughts can make you feel more or less depressed. By "thoughts" we mean "things that we tell ourselves."
  - Thoughts can have an effect on your body

- Thoughts can have an effect on your actions (what you do)
- Thoughts can have an effect on your mood (how you feel)

Provide a good example of how a thought can have an effect on your body, actions and mood.

Example: You're walking down a deserted street and you see a person walking quickly behind you. He looks serious, he is looking at you and you think that he is going to mug or rob you. Immediately, your body, your actions and your mood react to this thought. You start sweating, your heart races, and you feel a knot in your stomach. You start looking over your shoulder and walking faster. You feel nervous, afraid. The person reaches you, and quickly walks past you, getting farther and farther away. You then think he was just in a hurry.

X. A good way to think about this type of therapy is that you will learn specific ways to change your thoughts and your actions so that you feel better.

XI. *Explain the purpose of the intervention:*

The purpose of this intervention is to treat depression by teaching you different ways to better control how you feel. The four goals we want to work towards are:

1. To lessen or eliminate feelings of depression
2. To shorten the time you feel depressed
3. To learn ways to prevent or avoid getting depressed again
4. To feel more in control of your life

XII. *Pre-Session Meeting Closure*

Close this Pre-Session Introduction on depression by reviewing how we understand depression. You should use the diagram – Understanding Depression (Appendix A) – using information the participant has shared during the session.

ASK: Do you have any questions or comments about what we talked about today?

Proceed to next page to begin Session 1.

SESSION 1 – *HOW THOUGHTS AFFECT YOUR MOOD*

I. Thoughts affect mood:

Specific types of thinking make a difference in your mood.

1. Some thoughts make it **more** likely that you will become depressed.
2. Other thoughts make it **less** likely that your will become depressed.

II. What are thoughts?

Thoughts are ideas that we tell ourselves.

1. We talk in our own heads all of the time, but we are not always aware of it.
2. It is helpful to think of thoughts as ***things*** that have a real effect on our bodies and our minds.

III. What is depressed thinking like?

A. INFLEXIBLE:

For example, a depressed person might think: "I'll never get better."
Flexible thoughts that keep us from being depressed might be: "If I go to therapy, I am at least trying to feel better."

B. JUDGMENTAL:

A depressed person might think: "I'm a failure."
A flexible thinker may say, "Yes, I've failed at some things but that doesn't mean I'm a failure."

IV. What is *non-depressed* thinking like?

A. CHANGEABLE:

Depressed: "I **always** have been and **always will** be a coward."
Flexible: "I am afraid in **some** situations **sometimes**."

B. WHAT WE DO *VS.* WHO WE ARE:

Depressed thinker: "I was born to feel bad."
Flexible thinker: "I am doing things that have me down right now."

C. HOPE FOR CHANGE:

Depressed thinker: "Nothing has ever helped me."
Flexible thinker: "Nothing I have tried yet has helped, but this is new and the time might be right for me to start feeling better."

V. Differentiating Types of Thinking:

A. ***Constructive vs. Destructive thinking:***

1. Constructive thinking helps us **build ourselves** up and put ourselves together.
   a. For example, "I can learn to control my life to get more of what I want" is a constructive thought.
2. Destructive thinking **tears us apart**.

a. For example, you could think "I am no good at all" or "I did everything wrong raising my kids " or "I've made so many mistakes."

B. ***Necessary vs. Unnecessary thinking:***

1. Necessary thinking reminds us of the things we have to do.
   a. For example: "I must remember to fill out the Mood Gauge before I go to sleep tonight.
2. Unnecessary thinking doesn't change things, yet makes us feel bad.
   a. For example: "There is going to be an earthquake soon." Or "This country is going to be ruined."

C. ***Positive vs. Negative thinking:***

1. Positive thinking makes us feel better.
   a. For example, "Things are rough right now, but at least I'm here doing something to help myself."
2. Negative thinking makes us feel worse.
   b. For example, "It's just no use."

Homework:

(A) Mood Gauge. *Model how to complete the assignment and practice completing it together based upon the participants' mood yesterday.*

o This gauge allows us to evaluate the intensity of our mood at the end of each day.
o This activity will be completed each week. We want to see how you feel each day and how your mood changes over the course of this therapeutic intervention.
o At the end of each day, rate how you felt or generally how your mood was that day. For example, if your day was your "worst" you should circle number 1, if it was a regular day circle number 4 and if it was your "best" day, circle number 7.

(B) Thought Chart. *Track and Record your thoughts.*

o At the end of each day, read the list of negative and positive thoughts and mark each thought you had. Add up the total number of positive and negative thoughts. Look for a pattern between the types of thoughts and your mood for that day.

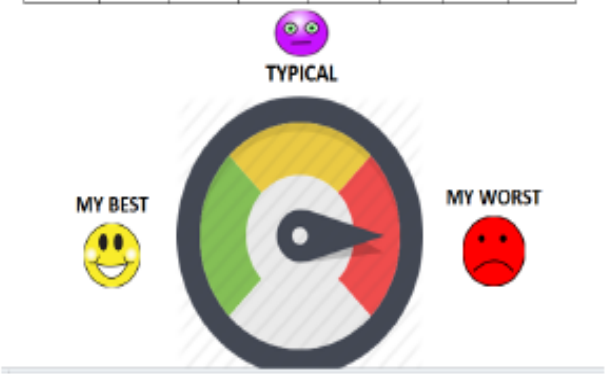
Refer to the Mood Gauge (Appendix B) and Thought Charts (Appendix D) in your manual. Have them bring the completed worksheets to next week's session for discussion.

Mood Gauge

Week Number _____

Name _____ Week Starting: _____

Gauge	Mon	Tues	Wed	Thurs	Fri	Sat	Sun
My Worst	1	1	1	1	1	1	1
Much Worse	2	2	2	2	2	2	2
Worse than Usual	3	3	3	3	3	3	3
Typical	4	4	4	4	4	4	4
Better than Usual	5	5	5	5	5	5	5
Much Better	6	6	6	6	6	6	6
My Best	7	7	7	7	7	7	7



Participant ID/ Name: _____

Thought Chart

Week Number _____ Week Starting _____

Mark an X next to the positive thoughts you had each day & total them at the bottom.

Daily Thoughts	Mon	Tues	Wed	Thurs	Fri	Sat	Sun
I can do better							
Today is a beautiful day							
I will learn to be happy							
My life is meaningful							
I deserve to be given credit							
Though things are bad now, they'll improve							
I did a good job							
I feel really good today							
This is fun							
I chose the best solution for this situation							
I am a good person							
I am optimistic/hopeful about my future							
I have a right to be happy							
I like to read/draw							
I handled this situation well							
I get along well with others							
I worked hard, so I deserves a break							
I am considerate towards others							
I have enough time to do things I want							
I am a good person							
I am honest							
This is interesting							
I can handle a crisis as well as anyone else							
I am lucky							
I am responsible and dependable							
My experiences have prepared me for the future							
I am intelligent							
I am attractive							
I am important to my family							
I always find a way to persevere through a difficult situation							
I am likeable							
Bad things happen but I am not a bad person							
Total Positive Thoughts:							

End of Session 1. Proceed to next page to begin Session 2.

