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The Minnesota Multiphasic Personality Inventory Pessimism–Optimism Scale as a Predictor in Depression Change After Medication Treatment and Placebo

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Walden University
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Dissertation Submitted in Partial Fulfillment

of the Requirements for the Degree of

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Abstract

The field of positive psychology has included research on the personality traits of optimism and pessimism and how they predict depression; however, there has been limited investigation on whether these characteristics can predict treatment outcomes. This study used a quantitative nonexperimental design with archived data provided by Summit Research Network, to evaluate whether pessimism or optimism, as measured by the Pessimism–Optimism (PSM) scale of the Minnesota Multiphasic Personality Inventory, had an effect on depression and somatic symptom change in 98 adults with depression who were randomized into imipramine, alprazolam, or placebo treatment. Seligman's explanatory style theory was used to guide the research. Repeated-measures mixed analyses of covariance were employed to examine treatment and explanatory style group differences using pretreatment symptoms and sex as covariates, with depression and somatic symptom scores as dependent variables. There was no significant effect for time or PSM categorization in either analysis. The somatic symptom change score significantly differed by treatment group, with the placebo group experiencing a significantly greater decrease than both drug groups. This study adds to the current literature on the role of explanatory style on treatment outcome and the influence of the placebo effect for individuals with depression and somatic symptoms. Positive social change implications include benefiting individuals with depression and healthcare providers by indicating that PSM categorization does not necessarily predict depression treatment outcome and does not need to be screened for at pretreatment.

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Dedication

To my mother: M. Jean Winsor

(November 21, 1934–February 1, 2014)

My Papa D: Carmen “Jimmy” Vetrino

(April 6, 1933–October 7, 2013)

And my brother: Clifford “Butch” Winsor

(October 11, 1956–March 26, 2011)

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As with any piece of research that results in the production of a dissertation, more names should be placed upon the cover other than the name of the researcher—the names of the unsung heroes, those who, to varying degrees, provided assistance, encouragement, and guidance, and without whom I would not have succeeded. I am very grateful to all those people, my heroes, who have given me so much of their time, love, and energy. In producing my dissertation, I faced and gained my final and greatest academic achievement, my Ph.D.

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Table of Contents

List of Tables

List of Figures

Chapter 1: Introduction to the Study

This study investigated associations between optimism–pessimism and treatment outcomes in individuals diagnosed with depression and treated with either medication or placebo. The study was based on the work of Seligman (1989), whose articulation of explanatory style theory resulted in the creation of the Optimism-Pessimism (PSM) scales of the Minnesota Multiphasic Personality Inventory (MMPI). Although several researchers have established a clear link between optimism–pessimism, and depression (Burkhart et al., 1980; Gross et al., 2000; Suzuki et al., 2014), most of the studies associating MMPI scales with psychological constructs such as depression are dated, as this field of inquiry peaked in the late 20th century with few researchers pursuing it in the past 20 years. For example, using a variety of personality inventories, including the MMPI in a study of college students, Burkhart et al. (1980) discovered that MMPI scales were highly correlated with depression. Gross et al. (2000) reviewed the literature and identified 18 studies that found a predictive correlation between MMPI scales and depression.

Recent research has reprised an interest in optimism–pessimism in the outcome of various medical issues (Murberg, 2012; Novotny et al., 2010; Singh et al., 2016). There is also some indication that optimism–pessimism plays a role in depression outcomes. In a recent study, Suzuki et al. (2014) discovered that patients with acute depression and treatment-resistant depression had high levels of pessimism and low levels of optimism, whereas patients with remitted depression and a nondepressed control group had high levels of optimism and low levels of pessimism. Despite the established association

between depression and optimism–pessimism, there is no research investigating whether or not optimism–pessimism can predict treatment outcomes in depression. This may be an important association because optimism–pessimism is a characteristic that can be manipulated with treatment such as cognitive-behavioral therapy and, therefore, could help individuals battle depression more effectively. Further, optimism–pessimism may not just play a role in predicting treatment outcomes but also in the placebo effect (Morton et al., 2009).

Depression is more than just a low mood; it is associated with a constellation of symptoms, such as somatic complaints, that is also likely associated with optimism–pessimism. Multiple researchers have linked depression with somatic complaints (Kampfhammer, 2006; Kurt et al., 2010). Kurt et al. (2010) claimed evidence for comorbidity of somatic, anxiety, and depressive symptoms, otherwise known as the "SAD" triad. Another goal of the research was to investigate the associations between optimism–pessimism scores and changes in somatic complaints during depression treatment. The somatic scale of the Symptom Checklist (the 61-item version; SCL-61, Derogatis et al., 1974) was used to assess somatic symptoms, as it is a widely used scale with good metrics (Davidson et al., 2010).

Deidentified archived treatment outcome data were used to determine whether PSM scores from the MMPI predicted depression and somatic complaint score change in depressed adults treated with medication or placebo. The study findings provide healthcare professionals with information that may lead to more effective treatment plans for patients with depression and somatic complaints. Depression is a problem for millions

of Americans (National Institute of Mental Health [NIMH], 2015), so discovering better ways to treat it presents a positive social change.

Chapter 1 begins with a description of the study's background and the gap in the existing literature that makes this study important. I also discuss the history and development of the MMPI and PSM scale and review the predictive ability of the MMPI and other instruments for depression and other conditions. The purpose of the study and the research questions are then presented, followed by the theoretical framework for the study. I also discuss the nature of the study and provide associated definitions and the study's assumptions, delimitations, limitations, and significance.

Background

Although previous research has demonstrated an association between the MMPI PSM scales and depression (Burkhart et al., 1980; Gross et al., 2000; Malinchoc et al., 1998; Peterson & Bossio, 2001; Seligman, 2000; Suzuki et al., 2014) as well as somatic complaints (Kurt et al., 2010; Murberg, 2012; Singh et al., 2016), a gap exists in the current literature regarding the association between the MMPI PSM scales and treatment outcome in depression and somatic complaints. The current study filled this gap by using archived treatment study data. There is no recent research on the predictive ability of the MMPI PSM scales with treatment outcomes regarding depression and somatic complaint symptoms. However, the PSM scale has been found to have a positive predictive ability with hip arthroplasty outcomes (Singh et al., 2016), lung cancer survival (Novotny et al., 2010), poor health and mortality (Seligman, 2000), pulmonary function (Kubzansky et al., 2002), and military aggression and risk-taking (Satterfield & Seligman, 1994).

Overall, these studies have associated an optimistic explanatory style assessed by the PSM scale with positive health outcomes. Additionally, the PSM pessimistic explanatory style has been associated with depression (Peterson & Bossio, 2001; Seligman, 2000); however, it has not been declared predictive of treatment outcome.

Suzuki et al. (2014) reported that participants with acute depression had pessimism over 3 times higher than healthy controls; they also had significantly lower levels of optimism than controls. Although depression and pessimism are strongly associated (Peterson & Bossio, 2001; Seligman, 2000), optimism–pessimism is related to health outcomes for many conditions (Peterson & Bossio, 2001; Seligman, 2000). It is unknown whether optimism–pessimism is related to depression treatment outcomes. Due to the wealth of recent positive research, there is a good reason to believe that the PSM subscales of the MMPI might be promising regarding predictive ability in treating depression.

Although Suzuki et al. (2014) identified a predictive relation between MMPI PSM scales and depression, they did not address outcomes in the treatment of depression. Furthermore, while research has been conducted on the association between placebos and optimism–pessimism (Caldwell-Andrews, 2001; Geers et al., 2005; Malani, 2006), no researchers have focused on treatment outcomes for associated symptoms such as somatic complaints. Including a focus on placebo treatment of depression and somatic complaints is a distinguishing factor that makes this study unique and addresses additional gaps in the available research.

Malani (2006) linked higher levels of optimism with positive placebo treatment outcomes for participants diagnosed with ulcers and high cholesterol. Geers et al. (2005) examined the role of optimism–pessimism as a personality characteristic in placebo effects. They provided individuals with placebos and found that pessimism was associated with reporting more negative side effects of the placebo. However, neither of these studies addressed the relation between optimism–pessimism, and outcome in a randomized, placebo-controlled treatment, which the current study did. This study adds to the literature to enable researchers to better understand the relation between outlook and outcome in treating depression in individuals treated with active medication or placebo.

Problem Statement

The study addressed the lack of information about how optimism–pessimism predicts depression treatment outcomes. Such information may help improve the treatment of depression and associated somatic complaints. Depression is a significant problem afflicting millions of individuals. The NIMH (2017) estimates that over 16 million adults in the United States, or 6.7% of the adult population, have suffered from a major depressive episode. Somatic complaints are less well-documented but tend to be comorbid with depression, as evidenced by the SAD triad (Kurt et al., 2010).

The MMPI is a reliable and valid assessment tool often used to help diagnose mental health disorders (Butcher & Willias, 2009; Camara et al., 2000). There has been exhaustive research on the MMPI and its potential predictive ability concerning treatment outcomes, including premature termination from psychotherapy (Chisholm et al., 1997), insomnia (Edinger & Means, 2005; Edinger et al., 2001), combat-related posttraumatic

stress disorder (PTSD; Forbes et al., 2003), PTSD, depression and dissociative severity in female childhood sexual abuse survivors (Johnson et al., 2001), alcoholism (Kranzler et al., 1996), chronic low back pain (Love & Peck; 1987), depression (Robinson et al., 1990), opiate addiction (Rounsaville et al., 1982), therapeutic relationship and treatment duration (Saltzman et al., 1976), gastric bypass surgery (Tsushima et al., 2004), adolescent substance abuse (Williams & Chang, 2000), and adolescent sexual offender recidivism (Worling & Curwen, 2000), among many other studies.

This study used a portion of the MMPI, the PSM scales, which focus on optimism–pessimism levels, to predict treatment outcomes of depression and somatic complaints. While the PSM scales have been used successfully to predict depression levels (Seligman, 1989, 2000; Suzuki et al., 2014), they have not been used to predict treatment outcomes. Because optimism–pessimism represents a construct that may change with targeted treatment, it is worth investigating the degree to which it may be associated with treatment outcome.

As reviewed above, interest in the MMPI as a predictive tool has waned in the past two decades. This study rejuvenates research on the predictive power of the MMPI, in the form of the PSM scales, regarding treatment outcomes in depression and somatic complaints. The research also investigated treatment outcomes in both the placebo and the pharmacologically based treatment modalities, providing an additional focus on the possible role of optimism–pessimism in the placebo response.

Purpose

The purpose of this quantitative chart review study was to use archived deidentified treatment outcome data to determine whether PSM T scores (further simplified to PSM scores; T score in statistics is the calculated difference represented in units of standard measurement) from the MMPI (independent variable) predicted depression score change, as measured by the Hamilton Psychiatric Rating Scale for Depression (HAM-D; Hamilton, 1980) and somatic complaint score change, as measured by the SCL-61 (dependent variables) in depressed adults, ages 21 to 65 years, from five metropolitan areas of the Western United States. The study investigated whether PSM scores predicted change in depression and somatic complaint measures in the treatment and placebo groups (second independent variable) of the archived treatment study data. The archived data were used in previous research by Mendels and Schless (1986), and the authors' research is referred to in the present study. In addition to the HAM-D, used to monitor depression levels of subjects at weekly intervals, Mendels and Schless (1986) utilized the SCL-61 to measure somatic symptoms.

The original research data were collected from a double-blind placebo-controlled treatment study of alprazolam, imipramine, and placebo in 98 adults with depression, in which 30 subjects were given alprazolam, 34 were given imipramine, and 34 were given the placebo. The authors reported that approximately half the participants taking alprazolam improved by over 50% on the HAM-D after 50 days. Nearly 17% of these participants improved from 25% to 50% on HAM-D scores during the same period (Mendels & Schless, 1986). Roughly 38% of the participants taking imipramine

experienced at least a 50% improvement in HAM-D scores, and about 29% experienced a 25% to 50% improvement in HAM-D scores. Approximately 18% of the placebo group experienced a (significant) 50% improvement in HAM-D scores, and nearly 15% experienced a 25% to 50% improvement.

The original research protocol included using the MMPI as an assessment; however, the MMPI data were never scored or used in the analysis, nor were the SCL-61 data (W. Smith, personal communication, March 14, 2017). In the current study, those data were accessed, and the PSM scores were calculated to determine if the PSM scale is a significant predictor of outcome. Pretreatment depression severity was accounted for as a covariate in the analysis of depression score change, and pretreatment somatic symptoms were accounted for as a covariable in the analysis of somatic symptom change to determine whether these variables correlated with the PSM scale and predicted treatment outcome (Novotny et al., 2010; Seligman, 2000; Singh et al., 2016; Suzuki et al., 2014).