SESSION 2 – *HOW THOUGHTS AFFECT YOUR MOOD cont.*

REVIEW:

1. **Mood Gauge**
2. **Thought Chart** → What are some thoughts you had this past week?
3. What type of depressed thinking do you typically engage in?

1. **Recognizing "Distorted Thinking"** *(Participant Illustration sheet in Appendix C)*

A. ***Polarized (All-or-Nothing) Thinking:***

1. You place people or situations in "either/or" categories, things are either "black-or-white," with no shades of gray or allowing for the complexity of most people and situations.
   a. Ex: We have to be perfect or we're a failure – there is no middle ground. If your performance falls short of perfect, you see yourself as a total failure.

B. ***Overgeneralizing:***

1. You make an overall negative conclusion beyond the current situation.
   a. Ex: "My spouse/partner didn't kiss me when they came home today; they don't love me anymore."

C. ***Catastrophizing:***

1. Predicting only negative outcomes for the future; and expect disaster to strike, no matter what. We begin to think in terms of *"ifs"* or *"what if"* scenarios.
   a. Ex: "If I fail this final, my life will be over/ruined!"

D. ***Discounting the Positive:***

1. Telling yourself that the good things that happen to you do not count.
   a. Ex: Your coworker tells everyone how great you are at baking, but you think she is exaggerating and just being nice.

E. ***Mental Filter (Tunnel Vision):***

1. Focusing your attention exclusively on, or seeing only, the negatives of a situation.
   a. Ex: only focusing on the fact that you have diabetes and not noticing the nice, sunny day or compliments people have given you.

F. ***Magnification & Minimization (Making More or Less of Things):***

1. Emphasizing your mistakes as greater than they are, while down playing positive things in a situation.
   a. Ex: "My boss/professor wants to talk to me about my presentation, so I know it was horrible and I failed."

G. ***Emotional Reasoning (Taking Your Feelings Too Seriously):***

1. We believe that what we feel must be true automatically. You assume that your unhealthy emotions reflect the way things really are – "I feel it, therefore it must be true.
   a. Ex: "If I feel stupid and boring, then I must be stupid and boring."

H. ***Shoulds/Musts (Perfectionism):***

1. Critical, moral imperatives (rules) that we put on ourselves and others. → What you believe people should do or say.
   a. Ex: "I should be a better person" or "I should quit eating dessert until I lose weight."
2. You may try to motivate yourself with "shoulds" or "oughts".
   i. Saying these "shoulds/oughts" to yourself make you feel guilty. → Doing things as best as you can or because you want to is better than out of guilt.
   ii. Thinking these "shoulds" about other people, you feel angry and let down by them when they don't do something as you expect them to.

I. ***Labeling Yourself or Others:***

1. Labeling someone or something without getting more information about the person or situation first.
   a. Ex: "Only delinquents would do something like that, my child would never do something I disapproved of."

J. ***Personalization (Self-Blame):***

1. You blame yourself for things that you may not have been able to control; or think that everything others do is a direct reaction to you.
   a. For example, something bad happens to one of your children or friends and you believe it was your fault because you couldn't prevent it.
   b. This typically manifests as, "If only I had done [insert action], then that wouldn't have happened."
      i. Usually these are things out of your control
      ii. Or when it is within your control you tend to ruminate over what happened and how it was your fault vs. "Yes I made a mistake but..."

2. Learning How to Disrupt Negative Thinking

I. INCREASING THOUGHTS THAT IMPROVE YOUR MOOD

1. **Increasing the number of good/positive thoughts in your mind.**
   a. Make a list of good thoughts about yourself and about life.

   Provide the participant with a blank sheet of paper to do this exercise and discuss it afterwards.

2. **Celebrate Personal Successes**

Most of the things we do are not noticed by others. Therefore, it is important for us to notice them and give ourselves credit for doing them.

a. For example: "I made it to therapy today, even though it was raining."

Ask the participant to identify a personal success or reason to congratulate themselves.

3. **Time Out!!**
When we feel nervous we can take a break and mentally give ourselves a time out.
a. Pause. Let your mind relax, let your mind take a break.
Allow your body to feel at peace. Feeling at peace can give you energy.

Lead the participant in a relaxation exercise after discussing this point (Appendix D). Pick the exercise you feel most comfortable leading. You can use sounds or music to help relaxation.

4. **Future Projection**
Imagine yourself taking the steps to move toward a time when things will be better.

Ask the participant to imagine his/her future in 1, 5 and 10 years. Encourage him/her to imagine it as detailed as possible [i.e., places, people, activities, etc.] The exercise can be oral or written.

II. DECREASING THOUGHTS THAT MAKE YOU FEEL BAD

1. **Interrupting Your Thoughts**
When a thought is ruining our mood:
a. Identify it.
b. Tell yourself: "This thought is ruining my mood so I am going to substitute it with a positive one or I am going to change it."

2. **Planned Worrying Time**
Schedule "worry time" each day so you can concentrate completely on necessary thinking and leave the rest of the day free of worry.
a. "Worry time" can be ten to thirty minutes long.

3. **Exaggerate Your Problems and Laugh!**
a. Have a good sense of humor.
b. Try laughing at your own worries.
c. Sometimes that can take the sting out of them.
Ask the participant to share the most embarrassing moment they have had.

4. **Consider the Worst Scenario**
Often, vague fears about what could happen make us more depressed than thinking things through and facing the worst possibilities.
a. Remember that the worst that can happen is only one of many possibilities.
b. Just because it is the worst, it is not the one most likely to happen.

5. **Training Yourself**

Just as we can help someone to do something difficult by coaching or instructing them, we can help ourselves by coaching ourselves. This is what we mean by learning to feel better.

a. Think about how you want the situation to turn out.
b. Is the outcome realistic?
c. Think about what steps are necessary to reach your goal.
d. Recognize that by doing this, you are trying to control your depression.

III. CHALLENGING NEGATIVE THOUGHTS WITH THE → A-B-C-D METHOD

When you feel depressed ask yourself what you are thinking, then talk back to the particular thought that is hurting or bothering you.

A → is the **A**ctivating event (what happened)

B → is the **B**elief (what you tell yourself is happening)

C → is the **C**onsequence (what you feel as a result of your thought)

D → is the way you **D**ispute the thought (challenge negative thoughts & create alternative positive thoughts)

Using the examples, help the participant understand and begin to work from the ABCD method. Then have the participant share an experience and help them work from this method.

Example A:

A → My best friend did not call me back.

B → Some beliefs you may have about this: "She doesn't want to be my friend anymore" or "You can't trust anyone"

C → Consequently, you felt mad, sad and hopeless. Another consequence is that when you next spoke with your friend, you treated them badly.

D → I can dispute these thoughts like this: "She may be busy and has not yet checked her messages"

Example B:

A → I have an F in two classes and it's already mid-semester.

B → I'm stupid, I can't do anything right. I'm going to have to repeat the 10th grade. I'm never going to be able to study or work at what I want.

C → I felt sad, frustrated and mad.