Through this study, I sought to discover whether optimism–pessimism predicted treatment outcomes in both the treatment and placebo groups based on the idea that if optimism–pessimism predicted an outcome, then using the MMPI PSM scales as an assessment tool would prove helpful in improving placebo-based depression treatment. The ultimate goal of the study was to examine whether levels of optimism–pessimism improved treatment outcomes.

Research Questions

RQ1: Does optimism-pessimism level affect depression level change after controlling for baseline depression severity in a group of depressed adults treated with antidepressants or placebo?

$H_0$1: There is no significant effect of optimism-pessimism, as assessed by PSM level, on depression symptom change, as assessed by HAM-D scores, at baseline and posttreatment after controlling for baseline depression measured using the MMPI depression score.

$H_1$1: There is a significant effect of optimism-pessimism, as assessed by PSM level, on depression symptom change, as assessed by HAM-D scores, at baseline and posttreatment after controlling for baseline depression measured using the MMPI depression score.

$H_0$3: There is no significant interaction effect between antidepressant treatment and PSM level on depression symptom change, as assessed by HAM-D scores, at baseline and posttreatment after controlling for baseline depression measured using the MMPI depression score.

$H_1$3: There is a significant interaction effect between antidepressant treatment and PSM level on depression symptom change, as assessed by HAM-D scores, at baseline and posttreatment after controlling for baseline depression measured using the MMPI depression score.

RQ2: Does optimism-pessimism level affect somatic symptom change after controlling for baseline somatic symptom severity in a group of depressed adults treated with antidepressants or placebo?

$H_0$1: There is no significant effect of optimism-pessimism, as assessed by PSM level, on somatic symptom change, as assessed by SCL-61 scores, at baseline and posttreatment after controlling for baseline depression measured using the MMPI hypochondriasis score.

$H_1$1: There is a significant effect of optimism-pessimism, as assessed by PSM level, on somatic symptom change, as assessed by SCL-61 scores, at baseline and posttreatment after controlling for baseline depression measured using the MMPI hypochondriasis score.

$H_0$3: There is no significant interaction effect between antidepressant treatment and PSM level on somatic symptom change, as assessed by the SCL-61, at baseline and posttreatment after controlling for baseline somatic symptoms measured using the MMPI hypochondriasis score.

$H_1$3: There is a significant interaction effect between antidepressant treatment and PSM level on somatic symptom change, as assessed by the SCL-61, at baseline and posttreatment after controlling for baseline somatic symptoms measured using the MMPI hypochondriasis score.

Framework

The theoretical framework of the study was Seligman's explanatory style theory, the theory upon which the MMPI PSM scales are based (Gillham et al., 2001; Peterson et al., 1988; Seligman, 1989). Seligman's explanatory style theory focuses on the mechanisms behind *how* people explain both good and bad events in their lives. How individuals make attributions regarding the causes of events influences the quality of their lives, including future physical health, emotional well-being, the risk for depression, work performance, and academic achievement (Novotny et al., 2010; Seligman, 1989). For example, people with a pessimistic explanatory style may attribute the cause of bad events to themselves and good events to something outside themselves, such as luck (Singh et al., 2016). Further, the pessimistic person expects a bad condition to persist, contaminating all areas of the person's life (a global explanation) and having a ruinous effect (Novotny et al., 2010; Singh et al., 2016). This is also called *catastrophic thinking*, or "ruminating about irrational worst-case outcomes" (Breazeale, 2011, para. 1), which, according to Seligman (2000), is much easier for a pessimistic person to do than an optimistic person. Seligman's explanatory style theory combines elements of cognitive-behavioral theory (Beck, 1967; Beck et al., 1983), the learned helplessness model (Abramson et al., 1978), and locus of control theory (Rotter, 1966) to focus on how overall outlook impacts perception, behavior, and affect.

This study used Seligman's explanatory style theoretical framework to explore the relation of PSM scores to treatment outcome in a randomized, double-blind treatment study of antidepressants and placebo in 98 adults with depression and somatic

complaints. The study controlled for pretreatment depression and somatic complaint severity and examined whether PSM scores predicted treatment outcomes for individuals in both antidepressant and placebo groups. According to the theory, optimism should have been related to positive depression and somatic symptom outcomes in both the active treatment and placebo groups.

Nature of the Study

This quantitative chart review study used archived deidentified data collected from a double-blind, placebo-controlled experimental study investigating the efficacy of alprazolam and imipramine compared to placebo in a sample of depressed individuals. The study design was quantitative because it sought to determine a predictive relation between MMPI PSM score and treatment outcome of depression and somatic complaints while controlling for pretreatment depression and somatic complaint levels. Mendels and Schless (1986) previously analyzed these study data to report treatment outcomes; however, the MMPI data were not used.

The depression and somatic symptom clinical subscale scores from the MMPI were used as covariates for the first and second research questions, respectively. There were two independent variables. One independent variable was the treatment group used in Mendels and Schless's (1986) research, with three levels: alprazolam, imipramine, and placebo. The other independent variable was the PSM score, which has three levels: pessimistic, mixed, and optimistic (Maruta et al., 2002). The dependent variable was change from the baseline depression score to address the first research question. The

dependent variable was change from baseline somatic complaint score to address the second research question.

All of Mendels and Schless's research participants were diagnosed with major depressive disorder (MDD) and scored in the clinically significant range on the HAM-D. The authors stated that the HAM-D depression scale was administered to all participants weekly for 7 consecutive weeks. The SCL-61, which measures somatic complaints, was administered to all participants weekly for 7 consecutive weeks. The archived raw data from MMPIs administered in the pretreatment phase were used to calculate PSM scores.

Definitions

Depression: A treatable medical illness that can cause significant sadness and a decrease in interest, ability, or enjoyment of the usual home, work, and social activities. Symptoms include pervasive sadness or depressed mood, a significant change in appetite, a significant change in or difficulty sleeping, decreased energy or fatigue, feelings of worthlessness or guilt, difficulty concentrating or making decisions, and persistent thoughts of suicide or death. For a diagnosis of clinical depression, symptoms must last 2 weeks or longer (American Psychiatric Association [APA], 2018).

Explanatory style: A theory developed by Seligman that describes how individuals explain the causes of events (both good and bad) in their lives, as well as how this influences the quality of their lives, including physical health, emotional well-being, the risk for depression, work performance, and academic achievement (Novotny et al., 2010; Seligman, 1989).

Optimism: The tendency to explain good events as internal, stable, and global; and the tendency to explain bad events as external, brief, and specific (Malinchoc & Shulman, 1994).

Pessimism: The tendency to explain good events as external, brief, and specific; and to explain bad events as internal, stable, and global (Malinchoc & Shulman, 1994).

Placebo: "A chemically inert substance that works under its presumed psychological effect" (Kirsch, 1978, pp. 255–256).

Placebo effect: A term used to denote the "therapeutic effect of a placebo administration" (Kienle & Kiene, 2001, pp. 31–50). Also described as an effect that is attributed to a medicine or medical procedure but not to the specific properties of the medicine or medical procedure (Wolf, 1959).

Placebo responder: A general term used to denote a person who has a positive response, or positive treatment outcome, using a placebo (Vallance, 2006).

Serotonin reuptake inhibitor (SSRI): Antidepressant medication used to treat symptoms of depression by increasing serotonin—a neurotransmitter that carries signals between brain cells—in the brain (Mayo Clinic, 2018).

Somatic symptoms: Somatic symptoms are body-based complaints and problematic conditions, such as fatigue, difficulty sleeping, poor appetite, heart palpitations, and musculoskeletal pain (Kampfhammer, 2006; Tylee & Gandhi, 2005).

Treatment-resistant depression: A lack of clinically significant response to a minimum of two types of antidepressant medication, in which a response is equivalent to a 50% reduction of depressive symptoms (Suzuki et al., 2014).

Assumptions

For this study, I assumed that the archived data on depression, somatic complaints, optimism–pessimism scales, and treatment outcomes used in the study by Mendels and Schless (1986) were valid and accurate. It also assumed that the HAM-D accurately measures depression and the SCL-61 measures somatic complaints. I assumed that the participants in the study by Mendels and Schless participated and answered the scales honestly and to the best of their ability. Overall, this study assumed that the previous study was reliable and valid. These assumptions were necessary because this study used archived data from a previous study to test its hypotheses.

Scope and Delimitations

The scope of the research problem is centered around how optimism–pessimism scores are associated with outcomes in the treatment of depression using antidepressants or placebo pills. The study focused on depression and somatic complaints because these were the symptoms treated in the archived study by Mendels and Schless (1986). The study participants included men and women between the ages of 18 and 60 years living in Oregon and Washington state. Specifically, the sample comprised 98 adults, including 53 men and 45 women. All of the participants had been diagnosed with MDD for at least 1 month.

The study excluded pregnant women and individuals with significant liver, kidney, gastrointestinal, cardiovascular, or pulmonary disease. Individuals with allergies to benzodiazepines or imipramine or who were addicted to alcohol or other drugs were omitted. Individuals taking a psychotropic drug, a potent analgesic, or an antihistamine;

who had taken another investigational drug within the past month; or who had taken other antidepressants, major tranquilizers, or benzodiazepines within the last 7 days were excluded from participation. Therefore, the study results were expected to be generalizable to adults with MDD living in the Northwest United States who met the study's inclusion criteria and fit the sample demographics but may not be generalizable to individuals who do not fit the sample description.

For this study to be relevant on a grand scale, it needed to be generalizable. The generalizability of any study results requires the researcher to extract the relevant facts from the study and arrive at reasonable conclusions regarding those relevant facts (Kukull & Ganguli, 2012). The relevant facts about this study regarded the relationships between the dependent and independent variables. If optimism–pessimism, measured by PSM scores, predicts treatment outcome, future research may investigate generalizability to other populations.

Archival data allowed for generalizability. The medications used in the Mendels and Schless (1986) study are still used today. The data were collected by a team that documented the adherence to the treatment protocol; therefore, the data were valid. Lastly, the data were collected by a team of professionals who could collect an amount and quality of information that would be impossible to collect prospectively on a smaller scale.

Internal validity was secured through reliable methods of diagnosing depression and somatic complaints in the study sample and monitoring depression and somatic symptom levels with valid and reliable measures. In the study by Mendels and Schless

(1986), depression was measured at weekly intervals using the HAM-D, and somatic complaints were measured using the SCL-61, both of which have demonstrated strong validity and reliability. The MMPI is also a well-established measure that has high validity and reliability.

Limitations

One of the main limitations of this study was utilizing data and results from a previous study; therefore, no prospective data could be collected. This was a significant limitation because the current study relied on the validity of the previous study to obtain valid and applicable results. Another limitation was that the treatment outcomes of depression and somatic complaints were obtained using specific measures: the HAM-D and the SCL-61. There are several measures for these symptoms that may differ. However, as previously stated, the HAM-D and SCL-61 have demonstrated strong reliability and validity.

One of the reasonable measures to address the study's limitations was exercising great diligence in examining the methods and results of the study by Mendels and Schless (1986). One of the ways that this was accomplished was by meeting with one of the previous study authors to discuss the previous study and become more familiar with its context and contents.

Significance

This study investigated whether a measure of optimism–pessimism, as assessed by the MMPI PSM scale, significantly predicted treatment outcomes as reflected by depression and somatic symptom scores in the archived data of a study of placebo-

controlled drug treatment in individuals with depression. The research focused on treatment outcome prediction, including the prediction of the placebo effect, which may help individuals and their families by aiding in understanding how optimism–pessimism impacts treatment and outcomes in depression and somatic complaints. This research may inspire approaches that could augment medical and psychological treatments, benefitting patients and health care professionals seeking effective treatment options.

The findings were significant because they showed whether optimism–pessimism impacts treatment outcomes of depression and somatic complaints. Such information may be useful in seeking treatment for depression. Families of individuals receiving treatment for these conditions will be better informed about how to support their treatment. Health care providers will have a more comprehensive and dynamic understanding of treating depression and somatic complaints. On the societal level, a better understanding of the association between optimism–pessimism and depression and somatic complaints, and placebo treatment of these conditions impacts how people generally think about these problems, perhaps causing a greater acceptance of and ability to resolve them.

Summary

Based on previous research into the predictive ability of the MMPI and PSM scales, I sought in this study to determine the predictive ability of the MMPI PSM scales in a placebo-controlled treatment of depression. The purpose of the study was presented in this chapter, as were the research questions, variables, and hypotheses. The theoretical framework was presented as Seligman's explanatory style theory, which provides a basis for understanding how or why optimism–pessimism, as measured by the PSM, could

predict the treatment outcome of depression. This chapter has presented the basis and rationale for the study.