D → I still have the chance to find opportunities to pull up my grades, such as doing extra credit work or finding a tutor. I might have to repeat the class during the summer, but it doesn't mean I won't graduate, or be able to do what I want. I'm smart and there are some classes that are harder for me than others.

Using the worksheet titled **Working with the ABCD Method** *(Appendix E) in the manual, use a situation that the participant has brought up in therapy to practice the ABCD method.*

IV. COMMON THOUGHTS THAT EXACERBATE DEPRESSION

Generate a discussion in which you and the participant change or modify the following thoughts to more positive and flexible ones.

A. What is wrong with these statements?

1. "I should be loved and approved of by everyone."
2. "I should always be able to do things well and work hard all of the time to feel good about myself."
3. "Some people are bad and should be punished."
4. "I will feel awful if things don't go the way that I want them to go."
5. "Other people and things I cannot change make me unhappy."
6. "I should worry about bad things that could happen."
7. "I can never be happy if I don't have someone to love me."
8. "I can't change the way I am; I was raised this way."
9. "I must feel sad when people I care about are having bad times."
10. "It will be awful if I don't do the right thing."

V. MORE PRACTICE WITH THE ABCD METHOD

A. Review:

A is the **A**ctivating event (what happened).
B is the **B**elief (or the thought that you tell yourself about what happened).
C is the **C**onsequence (what we feel after we have thought about what happened).
D is the **D**ispute (the way you feel can change the thought so that you do not feel so sad or angry).

VI. MODULE I REVIEW: What have you learned about yourself and your depression?

Homework:

- o Continue with the Mood Gauge & Thought Chart.
- o Practice some of the strategies discussed in today's session to increase positive thoughts and decrease negative thoughts.
- o Practice using the A-B-C-D Method

End of Session 2. Proceed to next page to begin Module II: Session 3.

Module II: Activities → *How our ACTIVITIES affect our mood*
(Session 3)

The main purpose of this module is that the participant increase his/her control over his/her life and learn to identify alternatives that will allow him/her to have more freedom and choices. Session 3 allows the participant to associate participation in pleasurable activities with depressive symptoms. There is a discussion on how the presence of depression can limit participation in pleasant activities, which in turn, increases depressive symptoms. During this session enjoyable activities are defined and obstacles for engaging in them are identified.

This module also works with how learning to establish clear goals can help decrease depression. Steps in establishing reachable goals are taught and practiced in session. Together with the interventionist, goals and activities are established that will help improve the participant's mood.

SESSION 3 – *HOW ACTIVITIES AFFECT YOUR MOOD*

REVIEW:

1. Homework – Mood Diary & Thought Charts
2. A-B-C-D Method
3. Purpose of Module II: working with activities and how they affect how we feel.

I. HOW OUR ACTIVITIES AFFECT OUR MOOD

Through our activities we can tell how we feel. However, the fewer pleasant activities people do, the more depressed they feel.

- Do you notice that you stop doing things because you feel depressed?

OR

- Do you notice you feel depressed because you stop doing things?

Most likely, the answer is BOTH!

- The less you do, the more depressed you feel. The more depressed you feel, the less you do. This becomes a "vicious cycle."

II. BREAKING THE CYCLE WITH PLEASURABLE ACTIVITIES

A. These activities can be described as:
- enjoyable
- inspiring
- meaningful
- relaxing
- rewarding

III. DEFINING PLEASURABLE ACTIVITIES

A. Many are everyday activities (i.e. reading, listening to music, watching TV, talking to friends, surfing the internet, dancing, getting a massage, etc.)

B. These activities differ for everyone so try not to compare what you are or are not doing with other people.

C. They are things you can do that make you feel happy and often make you feel relaxed.

D. While they can be special activities, they do not have to be for you to enjoy them.
   a. Consider this analogy: Just as the body needs an adequate level of nutrition (i.e., vitamins, minerals), the mind needs an adequate level of positive stimulation.

E. **In-Session Exercise:** Remember the last pleasant activity you did.
   1. What did you do and how long ago did you last do it?
   2. What enjoyment did you get from it?

3. How do your pleasant activities personally affect your mood?

IV. RECALLING PLEASURABLE ACTIVITIES

A. It is difficult to remember pleasant things, especially when we are depressed.

B. **In-Session Exercise:** Referring to the activities list *(See Appendix)*, have the participant identify three activities they consider pleasant.

What is currently preventing you from doing pleasant activities?
Ask for specific examples of obstacles to doing pleasant activities.

V. OBSTACLES TO ENJOYING ACTIVITIES

A. Our Thoughts:

a. What types of thoughts help you enjoy an activity?
b. What thoughts prevent you from enjoying an activity?
c. Have you ever enjoyed an activity that you thought you wouldn't?

B. Other People:

a. Have other people made it difficult for you to enjoy an activity?

VI. CLARIFYING THE ROLE OF ACTIVITIES ON MOOD

Enjoyable activities can help control your mood:

A. Telling yourself to "feel better" won't change the way you feel.

B. Engaging in activities will change the way that you are feeling.

C. Adequate levels of enjoyable activities keep us emotionally healthy.

1. Balancing things you have to do with things you like to do maintains good moods!

D. As we often have more control over the things we want to do, it is important to keep them in mind and do them!

VII. FINDING AND ENJOYING FREE ACTIVITIES

Pleasurable activities just don't appear or happen on their own.

A. Planning and scheduling activities is important towards breaking the cycle and a way to gain control over your life.

Ask the participant to think of a few things they can do for free. Refer the participant to the Things to Do for Free List.

B. Engaging in an activity doesn't require you to wait until you "feel like doing something".

1. Simply choose to do something and do it or go along with a friend's plan.
2. You may be surprised that you can still enjoy something you didn't think you would.
3. By actively participating you can influence your mood with your activities.
4. The more you practice this, the greater control you can achieve over your mood.

VIII. PLANNING TO OVERCOME DEPRESSION

A. To overcome depression:

1. Set "SMART" goals
2. Notice the positive things that you do

3. Reward yourself

B. KEEPING SIGHT OF YOUR GOALS

1. Setting goals is critical to overcoming depression
   a. Make clear and concrete goals so that you can tell when you have reached them
   b. Vague versus "SMART" goals:

VAGUE GOALS	→	CLEAR "SMART" GOALS
Be less depressed	→	Increase positive activities to feel less depressed
Be a good parent	→	Spend 1 hr per day doing something with your child
Be a better friend	→	Spend 2 hrs per week doing something with your friend
Be happy	→	Spend X hrs per week doing something you enjoy
Lose weight	→	Walk 30 min per day 3 days per week
Eat healthier	→	Portion food, eat fresh, consume 10% less calories

C. SETTING REALISTIC GOALS

1. What is and is not realistic is hard to determine ahead of time.
2. What is unrealistic at one time may be realistic at another.
3. However, if you find that you cannot meet **most** of your goals, then chances are that they are unrealistic for you **at this time**.

D. CREATING A PLAN TO ACHIEVE YOUR GOALS

1. Break down large goals into small steps.
2. Make each step towards the bigger goal attainable.
3. If your goal is to learn to be a good bowler, you may begin by finding out where the nearest bowling alley is and what the hours are that you may bowl there.
4. you may need to enjoy old activities in new ways.
5. you may learn to enjoy new interests to replace the old ones.
6. you can learn to develop abilities which you have not used before.

E. DEFINING YOUR GOALS

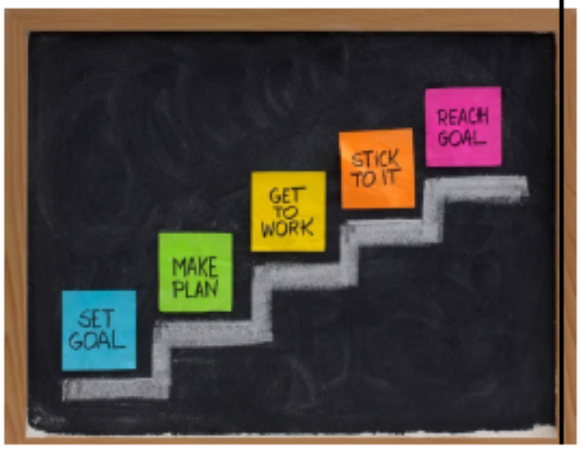
1. What are the obstacles you feel in reaching them?

E. DEFINING YOUR GOALS

1. What are the obstacles you feel in reaching them?

F. TIME MANAGEMENT

1. Make a list of tasks/goals to accomplish for the week.
2. Assign each task a priority:
   - "**H**" tasks have the highest priority
   - "**M**" tasks are the next priority
   - "**L**" tasks are the lowest priority
3. Schedule an "**H**" tasks in your week.



a. Is there room for any **M** or **L** tasks?
b. If not, just do the "**H**" tasks.

4. Intentionally schedule time for enjoyable activities each week.
   a. Do you have balance between tasks you MUST do vs. WANT to do?
5. Practice what works best for you.
   a. Remember: the more options you have, the more choices you have.

G. FUTURE PLANNING

1. Today we will think about individual goals, that is, goals that involve only yourself.
2. **In-Session Exercise:**
   a. Look at each of these types of Individual Goals & note which ones have already been met and which still need to be met:

Maslow's Basic Needs	Individual Goals
Physiological: food, water, shelter, sleep, clothes, breathing	**Lifestyle/ Economic**
Safety: health, employment, stability, resources (free of constant danger)	**Spiritual/Religious/Philosophical**
Love & Belonging: friendship, family, intimacy	**Educational / Vocational**
Self-Esteem: confidence, achievement, respect	**Health/Medical**
Self-Actualization: morality, creativity, spontaneity, acceptance (your potential)	**Creative/ Recreational**

H. MAIN TYPES OF GOALS

1. **Short-Term Goals**:
   a. things that you would like to do soon
      i. time frame: within 3-6 months, but less than 1 year
   b. these goals are often stepping stones towards meeting longer-term goals
2. **Long-Term Goals**:
   a. things you would like to do at some point in the future
      i. time frame: within 1-3 years, but often 5-10 years
   b. includes family, career and lifestyle goals
3. **Lifetime Goals**:
   a. things you most care about accomplishing in your life
      i. time frame: 10years or more

HOMEWORK:

1. Mood Gauge
2. Continue using A-B-C-D method
3. Complete the Enjoyment Prediction Worksheet *(Appendix)*
   a) For each day of the week, write down **one** activity that you plan to do in the first column.
   b) In the second column write down how much you expect to enjoy this activity using a percentage. For example:
      - 0% would mean that you would not enjoy this activity at all;
      - 50% would mean that you would enjoy this activity a moderate amount;
      - 100% would mean that you would enjoy this activity very much.
   c) The most important part of this assignment is completing each activity.
   d) After you have completed the activity, write down how much you actually enjoyed doing it using a percentage.
   e) Note any patterns in the comments column to discuss next week
4. Create 3 Personal Goals (Short, Long-Term & Lifetime) with the steps and deadlines to accomplish each goal *(See Appendix)*

End of Session 3. Proceed to next page to begin Module III: Session 4.

Module III: Relationships → *How our RELATIONSHIPS affect our mood*

(Sessions 4)

This final module introduces the concept of how our relationships affect our mood. Social support and how it helps us confront difficult situations is discussed. The participant learns to identify and strengthen their social support networks. The last session integrates themes from the previous modules. The interventionist together with the participant examines how thoughts affect the activities, social support and relationships the participant engages in. Exercises are used to teach assertive communication skills that will help the participant establish healthy satisfying relationships.

The therapeutic process ends reconsidering and integrating the main themes of each module. In the final session, an evaluation of the therapy experience is carried out with the participant to identify strengths and successes achieved. Recommendations related to follow up and areas to continue working on are discussed with the participant.

SESSION 4 – *HOW RELATIONSHIPS AFFECT YOUR MOOD*

REVIEW:
1. Homework
a. Mood Gauge
b. Activity Planning
c. Goal Setting
2. Module Introduction: how relationships and contact with people affect our mood

I. Higher levels of depression are related to:
A. Less contact with people.
B. Feeling uncomfortable with people.
C. Being more quiet, talking less.
D. Being less assertive, that is, not expressing your likes or dislikes.
E. Being more sensitive to being ignored, criticized or rejected.

II. The importance of a STRONG Social Support System
A. Generally, the stronger your social support system, the better you will be able to face difficult and stressful situations.
1. Social Support System = the people you are physically and/or emotionally close to and with whom you spend time and share moments of your life.
a. EX: your family, friends, neighbors, classmates, co-workers and acquaintances.

B. What is your social support network like?
Prompt a brief discussion with the participant with the following questions:
- Who are your friends?
- How often do you see them?
- What do you do?
- Who do you trust?

In-Session Exercise: Recreate your social support network using the social support worksheet *(See Appendix J).*

The participant should write their name in the center circle and write the name of someone in their network beginning with those closest to them in the innermost ring and those that are less close in the outer rings. In discussing this exercise, evaluate the quality and quantity of their network and whether it should be expanded or strengthened.

III. Guidelines regarding social support systems:
A. If your social support system is small, enlarge it.
1. Your network is too small if:
i. there is no one you trust to talk about your personal matters
ii. you have no one to go to if you need help
iii. you have no friends or acquaintances to do things with.
B. If your social support system is of a good size, appreciate it and keep it going.

1. Ways to maintain support system:
   i. Don't let disagreements cause separations between you and the people in your network.
   ii. Frequent communication helps maintain friendships.

IV. Meeting People:

Ask the following open questions, promoting a discussion.

- How do you make friends?
- What have your friends done to get closer to you?
- What does a friendly or sociable person do?

1. Doing something you enjoy doing with others is the easiest way to meet people without feeling too self-conscious.
2. When you are doing something you enjoy, you are more likely to be in a better mood and find it easier to be friendly to others.
3. Regardless of whether or not you meet someone whom you would like to get to know better, you will still have been doing something pleasant and are less likely to feel that your time was wasted on this activity.
4. There is less pressure on meeting people when the primary focus is the activity you are actually doing,
5. Because you are doing an activity you already enjoy when you meet people you would like to know better, they will already share at least one interest that brought you together.

V. Establishing and Maintaining Healthy Relationships through Assertiveness

There are three ways we can interact and communicate with others – by being passive, assertive or aggressive.