Chapter 2 presents a more in-depth look at the development of the MMPI PSM scales and their predictive abilities with treatment outcomes of depression, somatic complaints, and other symptoms and conditions. Chapter 2 also explores the SAD triangle, which represents evidence for the comorbidity of somatic, anxiety, and depressive symptoms. It discusses the use of placebo in treatment studies of depression and somatic complaints and delves more deeply into how PSM scales have been used with placebo treatment.

Chapter 2: Literature Review

Researchers have found that pessimism, as assessed by the PSM, is positively correlated with depression symptoms (Burkhart et al., 1980; Gross et al., 2000; Suzuki et al., 2014). Pessimism is also associated with increased somatic complaints (Murberg, 2012; Singh et al., 2016). Significant positive correlations between depression and somatic complaints have also been identified (Kampfhammer, 2006; Kurt et al., 2010). However, no research exists investigating the possible relation between optimism–pessimism and outcomes of depression and somatic complaints in a placebo-controlled treatment of depression. An investigation on whether and how symptoms change with optimism–pessimism would help support the previous research establishing associations among these variables.

This investigation invigorates research regarding the predictive ability of the MMPI in treatment outcome studies, between the PSM scales and depression as well as somatic complaints, in a placebo-controlled study of antidepressant therapy. There is currently no published research on this topic, and an investigation of the prediction of outcome in treating depression and somatic complaints represents a valuable contribution to the literature. While research on the predictive ability of the MMPI dates back as far as 1960 (Affleck & Garfield), research using the PSM scales of the MMPI began with Seligman (1989, 2000), on whose theory of explanatory style the PSM scales were based. Seligman's (1989) development of the PSM scales marked the initial use of a personality inventory to examine how optimism–pessimism relates to various mental and physical health outcomes. Since that time, the PSM has been used to study the relation of

optimism–pessimism to physical health conditions such as hip arthroplasty outcomes (Singh et al., 2016), lung cancer survival outcomes (Novotny et al., 2010), pulmonary function (Kubzansky et al., 2002), health and mortality (Seligman, 2000), and depression (Suzuki et al., 2014). However, no research has investigated the association between PSM and somatic complaints or depression treatment outcomes before the current study.

The information in this chapter addresses the strategy used to conduct the literature search and the study's theoretical foundation and conceptual framework. I elaborate on Seligman's development of explanatory style theory as well as the development of the MMPI to provide insight into personality structure and dynamics. Then, a literature review on this topic is presented, highlighting the predictive ability of the MMPI in depression and other health conditions and outcomes. In this chapter, I also thoroughly describe the PSM scale and its use in previous research. The relationship between depression and somatic complaints is explored, including the theory of the SAD triad. The use of placebos and the relation between optimism–pessimism, and placebo treatment are explored.

Literature Search Strategy

The literature search was conducted via ProQuest, an online database with an abundance of peer-reviewed academic studies, where searches were conducted under the headings of psychology, sociology, health, medicine, and psychiatry. The key search items used were *MMPI*, *MMPI-2*, *PSM*, *PSM scales*, *explanatory style*, *optimism-pessimism*, *depression*, *somatic complaints*, *placebo*, *placebo effect*, *placebo responder*, and combinations of these terms. The years searched were initially 2011 to 2017 but were

expanded to include studies conducted as far back as 1960, as the initial search revealed a profound lack of studies on the MMPI in recent years.

Theoretical Foundation

The leading theory upon which the study was founded was explanatory style theory. Explanatory style theory was developed by Seligman (1989) to describe how people explain or make sense of events and experiences in their lives. A pessimistic explanatory style is marked by attributing the causes of bad events and experiences as internal, personal, stable, and global and the causes of good events as external, transient, and specific (Malinchoc et al., 1998). A pessimistic explanatory style leads to hopelessness because individuals perceive that they are powerless to change their current condition or state. In contrast, an optimistic explanatory style is marked by attributing the causes of bad events and experiences as external, transient, and specific and attributing good events as internal, stable, and global (Malinchoc et al., 1998). In other words, individuals with an optimistic explanatory style will feel that they are more or less in control of their lives and will interpret unpleasant events as fleeting and changeable, whereas individuals with a pessimistic explanatory style feel that their unpleasant life situation is permanent and unchangeable.

Seligman's (1989) explanatory style theory led to the development of the PSM scales to measure optimism–pessimism as a personality feature. The PSM scores the person on a range of pessimism to optimism, where lower scores reflect a more optimistic explanatory style and higher scores reflect a more pessimistic explanatory style (Kubzansky, 2002; Novotny et al., 2010). Based on multiple studies associating

pessimism and learned hopelessness with negative outcomes (i.e., Kamen-Siegel et al., 1991; Segerstrom et al., 1996), Seligman (2000) concluded:

> (1) Pessimists are passive and have more bad life events than optimists. More bad life events are associated with shorter lives. (2) Pessimists, believing that "nothing I do matters," comply less well with medical regimens and take fewer preventive actions, like giving up smoking. (3) Pessimists become depressed at a markedly higher rate than optimists do, and depression is associated with mortality. (4) The immune system of pessimists functions less adequately than that of optimists. (pp. 133–134)

Seligman's (1989) explanatory style theory and subsequent development of the PSM scales provided a solid theoretical foundation upon which to test the hypotheses of this study: that pessimism is associated with worse treatment outcomes, and that optimism is associated with better treatment outcomes, regardless of whether the individual is in alprazolam, imipramine, or placebo treatment. In particular, the study applied Seligman's explanatory style theory to the outcome of a placebo-controlled treatment of depression.

Seligman's explanatory style has been used to assess the association between optimism–pessimism and several physical and mental health diagnoses (Kubzansky et al., 2002; Novotny et al., 2010; Seligman, 2000; Singh et al., 2016; Suzuki et al., 2014). Explanatory style has also been used to examine the association between optimism–pessimism and quality of life in breast cancer patients (Petersen, 2008), quality of life in heart transplant patients (Jowsey, 2012), and overall physical and mental functioning

(Maruta, 2002), and aggression and risk-taking behaviors in the military (Satterfield & Seligman, 1994).

Seligman's explanatory style theory was the optimal theoretical foundation for the study because it forms the basis of the PSM scales used to test the research hypotheses. Explanatory style describes how optimism–pessimism impacts how people view events in their lives, impacting how people live their lives (Seligman 1989, 2000). The study research questions built upon explanatory style theory by testing its applicability in predicting treatment outcomes in depression.

The PSM scale is a subscale of the MMPI (Buchanan, 1994), which contains many scales measuring various personality aspects. The MMPI was developed before and during WWII as a shared way for psychologists and psychiatrists to identify and diagnose psychological disturbances in patients (Buchanan, 1994). Initially, the MMPI "represented the operationalization of medical hegemony" (Buchanan, 1994, p. 148). The MMPI was based on earlier psychological tests that provided standardized inventories to measure psychological traits, focusing on noting psychopathologies (Buchanan, 1994). However, these early personality tests were "ineffective for treatment evaluation and had limited applicability to the low-income, poorly-educated patients often found in psychiatric institutions" (Buchanan, 1994, p. 149). They also did not appeal to traditional psychiatrists and psychodynamic psychologists, who wanted physiologic-based diagnoses (Buchanan, 1994).

The creators of the MMPI, Starke R. Hathaway, and J.C. McKinley, wanted a diagnostic tool that could condense the traditional lengthy psychiatric interview into a

quicker and structured process (Buchanan, 1994). Like older tests, the MMPI also sought to "standardize psychiatric diagnosis" (Buchanan, 1994, p. 151). Scales were initially developed to test for pathologies such as depression and schizophrenia, but the test was expanded to address a wide range of mental health symptoms, even characteristics such as introversion, and apply to a wide range of individuals (Buchanan, 1994). Validity indices were incorporated into the scales that enabled the MMPI to assess the "comprehension, compliance, and general 'test-taking attitude' of the respondent" (Buchanan, 1994, p. 152).

The initial development of the MMPI occurred in the early 1940s when psychology was still relatively new, and psychologists were only beginning to be recognized as health care professionals. At the time, psychologists considered the MMPI a mental health test that could increase their legitimacy and expertise as professionals (Buchanan, 1994). The MMPI "was based on an extension of psychologists' claims to the scientific measurement of intellectual processes" (Buchanan, 1994, p. 155). However, the emphasis on diagnosis and the disease model in the MMPI, as well as its use to classify persons as mentally ill or psychopathological, changed over the next several years into a way to measure psychological character types that would be open to interpretation by attending psychologists (Buchanan, 1994).

Peterson et al. (1983) implemented the Content Analysis of Verbatim Explanation (CAVE) technique with specific questions within the MMPI and developed the PSM scale (Colligan et al., 1994). Mental health clinicians have used the PSM scale to identify patients with a pessimistic personality style (Colligan et al., 1994) that is associated with

decreased physical health (Peterson et al., 1988) and increased propensity for depression (Seligman et al., 1979). Conversely, optimists experience more positive life events (Seligman, 1991) and have better functioning immune systems than pessimists (Segerstrom et al., 1996).

Peterson et al. (1988) and Maruta et al. (2000) used the PSM to relate pessimism to treatment and life outcomes. According to Seligman (2000), pessimism is related to feelings of helplessness related to depression symptoms and poor health. Maruta et al. (2000) reported that pessimism is associated with higher mortality rates and premature death. Peterson et al. (1988) examined the relation of pessimism to general health and concluded in a 35-year longitudinal study that a pessimistic attributional style was associated with poor health at ages 45 to 60 years, controlling for health status at the onset of the study. Maruta et al. (2000) tested the relation of the PSM scale to mortality in 839 patients. Thirty years after completing the MMPI, the researchers found that explanatory style was related to mortality. For each 10-point increase in PSM score at baseline, there was a 19% increase in mortality risk at the 30-year follow-up.

Seligman's explanatory style is the essential theory behind the MMPI's PSM scales; it was used to develop hypotheses to predict treatment outcomes in relation to optimism–pessimism in regard to both the active treatment condition and the placebo condition for the research study. The PSM has also been used to study the relation between optimism–pessimism and hip arthroplasty outcomes (Singh et al., 2016), lung cancer survival outcomes (Novotny et al., 2010), pulmonary function (Kubzansky et al., 2002), health and mortality (Seligman, 2000), and somatic complaints (Murberg, 2012).

Seligman's explanatory style theory was suitable for my study because it provided a reliable and established theoretical lens to understand the association between optimism–pessimism and the outcome of a placebo-controlled medication treatment for depression, including somatic complaints.

Literature Review

Predictive Ability of the Minnesota Multiphasic Personality Inventory in General

Because the PSM scale is a subscale of the MMPI, it is necessary to give some background on the academic literature and history of studies regarding the MMPI. The bulk of research into the predictive ability of the MMPI was conducted around the turn of the 20th century, and after that time, the trend died down with little research on the topic. A review of the literature shows mixed results on the ability of the MMPI to predict treatment outcomes (Blanchard et al., 2003; Chisholm et al., 1997; Edinger & Means, 2005; Edinger et al., 2001; Forbes et al., 2003; Johnson et al., 2001; Kent et al., 2000; Martz et al., 2005; Novotny et al., 2010; Singh et al., 2016; Suzuki et al., 2014; Weis et al., 2004).

The MMPI has demonstrated predictive ability and validity. The Infrequency (F) scale of the MMPI contains "items rarely endorsed in the keyed direction by members of the MMPI's original normative sample" and is used to detect false answering on the MMPI in the form of overreporting symptoms or "faking bad" (Fb; Blanchard et al., 2003, p. 199). The Infrequency-Psychopathology (Fp) scale elaborates on the F scale that features items that healthy individuals rarely endorse. The Fp scale is "similar in format to F and Fb but is comprised of items rarely endorsed in the keyed direction by patients

as well as [by healthy individuals]" (Blanchard et al., 2003, p. 199). The F–K index is arrived at by subtracting the Correction (K) scale, which measures underreporting of symptoms, from the F scale score and is used as an indicator of accurate symptom reporting.

Blanchard et al. (2003) found an association between the MMPI F–K index and Fp scale and faking bad in a study of 52 college students who were instructed to fake bad (overreporting or exaggerating of pathological symptoms), as well as in 432 psychiatric patients. The authors demonstrated that the F–K index identified the overreported pathological symptoms group. In addition, Martz et al. (2005) reported that elevated MMPI-2 depression scales (including the D, PK, and PS scales) predicted a more likely diagnosis of PTSD in veterans with disabilities. Along with many others, these studies have demonstrated the strong predictive ability of the MMPI with a long list of symptoms and behaviors. Given this, the continued or revived use of the instrument makes sense for future research, especially when newer scales such as the PSM are developed.