What's the difference between being passive, assertive and aggressive interaction styles?

1. Being **passive** means not expressing your feelings to others because you think they may be annoyed, feel bad or because they are superior to you. You may tend to "swallow" your feelings or feel rejected.
2. Being **aggressive** means treating others with hostility, anger and being insensitive to other people's needs and feelings because you feel yours are more important.
3. Being **assertive** means being able to say positive and negative things without feeling bad. Though you may not always say what you think, but it is important to feel that you have that you are able to. Things are said in a constructive way that can help resolve situations.

An assertive interaction and communication style will maintain the healthy relationship.

VI. Keeping Your Support System Healthy:

A. Establishing and maintaining regular contact is important: by phone or in person.

B. Suggesting activities to do with others.
1. Thoughts preventing you from reaching out to others:
a. "They will say 'no'."
b. "They are only saying 'yes' to be nice."
c. "They will go this time and find a way to avoid going out with me again."

VII. Three Focus Points to Feeling Better:

- Being alone.
- Being with others.
- Feeling good about your life.

VIII. Important Subsets of Focus Points:

A. Your thoughts
B. Your expectations
C. Your behavior
D. Your feelings

IX. Being Alone:

When you are alone, what are your _____like?

- thoughts
- actions or behavior
- feelings

X. Being With Others:

A. Your Thoughts
1. Helpful Thoughts for Feeling More Comfortable with Others:
By changing your perspective, you can change how you feel and behave.
a. Instead of: "I don't like reaching out first. What if they don't like me?"
b. Consider: "I have nothing to lose. May they we'll have a good conversation."
2. Thoughts that get in the way
a. Instead of: "Will s/he be a good friend to me?"
b. Consider: "If I am enjoying myself, others will want to join me."

B. Your Expectations
1. What can you expect of others?
2. What can others expect of you?
3. If expectations are too high, you will become disappointed, frustrated or bitter.
4. If expectations are too low:
a. you will not give yourself the chance to develop good/healthy relationships.
b. you are not giving others a chance to show you what they can bring to the relationship.

C. Your Actions

1. How do you come across?
   a. Facial Expressions:
      i. How often do you smile?
      ii. Do you make eye contact?
   b. Body Posture:
      i. Do you slouch or lean forward?
      ii. Do you look tired, worn out?
   c. Grooming:
      i. Is it appropriate for the time, place or situation?
   d. Speech:
      i. Is it too slow or too soft for others to hear?
      ii. Do you frequently raise your voice?
      iii. Do you speak with anger or irritation?
   e. Conversation Style:
      i. Do you show interest in what others say?
      ii. Do you ignore or criticize them?
      iii. Do you talk over them during conversations?
   f. Attitude:
      i. Do you complain a lot?
      ii. Are you in a bad mood?
      iii. Do you frequently offend others?

D. Your Feelings

Our emotions influence the way we relate to others. Identifying our feelings when we are with other people also helps us to evaluate the quality of our relationships.

1. Recognize how you feel and why you feel that way.
2. Communicate you feel appropriately and assertively.
3. Changing your perspective can help you be more assertive if you tend to be passive.
   a. Instead of: "It's not fair to say no to my neighbor when she asks me to babysit."
   b. Consider: "It's not fair to say yes to babysitting all the time, when I need time for myself."
4. Consistently responding appropriately when someone is unkind or disrespectful.
   a. Express your feelings, rather than bottle them up, so that you are not upset and angry later.

XI. Assertiveness Training: → Use only if participant expresses need

A. Practicing with Imagery

1. Imagine the scene as if it were a photograph
2. Imagine the action starting (as if it were a movie)
3. Imagine yourself saying something assertively
4. Imagine the response you get
5. If you like the way it came out, practice it again.
6. If you don't like it, change it and try again.

B. Learn by imitating others whose style you like.

C. Consult friends for alternative solutions or perspectives to handle a situation.

D. When you feel ready, try it out in real life.
1. See what happens.
2. Practice until you feel comfortable.

XII. Active Listening:

A. Check in with what someone said by asking them if you got it right.
1. This is done by repeating what they said in your OWN words (paraphrasing).
2. Ask them directly if how you understood them is what they meant.

B. People often argue about things without clarifying what the other person really meant to say.

XIII. Closure

When you finish the material for Session 8, discuss the following with the participant:

1) Inform him/her that you're finished with the material in the manual.
2) Briefly discuss how the participants' feelings toward the manualized intervention.
3) Explain that during the last session you will discuss your observations of the participant during the intervention and how they felt during the process.
4) State that the next meeting will be for the post-treatment assessment to objectively determine how their depression has changed or not after the intervention.
5) State that during the final session you will be offering recommendations about strategies to prevent relapses and to continue improving his/her mood.
6) Inform the participant that they will also receive a packet with the intervention diagrams and worksheet to maintain their depression on their own.

POST-TREATMENT ASSESSMENT

A. Administer the assessment measures from the Pre-Session meeting to establish the post-treatment baseline OR inform the participant they may complete online at home.

B. Optional:

1) Offer the participant information about his/her participation and progress throughout therapy.

2) Ask him/her for feedback about his/her experience in therapy. You can ask about what the participant learned thus far, liked most and least, what helped the most, etc.

3) Make a plan to manage possible relapses and discuss strategies to prevent them.

4) Offer post-treatment recommendations in terms of referral to other types of therapy or services if needed.

5) Closure and goodbye.

REFERENCES

Antoniades, J., Mazza, D., & Brijnath, B. (2014). Efficacy of depression treatments for immigrant patients: Results from a systematic review. *BMC Psychiatry, 14*, 176-188. http://www.biomedcentral.com/1471-244X/14/176

Butler, A., Chapman, J., Forman, E., & Beck, A. (2006). The empirical status of cognitive-behavioral therapy: A review of meta-analyses. *Clinical Psychology Review, 26*, 17-31. doi: 10.1016/j.cpr.2005.07.003

Cuijpers, P., Berking, M., Andersson, G., Quigley, L., Kleibeor, A., & Dobson, K. (2013). A meta-analysis of cognitive-behavrioural therapy for adult depression, alone and in comparison with other treatments. *Canadian Journal of Psychiatry, 58(7)*, 376-385.

Cully, J., & Teten, A. (2008). A Interventionist's Guide to Brief Cognitive Behavioral Therapy. Department of Veterans Affairs South Central Mental Illness Research, Education, and Clinical Center (MIRECC), Houston.

Goldberg, J. (2012, July 23). Cognitive Behavioral Therapy for Depression. Retrieved January 1, 2015, from http://www.webmd.com/depression/guide/cognitive-behavioral-therapy-for-depression

Hamm, T. (2015). 102 Things to Do on a Money-Free Weekend - The Simple Dollar. Retrieved May 15, 2015, from http://www.thesimpledollar.com/100-things-to-do-during-a-money-free-weekend/

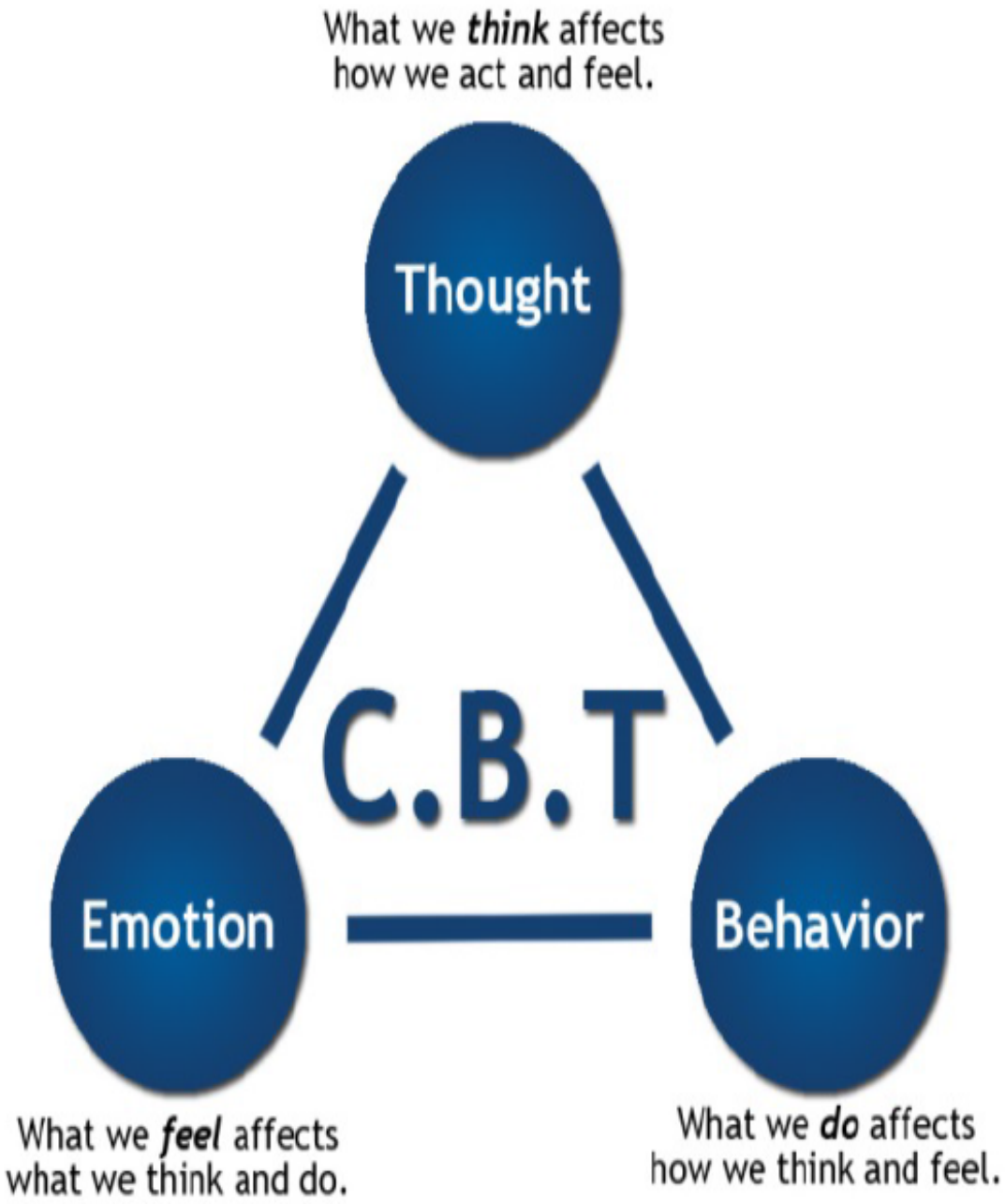
Muñoz, R. & Miranda, J. (1986, Revised 1993). Group Therapy Manual for Cognitive-behavioral Treatment of Depression. San Francisco General Hospital, Depression Clinic. Available from the author. University of California, San Francisco, Department of Psychiatry, San Francisco General Hospital, 1001 Potrero Avenue, Suite 7M, San Francisco, CA 94110.

Sørensen Høifoøt, R., Strøm, C., Kolstrup, N., Eisemann, M., & Waterloo, K. (2011). Effectiveness of cognitive behavioural therapy in primary health care: A review. Family Practice, 28(5), 489-504. doi: 10.1093/fampra/cmr017

Twomey, C., O'Reilly, G., & Byrne, M. (2014). Effectiveness of cognitive behavioral therapy for anxiety and depression in primary care: A meta-analysis. *Family Practice, 00(00)*, 1-13. doi: 10.1093/fampra/cmu060

Appendix & Participant Packet Guide

Understanding Depression and CBT



Mood Gauge

Week Number: _____

Name: _____ **Week Starting:** _____

Gauge	Mon	Tues	Wed	Thurs	Fri	Sat	Sun
My Worst	*1*	*1*	*1*	*1*	*1*	*1*	*1*
Much Worse	2	2	2	2	2	2	2
Worse than Usual	3	3	3	3	3	3	3
Typical	*4*	*4*	*4*	*4*	*4*	*4*	*4*
Better than Usual	5	5	5	5	5	5	5
Much Better	6	6	6	6	6	6	6
My Best	*7*	*7*	*7*	*7*	*7*	*7*	*7*

TYPICAL

MY BEST

MY WORST

Unhelpful Thinking Styles

When we are upset our thinking can change in unhelpful ways. Our thinking can become *distorted* or *unbalanced*. These are some of the most common unehlpful thinking styles. By recognising our unhelpful styles we can begin to change them.

All or nothing thinking

Sometimes called 'black and white thinking'

If I'm not perfect I have failed

Either I do it right or not at all

Over-generalising

*"**everything** is **always** rubbish"*

*"**nothing** good **ever** happens"*

Seeing a pattern based upon a single event, or being overly broad in the conclusions we draw

Mental filter

Only paying attention to certain types of evidence.

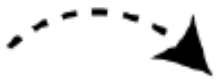
Noticing our failures but not seeing our successes

Disqualifying the positive

Discounting the good things that have happened or that you have done for some reason or another

That doesn't count

Jumping to conclusions



$2 + 2 = 5$

There are two key types of jumping to conclusions:

- **Mind reading** (imagining we know what others are thinking)
- **Fortune telling** (predicting the future)

Magnification (catastrophising) & minimisation

Blowing things out of proportion (catastrophising), or inappropriately shrinking something to make it seem less important

Emotional reasoning

Assuming that because we feel a certain way what we think must be true.

I feel embarrassed so I must be an idiot

should

must

Using critical words like 'should', 'must', or 'ought' can make us feel guilty, or like we have already failed

If we apply 'shoulds' to other people the result is often frustration

Labelling

Assigning labels to oursleves or other people

I'm a loser
I'm completely useless
They're such an idiot

Personalisation

"this is my fault"

Blaming yourself or taking responsibility for something that wasn't completely your fault. Conversely, blaming other people for something that was your fault.

Participant ID/Name: _____

Thought Chart

Week Number _____ Week Starting _____

Mark an X next to the positive thoughts you had each day & total them at the bottom.