Tsushima et al. (2004) examined the ability of MMPI-2 scales to predict treatment outcomes in morbidly obese patients post-Roux-en-Y gastric bypass (RYGBP) surgery. According to Tsushima et al., the Hysteria scale "measures reactivity to stress and potential for psychophysiological symptoms;" the Masculinity–Femininity scale "measures deviation from stereotypical gender interests and attitudes;" the Paranoia scale "measures emotional sensitivity and responsiveness to others' opinions;" and the Health Concerns scale "measures somatic complaining and worry about health" (p. 531). The authors found that RYGBP surgery subjects with higher scores on the MMPI Hysteria,

Paranoia, F (faking bad), and Health Concerns scales and subjects with lower scores on the Masculinity–Femininity scale lost less weight. Tsushima et al. thus concluded that these scales predicted treatment outcomes for obese patients engaged in RYGBP surgery.

Weis et al. (2004) found that the infrequency scale, the Hypochondriasis scale, and the Hysteria scale of the adolescent version of the MMPI predicted success at a military-style residential program in a study of 225 adolescents with histories of antisocial behavior. Higher scores on all scales were associated with higher withdrawal rates of participants in the program (Weis et al., 2004).

Although several researchers have successfully linked treatment outcomes to various scales of the MMPI, others have not identified an association or have found mixed results, especially regarding psychological treatments and constructs. Chisholm et al. (1997) examined the power of the MMPI to predict premature therapy termination in a sample of 86 adult clients receiving psychotherapy at a university clinic. Three MMPI-2 clinical scales (depression, psychopathic deviate, and anxiety) and four content scales (depression, antisocial practices, anxiety, and negative treatment indicators) were poor predictors of premature termination. The authors hypothesized that participants scoring higher on these seven scales would evidence higher levels of premature termination and worse levels of the psychotherapeutic outcome. However, participants with higher scores on the anxiety scale evidenced increased progress toward psychotherapeutic goals (Chisholm et al., 1997).

Forbes et al. (2003) examined the ability of various MMPI scales to predict combat-related PTSD in 141 Vietnam veterans. Higher scores on the social alienation and

marital distress scales were more likely to demonstrate PTSD avoidance symptomatology. Higher scores on the anger scale were related to increased alcohol use, and higher scores on the hypomania scale were associated with higher levels of hyperarousal. However, no MMPI-2 scales "predicted change in re-experiencing symptoms after accounting for initial symptom severity" (Forbes et al., p. 185). Kent et al. (2000) examined the ability of six MMPI scales to predict symptom change in 224 acute psychiatric patients during a 3-week treatment program. They found that higher pretreatment scores on the social introversion subscale predicted greater distress levels at discharge. None of the other five scales used in that study significantly predicted treatment outcomes.

Overall, various MMPI scales have demonstrated practical value and reliable predictive power in some areas but lack predictive ability in others. The use of the MMPI in research decreased after 2000 (Franklin, 2009). Despite the history of mixed results, it is worthwhile to revive research using the MMPI scales to investigate the use of this measure in predicting treatment outcomes. The development of the PSM scales is a promising opportunity.

Predictive Ability of the Minnesota Multiphasic Personality Inventory With Depression Symptoms

Although researchers have not been able to use the MMPI subscales to predict psychosis symptoms (Affleck & Garfield, 1960), suicide (Clopton & Jones, 1975), or work performance behaviors (Knatz et al., 1992), they have found an association between MMPI scores and help-seeking behaviors (Davis & Widseth, 1977), depression (Burkhart

et al., 1980; Gross et al., 2000; Suzuki et al., 2014), successful rehabilitation of prison inmates (Edwards, 1963), adolescent success at a military-style residential program (Weis et al., 2004), self-reported health status (Maruta et al., 2000), problem behaviors in police officers (Tarescavage et al., 2015), and nonepileptic seizures in individuals admitted to an EEG video monitoring unit (Yamout et al., 2017). The MMPI has demonstrated the utility and predictive power in some constructs but lacks predictive ability in others.

Burkhart et al. (1980) examined and compared the ability of the MMPI and other personality inventories to predict depression symptoms. The authors recruited 209 college students who were enrolled in a psychology course and asked them to complete four questionnaires, including the MMPI, the Profile of Mood States (POMS; developed by McNair et al., 1971), the Beck Depression Inventory (BDI; developed by Beck, 1967), and a shortened version of the Pleasant Events Schedule (PES; developed by MacPhillamy & Lewinsohn, 1974). Results showed that MMPI depression scales were highly predictive of depressive symptoms in the POMS, BDI, and PES, indicating that the MMPI depression scales are valid.

Suzuki et al. (2014) gave the MMPI to 25 remitted depressed patients, 21 acutely depressed patients, 34 antidepressant treatment-resistant depressed patients, and 64 healthy controls to examine levels of optimism–pessimism (using the PSM subscale) as well as hysteria and schizophrenia with depression condition. The healthy controls and patients with remitted depression had low levels of pessimism and high levels of optimism. In contrast, patients with acute depression and treatment-resistant depression had high pessimism and low levels of optimism (Suzuki et al.). The authors reported that

this finding was common in the literature and that, in general, “high depression scores [on the MMPI] are found to be modestly accurate in predicting depression” (p. 1).

Gross et al. (2000) examined 18 studies on the ability of the MMPI to predict depressive symptoms. The authors reported that the depression scale was “moderately useful in diagnosing depression in settings where the base rate of depression is high” (p. 473). Dahlstrom et al. (1972) found that the depression scale identified and measured clinical depression (Gross et al.). Hathaway and McKinley (1980) reported that the depression scale has very high validity: “in a sample of 50 depressed patients who met the criteria for manic-depressive psychosis … Scale *2* (D) [depression scale] had a classification accuracy of .97” (Gross et al., p. 464). In their review, Gross et al. concluded that the MMPI was useful “in differentiating depression from other mental disorders” (p. 473) and that the depression scale, in particular, is “moderately accurate in predicting depression” (p. 464).

Indeed, predicting who may benefit from a specific treatment for depression, either psychological or pharmaceutical, is worthwhile. According to multiple studies (see, e.g., Cipriani et al., 2005; Cipriani et al., 2009; Rost et al., 2002; Rush et al., 2008), antidepressant treatment efficacy is overall low; only about 11% to 30% of patients with depression experience a significant remission of symptoms after an 8 to 12-month period of treatment. The absence of predictive tools that might inform treatment choice in clinical psychiatry stands in contrast with other fields of medicine, such as critical care and cardiology, which have effective predictive models that inform treatment decision-making (Chekroud et al., 2016).

In response to this deficit, Chekroud et al. (2016) built a statistical model that can "enable prospective identification of patients who are likely to respond to a specific antidepressant" (p. 243). Using patient-reported data from 1,949 patients with depression, the study found that depression severity, fatigue, and restlessness were all associated with negative outcomes; however, no personality variables were examined as potential predictors of outcome, and the MMPI was not used (Chekroud et al.).

To date, various studies have had moderate success in demonstrating the MMPI's predictive ability in identifying depression and somatic symptoms; however, none have successfully predicted treatment outcomes for either. In other words, the MMPI has shown promise in diagnosing mental health conditions (Burkhart et al., 1980; Gross et al., 2000; Suzuki et al., 2014). However, the goal of the current study was to investigate the MMPI's predictive abilities between optimism–pessimism, as measured by the MMPI PSM scales, and treatment outcomes of depression and somatic complaints. Therefore, this research represents new findings regarding the predictive capacity of the MMPI.

Pessimism–Optimism Scale

Much of the research on the MMPI's predictive ability was conducted decades ago, to the point of exhausting the potential research that could be done with this assessment tool; however, the PSM scale has been used recently to investigate treatment outcomes for multiple medical diagnoses (Jowsey et al., 2012; Novotny et al., 2010; Seligman, 2010; Singh et al., 2016) as well as levels of depression (Suzuki et al., 2014), and shows promise in predicting treatment outcome for depression.

The development of the PSM scales was based on research in the 1980s by Peterson et al. (1988) and Seligman (1989), which showed that a "pessimistic explanatory style predicted stressful life events, poor health habits, and decreased feelings of self-efficacy" (Colligan et al., 1994, p. 76). The PSM scale, which assesses explanatory style from pessimistic to optimistic using 298 MMPI items, was added as an MMPI subscale due to this research (Malichoc et al., 1995).

According to Malinchoc et al. (1998), "people with a pessimistic explanatory style are more prone to depression, lower achievement, and greater health problems than people who have a more balanced view of life events" (p. 169). Individuals with a more optimistic explanatory style have been found to experience more positive health outcomes, including lower blood pressure, better recovery from surgery, and longer survival after cancer treatment (Novotny et al., 2010).

Novotny et al. (2010) found that higher levels of pessimism on the PSM scales predicted worse lung cancer survival outcomes in a study of 534 adults who had taken the MMPI at least 18 years before their diagnosis of lung cancer. Singh et al. (2016) reported that patients who rated themselves as having a more pessimistic explanatory style on the PSM scales experienced poorer hip arthroplasty treatment outcomes, including higher pain and activity limitation levels in a study of 507 patients who had undergone that surgery.

Jowsey et al. (2012) recruited heart transplant patients who had completed the MMPI before surgery, then asked them to complete the Health Status Questionnaire (HSQ) 4 years after their operations. They found that high presurgery pessimism scores

on the PSM scale predicted more depressive symptoms on HSQ. In contrast, high optimism scores predicted relatively low depressive symptoms and higher quality of life scores on the HSQ (Jowsey et al., 2012).

The above research demonstrates a strong association between explanatory style measured by the PSM scales and treatment outcomes in several medical conditions. In general, treatment outcomes are poorer for patients with more pessimistic explanatory styles and better for patients with more optimistic ones. Such research provides a solid foundation for testing the role of optimism–pessimism in the placebo-controlled treatment of depression and somatic complaints.

Depression and Somatic Complaints

According to the Anxiety and Depression Association of America (ADAA, 2016a),

> depression is the leading cause of disability in people ages 15 to 44. It has a debilitating impact on our society. MDD can be devastating and lead to complications including alcohol or drug abuse, chronic aches and pains, significant difficulty functioning at school or work, disruptions in relationships, feelings of social isolation, eating disorders, self-mutilation, and suicidality (Pietrangelo, 2015). Although somatic complaints are often a part of the symptom profile of depression, depression is a heterogeneous phenomenon with up to 1,000 different symptom combinations meeting DSM criteria for the diagnosis of MDD (Fried & Nesse, 2015). Somatic symptoms associated with depression have been strongly linked to negative long-term outcomes in cardiovascular disease (de Miranda Azevedo et al., 2014) and inflammation (Duivis et al., 2013).

Somatic issues are highly related to depression (Tylee & Gandhi, 2005), and both physical pain and chronic pain have been found to commonly occur with depression (Bair et al., 2003). According to Kapfhammer (2006), somatic symptoms are highly associated with depression and demonstrate a "high positive predictive value (PPV) for depression" (p. 230). The following symptoms have the correlated PPVs for depression: (a) fatigue - 60%, (b) problems sleeping - 61%, (c) musculoskeletal complaints - 43%, (d) three or more somatic complaints - 56%, and (e) back pain - 39% (Kampfhammer).

Kapfhammer (2006) concurs that the most prominent presenting problem for individuals diagnosed with depression in primary care is somatic complaints. Simon et al. (1999) reported that 69% of their sample who came to a doctor's appointment complaining of somatic symptoms were diagnosed with major depression (Tylee & Gandhi, 2005). In the study by Simon et al., at least 73% of the sample diagnosed with depression also had significant somatic symptoms (Tylee & Gandhi).

The patient health questionnaire (PHQ-15; Kroenke et al., 2010) is a validated scale that measures 15 somatic symptoms, ranging from various physical aches and pains to dizziness, fainting spells, diarrhea, and trouble sleeping. Kroenke et al. discovered evidence for the comorbidity of somatic, anxiety, and depressive symptoms known as the "SAD" triad in a study utilizing the PHQ-9 (Kroenke & Spitzer, 2002; which measures depression), the Generalized Anxiety Disorder-7 measure (Spitzer et al., 2006), and the PHQ-15. Kroenke et al. identified a significant overlap between depressive, anxiety, and somatic symptoms. The relation between somatic and psychological symptoms was especially strong in individuals with high levels of symptomatology. This study, as well

as the work of other researchers (see, e.g., Kroenke & Rosmalen, 2006; Kroenke, 2003), demonstrates that somatic symptoms rarely exist in isolation; rather, depressive, anxiety, and somatic symptoms tend to be comorbid and overlap experientially (Kroenke et al.).