Daily Thoughts	Mon	Tues	Wed	Thurs	Fri	Sat	Sun
I can do better							
Today is a beautiful day							
I will learn to be happy							
My life is meaningful							
I deserve to be given credit							
Though things are bad now, they'll improve							
I did a good job							
I feel really good today							
This is fun							
I chose the best solution for this situation							
I am a good person							
I am optimistic/hopeful about my future							
I have a right to be happy							
I like to read/draw							
I handled this situation well							
I get along well with others							
I worked hard, so I deserve a break							
I am considerate towards others							
I have enough time to do things I want							
I am a good person							
I am honest							
This is interesting							
I can handle a crisis as well as anyone else							
I am lucky							
I am responsible and dependable							
My experiences have prepared me for the future							
I am intelligent							
I am attractive							
I am important to my family							
I always find a way to persevere through a difficult situation							
I am likeable							
Bad things happen but I am not a bad person							
Total Positive Thoughts							

Participant ID/Name: _____

Thought Chart

Week Number _____ Week Starting _____

Mark an X next to the positive thoughts you had each day & total them at the bottom.

Daily Thoughts	Mon	Tues	Wed	Thurs	Fri	Sat	Sun
I am confused							
There is no love in the world/for me							
I am wasting my life							
I am afraid							
I will end up alone							
No one considers friendship important							
I have no patience							
Nothing is worth it							
It is too hard to go on							
I am stupid							
Anyone who thinks I'm nice doesn't know me							
Life has no meaning							
I am ugly							
I cannot do anything right							
I never express my feelings right							
I am not capable of loving							
I am worthless							
My hopes have vanished							
I am useless							
I have no luck and can never catch a break							
Everything gets ruined							
I won't be able to solve my problems							
I will never be able to change							
I won't be able to sleep							
Everything is my fault							
Nothing is fun or interesting							
I'll never stop being depressed							
Things will only get worse for me							
I am selfish							
Something is wrong with me							
I wish I were dead							
No one understands me							
Total Negative Thoughts							

WORKSHEET FOR THE ABCD METHOD

A. ACTIVATING EVENT (WHAT HAPPENED?)	C. CONSEQUENCE (HOW DID YOU REACT?)
B. BELIEF ABOUT WHAT HAPPENED (WHAT DID YOU TELL YOURSELF ABOUT WHAT HAPPENED THAT INFLUENCED YOUR REACTION?)	D. DISPUTING THE BELIEF (ALTERNATIVE THOUGHTS THAT WOULD HELP YOU IN A HEALTHIER MANNER)

Activities (Choose from 3-6 activities)	**Prediction** (How much do you think you'll enjoy these activities? - from 0 to 100%)	**Result** (How much did you actually enjoy these activities? - from 0 to 100%)	**Comments**


S
• SPECIFIC
• Define the goal as much as possible with no unclear language
• Who is involved, WHAT do I want to accomplish, WHERE will it be done, WHY am I doing this – reasons, purpose, WHICH constraints and/or requirements do I have?
M
• MEASURABLE
• Can you track the progress and measure the outcome?
• How much, how many, how will I know when my goal is accomplished?
A
• ATTAINABLE/ACHIEVABLE
• Is the goal reasonable enough to be accomplished? How so?
• Make sure the goal is not out or reach or below standard performance.
R
• RELEVANT
• Is the goal worthwhile and will it meet your needs?
• Is each goal consistent with the other goals you have established and fits with your immediate and long term plans?
T
• TIMELY
• Your objective should include a time limit. Ex: I will complete this step by month/day/year.
• It will establish a sense of urgency and prompt you to have better time management.

In-Session Practice Worksheet

SMART Goals Worksheet

(Specific, Measurable, Achievable, Realistic, Timely)

Goal Statement:

What do I need to reach this goal?

Where am I now?

Obstacles:	Solutions:

Setting Goals

MY PRIORITIES

1.

2.

3.

4.

5.

When I feel like giving up, I will tell myself…

GOAL: _____

Action Steps-

1.

2.

3.

DEADLINE: _____

GOAL: _____

Action Steps-

1.

2.

3.

DEADLINE: _____

GOAL: _____

Action Steps-

1.

2.

3.

DEADLINE: _____

GOAL: _____

Action Steps-

1.

2.

3.

DEADLINE: _____

GOAL: _____

Action Steps-

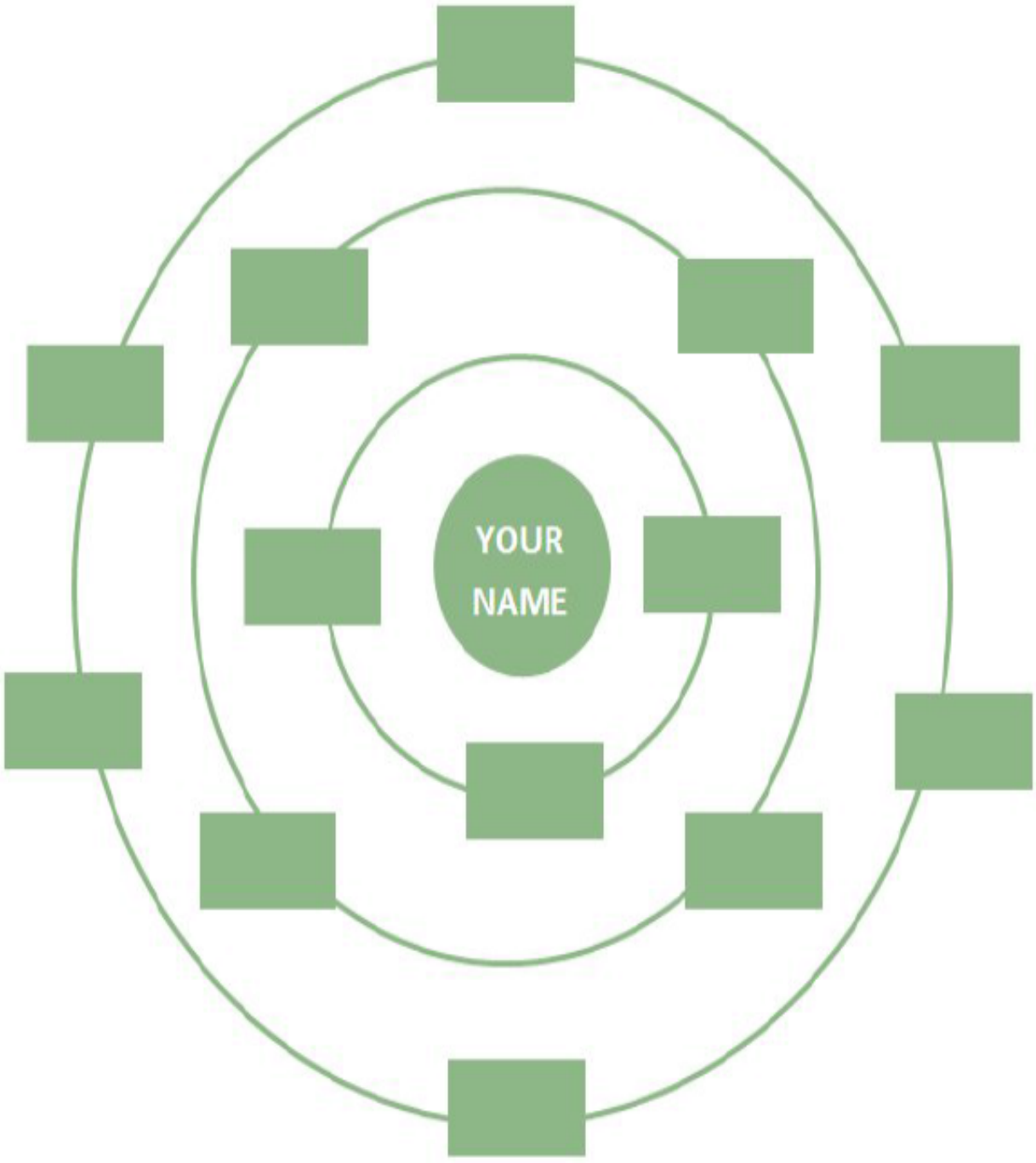
1.

2.

3.

DEADLINE: _____

SOCIAL SUPPORT IN-SESSION WORKSHEET



Fun Activities List

1. Go to the park
2. Watch the sunset
3. Pack a picnic lunch
4. Play board games
5. Play card games
6. Do a mobile scavenger hunt
7. Host a potluck party
8. Get your favorite ice cream/fro-yo
9. Make a romantic dinner
10. Cook or grill something new
11. Go to the museum
12. Go to the zoo
13. Create a scrapbook (digital)
14. Visit your city's tourist attractions
15. Organize a community cleanup
16. Go to the farmers market
17. Pick fruit
18. Bake a pie or cake
19. Go fishing
20. Go camping
21. Go to the beach
22. Go to the library
23. Host a themed movie marathon
24. Rearrange your furniture at home
25. Write a bucket list then start it
26. Put together a puzzle
27. Go on a bike ride
28. Take an art class
29. Go bowling
30. Help a friend in need
31. Plant a garden
32. Go dancing
33. Go to a sporting event
34. Go hunting
35. Get a massage
36. Host a Minute to Win It party
37. Write a letter to someone
38. Have a quiet evening
39. Go swimming
40. Have family gathering
41. Fly a kite
42. Have a discussion with friends
43. Spend an evening with good friends
44. Start or Restart a hobby
45. Go to the gym
46. Practice karate or yoga
47. Make a gift for someone
48. Go birdwatching
49. Go people watching
50. Join a club (sewing, garden, etc.)
51. Do karaoke with friends
52. Start a book club
53. Go to the salon (hair/nails)
54. Play tennis
55. Kiss or flirt with your partner
56. Play with the kids
57. Play with your animals
58. Go to a play or concert
59. Go for a day trip/drive
60. Refurbish furniture
61. Listen to music
62. Walk on the riverfront
63. Complete a goal or task
64. Write in a diary or journal
65. Meditate
66. Have lunch/coffee with a friend
67. Go horseback riding
68. Take up photography
69. Go rock climbing
70. Take a group fitness class
71. Have a BBQ
72. Go window shopping
73. Go antique shopping
74. Do crossword puzzles
75. Reflect on your improvements
76. Buy something for yourself
77. Read about a different religion
78. Surf the internet
79. Learn to knit or crochet
80. Think about your good qualities
81. Take a sauna or steam bath
82. Go skiing
83. Go skating (roller or ice)
84. Take a ballet, tap or jazz class
85. Learn a new instrument
86. Learn a new language

Deep breathing for stress relief

With its focus on full, cleansing breaths, deep breathing is a simple, yet powerful, relaxation technique. It's easy to learn, can be practiced almost anywhere, and provides a quick way to get your stress levels in check. Deep breathing is the cornerstone of many other relaxation practices, too, and can be combined with other relaxing elements such as aromatherapy and music. All you really need is a few minutes and a place to stretch out.

How to practice deep breathing

The key to deep breathing is to breathe deeply from the abdomen, getting as much fresh air as possible in your lungs. When you take deep breaths from the abdomen, rather than shallow breaths from your upper chest, you inhale more oxygen. The more oxygen you get, the less tense, short of breath, and anxious you feel. So the next time you feel stressed, take a minute to slow down and breathe deeply:

- Sit comfortably with your back straight. Put one hand on your chest and the other on your stomach.
- Breathe in through your nose. The hand on your stomach should rise. The hand on your chest should move very little.
- Exhale through your mouth, pushing out as much air as you can while contracting your abdominal muscles. The hand on your stomach should move in as you exhale, but your other hand should move very little.
- Continue to breathe in through your nose and out through your mouth. Try to inhale enough so that your lower abdomen rises and falls. Count slowly as you exhale.

If you have a hard time breathing from your abdomen while sitting up, try lying on the floor. Put a small book on your stomach, and try to breathe so that the book rises as you inhale and falls as you exhale.

Guided imagery for stress relief

Visualization, or guided imagery, is a variation on traditional meditation that can help relieve stress. When used as a relaxation technique, guided imagery involves imagining a scene in which you feel at peace, free to let go of all tension and anxiety. Choose whatever setting is most calming to you, whether a tropical beach, a favorite childhood spot, or a quiet wooded glen. You can do this visualization exercise on your own, with a therapist's help, or using an audio recording.

Close your eyes and let your worries drift away. Imagine your restful place. Picture it as vividly as you can—everything you can see, hear, smell, and feel. Guided imagery works best if you incorporate as many sensory details as possible. For example, if you are thinking about a dock on a quiet lake:

- See the sun setting over the water
- Hear the birds singing
- Smell the pine trees
- Feel the cool water on your bare feet
- Taste the fresh, clean air

Progressive muscle relaxation for stress relief

Progressive muscle relaxation is another effective and widely used strategy for stress relief. It involves a two-step process in which you systematically tense and relax different muscle groups in the body.

With regular practice, progressive muscle relaxation gives you an intimate familiarity with what tension—as well as complete relaxation—feels like in different parts of the body. This awareness helps you spot and counteract the first signs of the muscular tension that accompanies stress. And as your body relaxes, so will your mind. You can combine deep breathing with progressive muscle relaxation for an additional level of relief from stress.

Progressive Muscle Relaxation Sequence

- Right foot
- Left foot
- Right calf
- Left calf
- Right thigh
- Left thigh
- Hips and buttocks
- Stomach
- Chest
- Back
- Right arm and hand
- Left arm and hand
- Neck and shoulders
- Face

Most progressive muscle relaxation practitioners start at the feet and work their way up to the face. Also:

- Loosen your clothing, take off your shoes, and get comfortable.
- Take a few minutes to relax, breathing in and out in slow, deep breaths.
- When you're relaxed and ready to start, shift your attention to your right foot. Take a moment to focus on the way it feels.
- Slowly tense the muscles in your right foot, squeezing as tightly as you can. Hold for a count of 10.
- Relax your right foot. Focus on the tension flowing away and the way your foot feels as it becomes limp and loose.
- Stay in this relaxed state for a moment, breathing deeply and slowly.
- When you're ready, shift your attention to your left foot. Follow the same sequence of muscle tension and release.
- Move slowly up through your body — legs, abdomen, back, neck, face — contracting and relaxing the muscle groups as you go.