Somatic complaints and pessimism are related to depressive symptoms; however, the connection between somatic complaints and optimism or pessimism is unknown. Given the research reviewed above, it can be assumed that a relation exists between optimism–pessimism, and somatic symptoms; however, few studies have directly examined this potential relation. Murberg (2012) found that optimism was significantly negatively correlated with somatic symptoms in a group of healthy high school students. However, little is known about how optimism–pessimism relates to somatic symptoms in depressed individuals. Given that optimism–pessimism may be a characteristic that can be manipulated with treatment, investigating its possible association with somatic symptoms may be an important step in developing treatment options tailored for specific depression symptom profiles.

The comorbidity of depression and somatic complaints may also have implications for an overlap in treatment strategies for the two. However, just as the association between somatic complaints and optimism–pessimism is unclear, so is the association between optimism–pessimism and treatment outcomes of depression and somatic complaints. However, considering the comorbidity of somatic complaints and depression and the positive correlations between optimism–pessimism, and depression, there may also be an association between optimism–pessimism and somatic complaints. This is a gap in the literature that the current study addressed.

Placebo Response Research

The PSM was used in this study as an independent variable to determine if it could predict outcomes in both active and placebo groups in a set of archived data. It has been noted that "placebo effects cause more optimistic patients to respond better to treatment than less optimistic patients" (Malani, 2006, p. 236). Therefore, there is an implied association between explanatory style and placebo response. There is also reason to expect depressed patients who score higher on optimism might have more positive responses to both medication and placebo medication.

Geers et al. (2005) reviewed the research on placebo effects. They found that "the magnitude of the placebo effect differs widely depending upon the expectations of the participants and the meaning that they ascribe to the situation" (p. 121). Geers et al. administered the Revised Life Orientation Test (LOT-R; Scheier et al., 1994) to assess for optimism–pessimism. They reported that pessimists in the deceptive expectation group (they were told they would have an unpleasant side effect) experienced significantly more negative placebo effects than optimists in the same group (Geers et al.,). There were no differences between the pessimist and optimist groups in the conditional expectation group. Participants were told they could have an unpleasant side effect or no effect. A third group was given a placebo pill that they were told would have no effect on them. The findings demonstrate a possible link between optimism–pessimism and the placebo effect, indicating that pessimists may have a stronger propensity than optimists to experience a negative placebo effect when told to expect a negative response.

The study by Geers et al. (2005) harkens back to Seligman's (1989, 2000) explanatory style theory and early research on the effects of having a pessimistic attitude, in which pessimism was positively correlated with morbidity and mortality. Several studies showed associations between pessimism and poor life and treatment outcomes (Novotny et al., 2010; Singh, 2016). However, none of these studies used a placebo group. The current study included aspects of the research conducted by Geers et al. (2005), Novotny et al. (2010), Seligman (1989, 2000), and Singh (2016) and provided additional information regarding placebo treatment effects in correlation with optimism–pessimism.

Placebo Effect

Researchers have argued that placebo effects are partially responsible for treatment outcomes in virtually every area of medicine (Harrington, 1997; Jospe, 1978; Kirsch & Moerman, 2002; Peters, 2001; Shapiro & Shapiro, 1997; White et al., 1985). The impact of the placebo effect and statistical artifacts such as regression to the mean is planned for and mitigated using placebo-controlled research. One recent review of 252 placebo-controlled trials of pharmaceutical treatment of depression estimated that the placebo effect was responsible for 35 to 40% improvement in symptoms (Furukawa et al., 2016). It is important to determine the predictors of the placebo effect to attempt to control those variables. In treatment, the goal is to maximize placebo effects to improve outcomes. The research aims to control placebo effects to most accurately assess the effect of the "active" treatment.

The term “placebo effect” definition varies according to the source and context and is therefore multifaceted and almost polymorphic. While Kienle and Kiene (2001) describe the placebo effect simply as the “therapeutic effect of a placebo administration” (p. 40), Ernst (2001) defines it in more mathematical terms as the “difference in outcome between a placebo-treated group and an untreated control group in an unbiased experiment” (p. 182). Wolf (1959), in a matter-of-fact way, states it is “any effect attributable to a pill, potion, or procedure, but not to its pharmacodynamic or specific properties” (p. 287). Lastly, Andrews (2001) defines the placebo effect as “the sensitivity of patients to the encouragement that comes from being treated” (p. 192).

The placebo effect points intriguingly toward the complexity of the brain and how expectations and attitudes can manifest in changes in perceptions of symptoms. Seligman (2000) connected learned helplessness to poor health outcomes, which indicates that a person’s attitude impacts his or her state of being. Likewise, a person’s attitude and expectations around a placebo may also likely affect his or her outcomes (Vallance, 2006).

The placebo effect plays a significant role in medical research and the treatment of depression (Andrews, 2001). In a systematic review of 19 placebo-controlled antidepressant trials, Andrews reported that half of the improvement in patients was attributed to the placebo effect, while only one quarter was attributed to active medication. The placebo response rate in studies of depression is significantly higher than in studies on anxiety, schizophrenia, and agoraphobia (Andrews). Vallance (2006) agreed with these findings, stating, “a large proportion of the clinical effect of antidepressant

medication is attributable to the [placebo] effect" (p. 287). Due to its remarkable capacity for therapeutic impact in depressed patients, Andrews concludes that the placebo effect should be potentiated in treating depressed patients. Even though both Andrews and Vallance concurred that further research needs to be conducted, no further research has been published on this topic in the past decade.

Pessimism–Optimism Scales and Placebo

The research reviewed in this chapter implies an association between the explanatory style of the PSM scale and placebo response. It provides evidence that depressed patients scoring higher on optimism might have more positive responses to medication and placebos. The development of the PSM scale postdates most of the research focused on placebo response prediction. The PSM scale has not been used in investigations examining the prediction of placebo outcomes and represents a new mode of testing the association between placebo and depression outcomes.

Rief and Petrie (2016) describe a psychological expectation model that can be used to understand how expectation plays a vital role in the placebo effect, stating, "Placebo effects occur when a medical treatment and its context trigger specific expectations about a positive therapeutic outcome" (p. 2). Optimistic expectations increase the placebo effect, while pessimistic expectations are thought to increase nocebo effects. "Pre-existing optimistic expectations can amplify the positive effects of treatments (placebo effects), but negative expectations can also induce adverse treatment effects, such as side effects or the absence of treatment-typical improvements (nocebo

effects)" (Rief & Petrie, p. 2). According to Rief and Petrie, optimism and pessimism play significant roles in treatment outcomes.

Although previous research on the PSM scales has explored its predictive ability regarding depressive symptoms (Peterson & Bossio, 2001; Seligman, 2000; Suzuki et al., 2014), this study furthered existing research by examining whether the PSM scales can be used to predict treatment outcomes in depressed individuals enrolled in a medication trial, and, additionally, whether the PSM scale can predict placebo response. Both depression symptoms and somatic complaints were used as dependent variables, and both active and placebo treatment groups were examined for potential change.

Research into the predictive ability of the MMPI PSM scales by Seligman and others in positive psychology is a promising development that needs to be pursued. Given the success of research on the PSM scale with other outcomes (see Novotny et al., 2010; Singh et al., 2016; Suzuki et al., 2014), the use of the PSM to predict outcomes in the treatment of depression in both active and placebo treatment is a significant gap that needed to be investigated. Previous research has clearly shown that explanatory style significantly impacts placebo treatment outcomes with several conditions (Geers et al., 2005; Malani, 2006). This study broke new ground by testing the impact of explanatory style on placebo treatment of depressed patients.

The current research findings benefit individuals in treatment for depression, healthcare professionals seeking treatment options for their patients, and society in general by identifying how optimism–pessimism interacts with the pharmaceutical treatment of depression. The findings contribute to psychological treatment literature by

noting themes that might be incorporated into treatment approaches to make them more effective. In addition, the secondary focus on predicting placebo responses will contribute to the research in this field, which thus far has not led to identifying individuals who are more likely to be placebo responders.

Summary and Conclusions

The literature review provided in this chapter demonstrates the predictive ability of many scales on the MMPI, including overreporting symptoms (Blanchard et al. 2003), a diagnosis of PTSD in veterans with disabilities (Martz et al., 2005), and success at a military-style residential program (Weis et al., 2004). However, the reliability of the MMPI as a predictive tool is inconsistent. Chisholm et al. (1997) found three MMPI-2 clinical scales (depression, psychopathic deviate, and anxiety) and four content scales (depression, antisocial practices, anxiety, and negative treatment indicators) to be poor predictors of premature termination in therapy. Although a few studies have successfully demonstrated the MMPI's predictive ability to identify depression and somatic complaints (Burkhart et al., 1980; Gross et al., 2000; Suzuki et al., 2014), no studies have been able to predict treatment outcomes for either successfully. Therefore, research into the capacity of the MMPI to function as a predictive tool has yielded mixed results.

Peterson et al. (1988) and Seligman (1989) found that a "pessimistic explanatory style predicted stressful life events, poor health habits, and decreased feelings of self-efficacy" (Colligan et al., 1994, p. 76). The studies reviewed in this chapter include findings that relate pessimistic explanatory styles to worse outcomes, while optimistic explanatory styles have predicted better outcomes. These findings provide a solid

foundation for using the PSM scales to predict outcomes in a placebo-controlled depression and somatic complaints treatment.

Multiple researchers have found that depression and somatic complaints are highly correlated (Bair et al., 2003; Kapfhammer, 2006; Tylee & Gandhi, 2005). Further, Kurt et al. (2010) found that somatic complaints, anxiety, and depressive symptoms are comorbid, known as the "SAD" triad. This research on depression and somatic complaint outcomes provides a context in which current research outcomes may be better understood.

Having investigated the possible association between optimism–pessimism and placebo treatment of depression and somatic complaints, the current study has expanded previous research findings. Malani (2006) claimed that optimistic patients respond more favorably to placebo treatment than pessimistic patients. Geers et al. (2005) found little correlation between pessimists and adverse placebo effects. According to Rief and Petrie (2016), optimistic expectations increase placebo effect, and pessimistic expectations are thought to increase nocebo effects.

The current study addressed the lack of knowledge about the association between optimism–pessimism and treatment outcomes in depression by exploring the potential of PSM scales to predict pharmacological and placebo treatment outcomes of depression and somatic complaints. Therefore, this study builds upon and expands previous research using the MMPI to predict depression and somatic complaints (Burkhart et al., 1980; Gross et al., 2000; Suzuki et al., 2014). This study has developed knowledge that would enable treatment providers to be more effective.

Chapter 3: Research Method

Introduction

This study used archived data from a previous study that examined pretreatment and posttreatment data in three treatment groups. Two groups were administered an antidepressant medication (alprazolam and imipramine), and one was administered a placebo (Mendels & Schless, 1986). The authors investigated potential cause/effect relationships between the three treatment groups (imipramine, alprazolam, and placebo) and changes in depression, anxiety, and somatic symptom scores. Mendels and Schless collected MMPI profiles from the participants, in addition to depression and somatic symptoms, but did not use these data in their analysis. The current study used the MMPI data to investigate whether or not PSM scale scores predicted treatment outcomes in the archived data from that research.

The study aimed to investigate whether optimism-pessimism, as measured by the PSM scale (Malinchoc et al., 1995) of the MMPI, could predict treatment outcome in a placebo-controlled pharmacological drug treatment study for depression. The independent variables in the current study were the MMPI PSM scores and the treatment groups, and the dependent variables were the pretreatment and posttreatment scores of the HAM-D (Hamilton, 1980) as well as the SCL-61 (Derogatis et al., 1974) from the archived data from the Mendels and Schless (1986) study. This chapter describes the research design, rationale, and methodology of the study. The sampling procedures, data collection process used in the archival data, instrumentation, and data analysis plan are

also explained. Lastly, threats to validity (external and internal) and ethical procedures are described.

Research Design and Rationale

The current study used a quantitative design and archival data. The original study had an experimental design (Mendels & Schless, 1986) that used a control group and two experimental groups. The current design was appropriate to address the research questions because the variables could be expressed quantitatively and with a large sample of participants. The research questions focused on cause/effect correlations between independent variables and a dependent variable through the original design of randomly assigning participants to treatment groups to control the distribution of potentially confounding variables associated with the independent variables. The original research study (Mendels & Schless, 1986) used an experimental design to assess treatment outcomes for the three treatment groups. However, it did not examine a possible predictive relation between the MMPI scales and the dependent variable, which was the change in depression score. The current study accessed the archived data to examine whether optimism/pessimism, as assessed by the PSM scale of the MMPI, was associated with depression scores (Malinchoc et al., 1995).

The first research question addressed whether the PSM score predicted a change in depression score after treatment with antidepressants or a placebo. The dependent variable used to address this question was the change in the depression between the baseline (Week 0) and the final posttreatment assessment (Week 10) as assessed by the HAM-D instrument (Hamilton, 1960, 1980). The second research question addressed

whether PSM score predicted a change in somatic symptom score after treatment with antidepressants or placebo. The dependent variable was a change in somatic symptoms between the baseline (Week 0) and the final posttreatment assessment (Week 10) as assessed by the SCL-61 instrument (Derogatis et al., 1974).

Covariates were used to control for pretreatment levels of depression, and somatic complaints, such as pretreatment depression (Burkhart et al., 1980; Gross et al., 2000; Suzuki et al., 2014) and pretreatment somatic symptoms (Murberg, 2012; Singh et al., 2016), have been demonstrated to predict treatment outcome, with individuals with lower pretreatment scores (mild to moderate symptom levels) experiencing more significant improvement than those with high pretreatment scores. Using pretreatment assessment levels of symptoms as covariates controlled the influence of initial symptom levels on treatment outcomes. Pretreatment depression severity as measured by the MMPI depression scale (Hathaway & McKinley, 1940) was used as a covariate in the first analysis, and pretreatment somatic complaint severity as measured by the MMPI hypochondriasis scale (Hathaway & McKinley, 1940) was used as a covariate for the second analysis.

The original study employed an experimental design because it "seeks to determine if a specific treatment influences an outcome" (Creswell, 2018, p. 247). The current study was a quantitative chart review study that used archived data. This study used a correlational analysis to study the effect of the participants' optimism/pessimism as an independent variable on the dependent variables of depression and somatic symptom scores. The three treatment groups (imipramine, alprazolam, and placebo) were

also used as independent variables. They were used as the only independent variables in the original research and demonstrated a significant impact on change in depression scores. In the previous study by Mendels and Schless (1986), the group taking alprazolam experienced the most improvement in depressive symptoms after treatment. Almost half the participants reported an over 50% symptom reduction on the HAM-D. In the group taking imipramine, 38% of the participants experienced at least a 50% improvement in symptoms of HAM-D (Mendels & Schless, 1986). Eighteen percent of the individuals in the placebo group reported the same degree of improvement.

This design was an appropriate way to address the research questions. Using archival data allowed for minimal constraints in time regarding gathering data. It also allowed for data that the original researchers did not utilize to develop deeper insight into how participant psychological profiles moderated treatment effects on patient outcomes. The original study remains relevant, as the medications are still widely prescribed today (Olin, 2014; Stahl, 2000). The MMPI instrument (Hathaway & McKinley, 1940) is a valid and reliable measure for depression and hypochondriasis. It continues to be used in clinical settings and research due to its predictive validity (Scholte et al., 2012). In addition, the data were collected by a team that documented adherence to the treatment protocol. The data used in the current study were collected during the original study but were not incorporated in any quantitatively meaningful way. The data were inspected for completeness and accuracy and then inputted into a database for digital access. All of the scored data were rescored to ensure accuracy.

Methodology

Population

The target population of the archived study was men and women aged 18 to 60 years who had MDD for at least 1 month prior to the study and could read and understand the symptom checklist, communicate intelligently with research personnel, and sign an informed consent. The study excluded pregnant women; individuals with significant liver, kidney, gastrointestinal, cardiovascular, or pulmonary disease; persons who were allergic to benzodiazepines or imipramine or addicted to alcohol or other drugs; persons who were taking a psychotropic drug, a potent analgesic, or an antihistamine; and those who had taken another investigational drug within the last month, or who had taken other antidepressants, significant tranquilizers, or benzodiazepines within the past 7 days. The screening included patient history and physical examination, psychiatric background, laboratory evaluation, electrocardiography, and clinical rating scales.

Sampling and Sampling Procedures

Procedures for recruitment for the original study (Mendels & Schless, 1986) involved advertising in local newspapers seeking adults who had been diagnosed with depression to participate in a study involving antidepressant medication. The study would last 3 months and required weekly appointments. The initial appointment was used to collect information regarding participants' histories and conduct a physical examination, including a psychiatric background, an electrocardiogram report, and laboratory evaluations. Several self-report evaluations were administered, including the MMPI (Hathaway & McKinley, 1940), the Raskin Depression Scale (Raskin et al., 1969), the

Hamilton Psychiatric Rating Scale for Depression (Hamilton, 1960, 1980), and the Core Symptoms—Major Depressive Episode Checklist (APA, 1980).

Weekly appointments were scheduled during the 10 weeks of the medication trial. These visits included administering the HAM-D (Hamilton, 1960, 1980), the Core Symptoms—Major Depressive Episode checklist (APA, 1980), and the SCL-61 (Derogatis et al., 1974). Information was collected at these weekly appointments, including side effects, the physician's global impressions, vital signs, medication dosage, and the patient's global impression of change. The sample size in the original study included 30 participants per group (alprazolam, imipramine, and placebo), and the researchers reported a medium effect size ($r = 0.3$) at power = .80 and $a = .05$ (Mendels & Schless, 1986), with the group taking alprazolam experiencing the greatest improvement in symptoms.

A research group kept the data accessed for the current study in Washington State. All identifying information was removed from the paper files, which were transferred to my possession for analysis when the study was approved.

Instrumentation and Operationalization of Constructs

The Minnesota Multiphasic Personality Inventory (MMPI)

The MMPI was developed by Starke R. Hathaway and J. C. McKinley in 1940. This instrument is a widely known personality test primarily designed to diagnose individuals into categories of neuroses and psychoses (Duckworth & Anderson, 1986). The test-retest reliability of the MMPI (Hathaway & McKinley, 1940) subscales ranges from .50 to .80. The MMPI (Hathaway & McKinley, 1940) scale with the lowest

reliability score is the Schizophrenia (Pa) scale, and the scale with the highest reliability score is the Social Introversion (Si) scale—both of which are clinical scales (Wise et al., 2010). The MMPI (Hathaway & McKinley, 1940) is a reliable and valid assessment tool often used to help diagnose mental health disorders (Butcher & Williams, 2009; Camara et al., 2000).

The validity, reliability, and clinical utility of the MMPI (Hathaway & McKinley, 1940) are supported by multiple studies (e.g., Bloomquist & Harris, 1984; Tarescavage et al., 2015; Tarescavage et al., 2013). In a study of 712 police officers and applicants, the MMPI (Hathaway & McKinley, 1940) evidenced good predictive validity for personality problems, including thought dysfunction (Tarescavage et al., 2015). Bloomquist and Harris (1984) "examined the utility of the three MMPI family scales with 110 undergraduates. Findings suggest that the MMPI family scales are reliable and concurrently valid measures of an individual's perception of interpersonal family relationships" (p. 1209). In a study of 759 bariatric surgery candidates, results supported the reliability, validity, and clinical utility of the restructured form (RF) scales for the MMPI (Hathaway & McKinley, 1940; Tarescavage et al., 2013).

Wise et al. (2010) conducted a literature review of studies that used the MMPI (Hathaway & McKinley, 1940) from 2000 to 2010 and found that only 27% of the men and 29% of the women in these samples had alpha coefficients that were greater than .80. According to Tavakol and Dennick (2011), the general range of acceptable alpha values to determine the internal consistency of a test is 0.70–0.95. Wise et al. reported that six out of 10 MMPI (Hathaway & McKinley, 1940) clinical scales demonstrated alpha

coefficients of under 0.70; one out of 15 content scales was under 0.70; 10 of the 16 supplemental scales were under 0.70, and two of the 10 restructured scales and three of the five PSY scales scored under 0.70 for alpha coefficients. Overall, the MMPI (Hathaway & McKinley, 1940) has low internal consistency: 22 out of 56 scales (39%) scored alpha coefficients under 0.70. However, of the four out of 10 scales that scored above .70, two were the hypochondriasis and depression scales (the two used in this study). The reliability coefficients for the hypochondriasis scale measured .77 for men and .81 for women (Wise et al., 2010). The test-retest reliability was between .80 and .90, making hypochondriasis one of the most stable scales (Duckworth & Anderson, 1986). The reliability coefficients for the depressive scale measured .85 for men and .86 for women. The test-retest reliability scale measured between .84 and .88 (Wise et al., 2010).

Most MMPI (Hathaway & McKinley, 1940) test-retest reliability scores are over 0.70 (Wise et al., 2010). However, test-retest scores were generally lower for clinical and supplemental scales as with alpha coefficients. This may point to reliability problems in the clinical and supplemental items. The generally low alpha coefficient scores may also indicate the MMPI's (Hathaway & McKinley, 1940) multifactorial content (Wise et al., 2010). The MMPI (Hathaway & McKinley, 1940) lacks internal consistency and covers many personality components, which means that it may be more multidimensional than the unidimensional test. Nonetheless, this lack of internal consistency may point to potential weaknesses in this study. An older review of a literature study examining psychometric qualities of the first version of the MMPI (Hathaway & McKinley, 1940)

reported reliability scores ranging from 0.71 to 0.84 and stability values ranging from 0.63 to 0.86 (Hunsley et al., 1988).

The Hamilton Psychiatric Rating Scale for Depression (HAM-D; Hamilton, 1980)

The HAM-D was published by Max Hamilton in 1960 and revised multiple times until 1980 (Hamilton, 1960, 1980). The HAM-D (Hamilton, 1980) was an appropriate scale to measure depression in the archived study. It assesses mood, insomnia, guilt, agitation, suicide ideation, weight loss, anxiety, and somatic symptoms. This measure has been tested with various populations, including clinical and nonclinical children, adolescents, and adults, to determine the severity of depression and depressive symptoms (Worboys, 2012).

In a prospective and observational study, including a 4-week follow-up of 173 patients with manic depression and bipolar disorder, the HAM-D (Hamilton, 1960, 1980) was found to exhibit internal consistency, appropriate convergent and discriminant validity, and test-retest reliability (González-Pinto et al., 2009). In a 422 terminal cancer patients study, the HAM-D (Hamilton, 1980) exhibited high reliability and concurrent and convergent validity with a clinical depression diagnosis (Olden et al., 2009). The HAM-D (Hamilton, 1980) also demonstrated a high degree of predictive accuracy and excellent levels of specificity and sensitivity (Olden et al., 2009). The reliability and interrater reliability of the HAM-D (Hamilton, 1980) were sufficient. Discriminant validity was established using a receiver operating characteristics (ROC) curve analysis, confirming high sensitivity and specificity levels (Olden et al., 2009). Other researchers

have also established high levels of validity and reliability (see Halfaker et al., 2011; Sajatovic et al., 2015).

The Symptom Checklist-61 (SCL-61; Derogatis et al., 1974)

The SCL-61 is a widely used screening instrument initially developed as the Hopkins Symptom Checklist (HSCL) in the 1950s by John Hopkins University researchers Parloff et al. (1954). The original 25-item HSCL was subsequently revised and expanded to a 61-item Symptom Checklist (SCL; Derogatis et al., 1974), the screening instrument used in the archived study by Mendels and Schless (1986). Based on somatic symptoms of outpatients, the SCL-61 (Derogatis et al., 1974) examined five distinct groupings of symptomology: obsessive-compulsive, somatization, interpersonal sensitivity, depression, and anxiety (Derogatis et al. 1974).

The validity and reliability of the SCL-61 (Derogatis et al., 1974), as well as previous versions of the HSCL, have been tested in both normative and neurotic-depressive samples, demonstrating high levels of internal consistency and reliability that were tested with coefficients alpha, as well as high test-retest reliability as measured through test-retest coefficients, and good levels of interrater reliability (Derogatis et al., 1974). In a study of 37 older adults in primary care, the HSCL-25 evidenced a sensitivity rate of 94% for identifying depression (Fröjdh et al., 2004). In a study of 158 people with HIV living in rural Uganda, the HSCL evidenced alpha coefficients of 0.83 to 0.91 (Ashaba et al., 2018). In a study of 116 patients in Afghanistan health care centers, the HSCL-25 showed an alpha coefficient of 0.73 in identifying mental disorders

(Ventevogel, 2007). The HSCL is thus established as a reliable and valid measure for determining depression and somatic complaints.

Operationalization of the Variables

Independent Variables

Two independent variables were used in the study analysis. The first was the treatment group, which has three categories (alprazolam, imipramine, and placebo). The second independent variable is optimism-pessimism. This continuous variable was measured using the PSM scores (Malinchoc et al., 1995) calculated from each participant's MMPI (Hathaway & McKinley, 1940) data. In order to use optimism–pessimism as an independent variable, it was converted to a categorical variable as described below. The MMPI was administered to all participants at baseline in the original study.

To calculate the PSM score, two sets of numbers were used. There are 85 “good event” or CoPos items and 178 “bad event” or CoNeg items. Each item bears a composite weight ranging from 10.33 to 20.33 on the CoPos table and from 6.33 to 20.33 on the CoNeg table (Malinchoc et al., 1995). Once the values are obtained, the scores are then classified as pessimistic (having a PSM score greater than 60), mixed (a PSM score in the 40 to 60 range), or optimistic (a PSM score less than 40) (Maruta et al., 2002).

The treatment group (alprazolam and imipramine) was the main variable of interest in the original research (Mendels & Schless, 1986). It was found to impact depression scores, as discussed earlier in the chapter. Because the treatment groups had already been examined as the main variable of interest in the original Mendels and

Schless (1986) research, it was not used as a main variable of interest. However, the impact of the treatment group was taken into account, given its demonstrated impact on the outcome, and used as a second independent variable. This allowed for the examination of interaction effects to identify whether the combination of PSM score and treatment group predicted change differently and examined how optimism-pessimism interacted with the placebo response in particular.

Dependent Variables

The two dependent variables of this study were the depression score as measured by the HAM-D (Hamilton, 1960, 1980) and the somatic complaint score as measured by the SCL-61 (Derogatis et al., 1974). The HAM-D (Hamilton, 1960, 1980) was administered during pretreatment and each week during the 10-week study. Somatic complaints were assessed simultaneously using the SCL-61 (Derogatis et al., 1974). The pretreatment HAM-D (Hamilton, 1960, 1980) score (Week 0) and the posttreatment Week 10 HAM-D (Hamilton, 1960, 1980) score were used to address the first research question. Similarly, the baseline pretreatment SCL-61 (Derogatis et al., 1974) score (Week 0) and Week 10 posttreatment SCL-61(Derogatis et al., 1974) score were used to address the second research question.

Covariates

The original research study (Mendels & Schless, 1986) did not use covariates in its analysis; however, adding covariates in each of the equations allowed for pretreatment severity of symptoms to be taken into account in predicting outcome and enabled a more conservative analysis of the data. The MMPI (Hathaway & McKinley, 1940) was

administered to all participants at pretreatment during the original study; however, the scores were not used by the original researchers, making the subscales of the MMPI valuable data for the current analysis

The depression severity scores computed from the MMPI (Hathaway & McKinley, 1940) were used as a covariate in the analysis to address the first research question. Somatic complaint severity was also measured at pretreatment by the hypochondriasis scale on the MMPI (Hathaway & McKinley, 1940) and therefore used as a covariate in the analysis addressing the second research question.

Data Analysis Plan

SPSS version 25 was used for the data analysis. All the archived data are in paper format and were checked for completeness. Data were entered into a Microsoft Excel spreadsheet, and coded scores were imported into SPSS version 25. In order to avoid biased results, the data were checked for univariate and multivariate outliers. The univariate outliers are those with *z* scores greater than 3.3 as defined by the SPSS program. Multivariate outliers were found by computing a Mahalanobis Distance for each case. Once that was done, those scores were screened similarly to the univariate outliers. The data were then reviewed to identify missing values and to rescore and recheck the data for accuracy. In order to be included in the analysis, cases were required to include MMPI scores (specifically the depression, hypochondriasis, and PSM scales), as well as the HAM-D and SCL-61 pretreatment posttreatment scores. Cases with any of that data missing were to be excluded from the study.

The statistical models used to analyze the study designs were repeated measures mixed with ANCOVA. Two repeated measures mixed ANCOVA models were developed, as presented in the table below. The first model has the HAM-D depression score as the dependent variable. The second model has the SCL-61 somatic complaint scores as the dependent variable.

Table 1

Analysis Design by Research Question

Research question	Variable	Statistical test
RQ1: Does optimism-pessimism level affect depression level change after controlling for baseline depression severity in a group of depressed adults treated with antidepressants or placebo?	IVs: Treatment group PSM category DV: Change in depression as measured by HAM-D score Covariate: Pretreatment MMPI depression scale score Within Subjects Factor: Time: Pretreatment and posttreatment	Repeated-measures ANCOVA
RQ2: Does optimism-pessimism level affect somatic symptom change after controlling for baseline somatic symptom severity in a group of depressed adults treated with antidepressants or placebo?	IVs: Treatment group PSM category DV: Change in somatic symptoms as measured by SCL-61 score Covariate: Pretreatment MMPI hypochondriasis scale score Within-subjects factor: Time: Pretreatment and posttreatment	Repeated-measures ANCOVA

The assumptions for repeated measures of ANCOVA (Neter et al., 1996) are as follows:

1. Random assignment and independent sampling. This means that each case was assumed to be independent of the other and that cases were randomly assigned to distribute extraneous variables that impacted treatment outcomes evenly. The assumption was addressed by the original research design, which utilized random assignment to treatment groups.
2. Multivariate normality of error variance. This was examined by plotting the residuals visually to examine the data slopes. A log transformation was performed on the data when this assumption was violated.
3. Multivariate homogeneity of error variance. A Levene's test of equality of variances was performed to test for this assumption before the analysis. A Kruskall-Wallace nonparametric analysis was performed when this assumption was violated instead of an ANCOVA.
4. Independence of covariate and treatment effect. An ANOVA was performed to examine the relation between covariate and treatment effect to determine if the covariate differed across the groups. When there was a violation of this assumption, an alternative analysis plan was considered, including a regression analysis or a Kruskall-Wallace analysis.
5. Homogeneity of regression slopes. Violations of this assumption can be tested by examining the interaction effect of the covariate and the independent

variable. A Johnson-Neyman strategy was used when there was a significant violation of the homogeneity.

Research Questions

RQ1: Does optimism-pessimism level affect depression level change after controlling for baseline depression severity in a group of depressed adults treated with antidepressants or placebo?

$H_0$1: There is no significant effect of optimism-pessimism as assessed by PSM level on depression symptom change, as assessed by HAM-D scores, at baseline and posttreatment after controlling for baseline depression measured using the MMPI depression score.

$H_1$1: There is a significant effect of optimism-pessimism as assessed by PSM level on depression symptom change, as assessed by HAM-D scores, at baseline and posttreatment after controlling for baseline depression measured using the MMPI depression score.

$H_0$3: There is no significant interaction effect between antidepressant treatment and PSM level on depression symptom change, as assessed by HAM-D scores, at baseline and posttreatment after controlling for baseline depression measured using the MMPI depression score.

$H_1$3: There is a significant interaction effect between antidepressant treatment and PSM level on depression symptom change, as assessed by HAM-D scores, at baseline and posttreatment after

controlling for baseline depression measured using the MMPI depression score.

RQ2: Does optimism-pessimism level affect somatic symptom change after controlling for baseline somatic symptom severity in a group of depressed adults treated with antidepressants or placebo?

$H_0$1: There is no significant effect of optimism-pessimism, as assessed by PSM level, on somatic symptom change, as assessed by SCL-61 scores, at baseline and posttreatment after controlling for baseline depression measured using the MMPI hypochondriasis score.

$H_1$1: There is a significant effect of optimism-pessimism, as assessed by PSM level, on somatic symptom change, as assessed by SCL-61 scores, at baseline and posttreatment after controlling for baseline depression measured using the MMPI hypochondriasis score.

$H_0$3: There is no significant interaction effect between antidepressant treatment and PSM level on somatic symptom change, as assessed by the SCL-61, at baseline and posttreatment after controlling for baseline somatic symptoms measured using the MMPI hypochondriasis score.

$H_1$3: There is a significant interaction effect between antidepressant treatment and PSM level on somatic symptom change, as assessed by the SCL-61, at baseline and posttreatment after controlling for

baseline somatic symptoms measured using the MMPI hypochondriasis score.

Threats to Validity

Threats to validity include both external and internal factors. The data used for this study were archived; therefore, no prospective controls could be used for data collection. The initial study may have encountered extraneous effects wherein participants may have been exposed to events that could interact or interfere with the study effects. For example, the interviewer may have unintentionally led the individual to answer questions in a certain way, or individuals may have thought they needed to respond a certain way to participate in the study. Possible internal validity threats include inaccurate data scoring; all data were re-scored to minimize this threat.

Threats to external validity occur when researchers make inaccurate references from the sample to general populations, settings, or past or future situations (Creswell, 2009). In order to address external validity threats, no generalizations were made outside of the selected population. The Mendels & Schless study (1986) may have encountered selection bias as volunteers were recruited through ads to participate in the study. In order to participate, specific scores on the depression scales needed to be met. Those who did not have a HAM-D score of at least 26 and a Raskin Depression Scale score of at least ten were excluded from the study. Because these participants may have volunteered for a specific purpose and were self-selected, their responses during the study may have been affected by self-selection bias. The findings may not be generalizable to the population of depressed adults.

The PSM scale has recent, relevant research and is a topic of ongoing investigations. The archived data used in this study were collected over 30 years ago. The description of depression has been subsequently redefined by the DSM-5 (APA, 2013). Therefore, the participants may have presented with a slightly different description of depression than what is used by the DSM-5 today. Additionally, the MMPI has been updated as the MMPI2 and the MMPI2 RF (Butcher et al., 1989), and over time several questions have changed or been omitted. Researchers' understanding of key concepts may be different now because of updated research; however, the MMPI clinical scales and the medications employed in the original study are still used clinically; therefore, the findings of the present study may be useful in informing future studies.

The setting of the original study may have limited generalizability. Participants came into a structured setting to complete the weekly depression scales and were limited to a specific geographic location. Another potential threat that may impact validity is that the participants were informed that they were in a placebo-controlled study. Individuals in the placebo group may have correctly guessed their group assignment.

Maturation and regression to the mean also represented threats to validity. Time passing can impact symptoms, as can monitoring symptoms, and several participants may have experienced changes in symptoms during their time in the study. Attrition could be viewed as another possible threat to validity, as individuals may have dropped out if they were not experiencing results in their treatment. This would have biased the original study's findings by including individuals who benefitted from treatment and excluding those who left the study when they found that treatment was not beneficial.

Another possible threat to validity was statistical conclusion validity. Garcia-Perez (2012) defines this as the "degree to which the conclusions found in the data are reasonable" (p. 2). This study accounted for possible statistical conclusion validity issues by increasing statistical power close to 1.0 and using measurements with good reliability (Trochim, 2006).

Ethical Procedures

Institutional Review Board (IRB) approval was obtained, and data was stored on a secure computer. Physical data were locked in a filing cabinet. The researcher and her committee members were the only individuals with access to the data, and all data provided and used in the current study were anonymous from an archived data set. The initial researchers followed the ethical procedures of informed consent, and the names and personal information of the subjects were removed from the data. The data were returned to the initial researchers upon completion of the study, and the raw data were kept secure in a locked office while it was being scored and input.

Summary

The current study applied archived data from Mendels and Schless's (1986) research using a quantitative research design. The study sought to determine if optimism–pessimism levels predicted change in depression and somatic symptom scores with treatment in three study participants: those receiving treatment with alprazolam, imipramine, or placebo. Two research questions examined if the PSM pretreatment score could predict depression and somatic scores changes. The severity of pretreatment depression and somatic symptoms was accounted for by using covariates. Chapter 4

presents the statistical analyses of the relationships between the study variables, ultimately determining the answers to the research questions and which hypotheses were confirmed and not confirmed.

Chapter 4: Results

Introduction

Through this study, I aimed to investigate whether the level of optimism/pessimism can significantly predict treatment outcomes of antidepressant medications and placebo in individuals enrolled in a clinical trial for depression. The study used archived data from research conducted by Mendels and Schless (1986) of 100 participants who were treated with alprazolam (n = 33), imipramine (n = 34), or placebo (n = 33). Mendels and Schless administered the MMPI to all participants at pretreatment. They measured pretreatment and posttreatment levels of depression with the HAM-D (Hamilton, 1980) and somatic symptoms with somatic scores on the SCL-61 (Derogatis et al., 1974).

The first research question focused on whether participants' levels of optimism/pessimism predicted changes in depression scores after controlling for baseline depression severity in adults diagnosed with depression and treated with either antidepressants or a placebo. The second research question addressed whether participants' levels of optimism/pessimism were related to changes in the severity of somatic symptoms of depression after controlling for baseline somatic symptom severity in adults diagnosed with depression and treated with either antidepressants or placebo. Pretreatment MMPI depression scores were used to control for baseline depression severity, and pretreatment MMPI hypochondriasis scores were used to control for baseline somatic symptom severity.

In this chapter, I describe the data collection procedures and provide descriptive statistics of the data. In the results section, I describe and develop the statistical models to address the research questions. Assumptions of the models and the study's findings are presented, and the chapter ends with a summary.

Data Collection

The original researchers obtained the archived data, scored the data, and entered the data into a Microsoft Excel spreadsheet. After the MMPI data were scored, the participants were categorized as pessimistic (PSM-T scores equal to or greater than 51) or optimistic (PSM-T scores equal to or less than 50). This represented a change from the original plan of separating the participants into three groups, as only five individuals were in the “mixed” group.

The participant data were reviewed before the analysis, and five participants were deleted from the study for missing HAM-D or SCL-61 scores. This included one 53-year-old man in the imipramine/pessimist group, a 21-year-old woman in the placebo/pessimist group, a 21-year-old woman in the alprazolam/pessimist group, and a 39-year-old woman in the alprazolam/pessimist group, and a 36-year-old woman in the imipramine/optimist group.

Age and sex were the only demographic variables included in the acquired data. Table 2 shows the participants identified as optimists or pessimists across alprazolam, imipramine, and placebo treatment groups. Table 3 shows the sex of the participants by treatment group, and Table 4 shows the age of the participants by treatment group.

Table 2

Numbers of Participants Identified as Optimists/Pessimists Across Treatment Groups

Treatment group	Optimist	Pessimist	Total
Alprazolam	9	22	31
Imipramine	8	24	32
Placebo	3	29	32
Total	20	75	95

A chi-square test was conducted to determine whether the distribution of participants in the optimist-pessimist categories was different across treatment groups. Overall, more individuals were categorized as pessimists (78% of the sample) than optimists. There was no significant disparity in distribution between the groups ($\chi 2$ [2, n = 95] = 4.11, p =.128).

Table 3

Sex Distribution of Participants by Treatment and Optimist/Pessimist Group

	Optimist group	Pessimist group	Total
Male	19	9	28
Female	1	66	67
Total	20	75	95

	Male	Female	Total
Alprazolam	8	23	31
Imipramine	14	18	32
Placebo	6	26	32
Total	28	67	95

To test whether participant sex was evenly distributed across the optimist/pessimist groups, another chi-square test was conducted, with the results indicating a significant difference that is also clear in Table 2 ($\chi 2$ [1, n = 95) =52.33, p < .001). Men were more likely to be categorized as optimists, and women were more likely to be categorized as pessimists. This indicated that sex needed to be considered in the hypothesis testing analysis. The chi-square test to determine whether sex was evenly distributed by treatment group was not significant; however, there was a trend ($\chi 2$ [2, n = 95] = 4.11, p =.13), which was apparently due to women being slightly more likely to be assigned to the alprazolam and placebo groups than the imipramine group.

Table 4

Age of Participants by Treatment Group

	Optimist		Pessimist		Total	
	N	Mean age (*SD*)	*N*	Mean age (*SD*)	*N*	Mean age (*SD*)
Alprazolam	9	39.44 (6.31)	22	38.50 (15.65)	31	38.77 (13.50)
Imipramine	8	40.50 (9.27)	24	37.08 (12.01)	32	37.94 (11.35)
Placebo	3	34.33 (3.51)	29	43.10 (12.50)	32	42.28 (12.19)
Total	20	39.10 (7.36)	75	39.83 (13.44)	95	39.67 (4.76)

An independent *t* test was used to determine whether there was a difference in mean age between the optimist and pessimist groups. Levene's test for equality of variances was significant (F = 17.09, p < .001); therefore, the equal variances were not assumed statistic was used and was not significant (t [56.34] = -.32, p = .75), indicating

that there was no age difference by optimist/pessimist groups. There was also no difference in mean age between the treatment groups (F [2,92] = 1.11, p = .33).

Results

First, the assumptions for the comparisons were tested, and then the MMPI Depression and Hypochondriasis scores were compared according to the group (see Table 5). Before hypothesis testing, the data were examined for outliers and plotted. The planned covariates for the model were then examined to determine if they differed by treatment group or optimist/pessimist group.

Table 5

MMPI Baseline Depression and Hypochondriasis Scores by Treatment Group

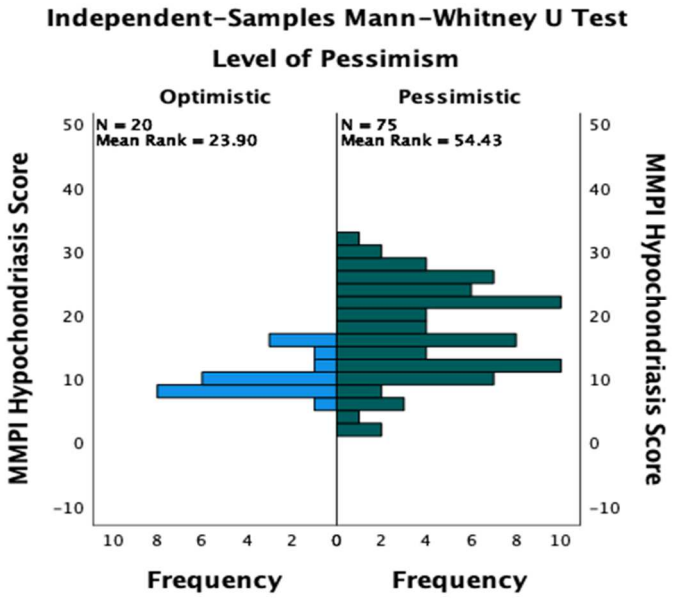
	MMPI Depression score						MMPI Hypochondriasis score					
	Optimistic		Pessimistic		Total		Optimistic		Pessimistic		Total	
	N	Mean (*SD*)	*N*	Mean (*SD*)	*N*	Mean (*SD*)	*N*	Mean (*SD*)	*N*	Mean (*SD*)	*N*	Mean (*SD*)
Alprazolam	9	34.11 (3.822)	22	39.14 (4.521)	31	37.87 (4.918)	9	8.44 (2.651)	22	20.05 (7.088)	31	16.68 (8.105)
Imipramine	8	37.13 (4.390)	24	39.29 (6.175)	32	38.75 (5.792)	8	10.38 (3.462)	24	16.25 (8.141)	32	14.78 (7.653)
Placebo	3	36.67 (3.512)	29	35.69 (5.224)	32	35.78 (5.053)	3	10.67 (2.082)	29	15.38 (6.144)	32	14.94 (6.026)
Total	20	35.70 (4.092)	75	37.93 (5.586)	95	37.46 (5.365)	20	9.55 (2.982)	75	17.03 (7.287)	95	15.45 (7.280)

All the assumptions for the comparisons were met with the exception that the equality of variance test was significant for the MMPI Hypochondriasis scores (F [5, 89] = 6.32 p < .001). There was not a statistical difference in the Hypochondriasis score by treatment group (p = .67); however, there was a difference between the optimist/pessimist groups on the MMPI Hypochondriasis score, with the pessimist group scoring higher than the optimist group (U = 268, p < .001; see Figure 1). There was no statistically significant difference between the means of MMPI baseline depression scores by either

treatment group (p = .47) or optimist/pessimist group (p = .13). Hence, Mann-Whitney U tests were used for these comparisons.

Figure 1

Distribution of MMPI Hypochondriasis Scores Across the Two Groups



Examination of the Pretest Scores

The assumptions were met for a multivariate comparison (MANOVA) for the HAM-D and SCL-61 pretreatment scores. The overall model was significant (F [2, 88] = 1388.95 p < .001), with a nonsignificant effect for treatment group (F [4, 176] = 1.22 p = .30) and a significant effect for optimist/pessimist group (F [2, 88] = 5.68, p < .005). The between-subjects effects comparisons revealed no significant differences for pretreatment HAM-D scores by the optimist/pessimist group (p = .243) but a significant difference for SCL-61 somatic symptom scores by the optimist/pessimist group (p < .001). The

pessimist group's somatic symptom mean score (138.21, *SD* = 25.06) was significantly higher than the optimist somatic symptom mean score (119.30, *SD* = 23.90). This was consistent with the finding that MMPI somatic symptoms differed by group, supporting using the MMPI somatic symptoms scale as a covariate in the analysis.

Table 6

Baseline HAM-D Depression and SCL-61 Somatic Symptom Severity Scores by Treatment Group

	HAM-D baseline depression scores						SCL-61 baseline somatic symptom severity scores					
	Optimist		Pessimist		Total		Optimist		Pessimist		Total	
	N	Mean (*SD*)	*N*	Mean (*SD*)	*N*	Mean (*SD*)	*N*	Mean (*SD*)	*N*	Mean (*SD*)	*N*	Mean (*SD*)
Alprazolam	9	27.78 (3.073)	22	30.14 (4.764)	31	29.45 (4.426)	9	127.78 (24.422)	22	143.59 (26.303)	31	139.00 (26.393)
Imipramine	8	29.25 (2.493)	24	27.79 (5.217)	32	28.16 (4.691)	8	115.38 (25.740)	24	141.42 (24.193)	32	134.91 (26.742)
Placebo	3	27.67 (6.429)	29	31.17 (4.260)	32	30.84 (4.487)	3	104.33 (1.155)	29	131.48 (24.120)	32	128.94 (24.294)
Total	20	28.37 (3.345)	75	29.79 (4.883)	95	29.48 (4.624)	20	119.30 (23.901	75	138.21 (25.061)	95	134.23 (25.884)

Examination of the Posttest Scores

Next, the posttest HAM-D and SCL-61 somatic symptom scores were examined. Levene's tests were not significant for either variable but demonstrated a trend for the posttest SCL-61 scores ($p = .22$ for the posttest HAMD scores, $p = .07$ for the posttest SCL-61 scores). Therefore, Pillai's trace statistic was used instead of Wilks's lambda statistic, which is more conservative. The overall model was significant (F [2, 88] = 308.50, $p < .001$). The main effect for treatment group was significant (F [4,178] =3.31, $p < .01$), and the main effect for optimist/pessimist group demonstrated a trend (F [2,88] =2.72, $p = .07$). The interaction effect was not significant ($p = .69$).

The post hoc least significant difference (LSD) tests were conducted to examine treatment group differences. They showed a statistically significant difference in mean posttest SCL-61 somatic symptom scores between the alprazolam and placebo groups ($p < .001$) and the imipramine and placebo groups ($p < .03$). The placebo group had the lowest mean score (12.12, $SD = 7.23$) compared to the alprazolam (20.13, $SD = 10.00$) and imipramine (17.06, $SD = 9.47$) groups.

Table 7

Posttest Depression Severity Score (Posttest HAM-D Scores)

	Optimist		Pessimist		Total	
	N	Mean (*SD*)	*N*	Mean (*SD*)	*N*	Mean (*SD*)
Alprazolam	9	22.22 (7.612)	22	13.27 (10.837)	31	20.13 (9.976)
Imipramine	8	17.50 (11.326)	24	16.92 (9.036)	32	17.06 (9.466)
Placebo	3	19.33 (10.116)	29	11.48 (6.780)	32	12.12 (7.228)
Total	20	19.75 (9.369)	75	15.51 (9.344)	95	16.40 (9.460)

Table 8

Posttest Somatic Symptom Severity (Posttest SCL-61 Scores)

	Optimistic		Pessimistic		Total	
	N	Mean (*SD*)	*N*	Mean (*SD*)	*N*	Mean (*SD*)
Alprazolam	9	123.11 (23.814)	22	124.41 (37.453)	31	124.03 (33.667)
Imipramine	8	116.63 (26.511)	24	120.29 (34.650)	32	119.38 (32.436)
Placebo	3	81.33 (13.279)	29	91.24 (28.643)	32	90.31 (27.586)
Total	20	114.25 (26.962)	75	110.27 (36.271)	95	111.11 (34.428)

Hypothesis Testing

After the variables were examined, the research questions were addressed to determine whether the null hypotheses should be rejected or retained. First, the assumptions of the ANCOVA were tested to determine if the analysis plan needed to be adjusted or if variables needed to be converted.

Testing of ANCOVA Assumptions

According to Neter et al. (1996), assumptions for an ANCOVA include the following:

Random Assignment and Independent Observations. Each subject was assumed to be independently and randomly sampled from the population and assigned to a treatment group. This assumption was addressed by the original research design, which utilized random assignment participants to treatment groups.

The Dependent Variable Must Be Continuous. This assumption was met, as both dependent variables were measured on a continuous scale.

Independent Variables Are Categorical With at Least Two Levels. This model has two independent variables, each with at least two levels: the treatment group and the optimism/pessimism group. Therefore, this assumption was met.

The Covariates (Pretreatment or Control Variable) Is/Are Continuous. The covariates were measured on continuous scales; therefore, this assumption was met.

Independent Observations. Each individual fell into only one group; therefore, this assumption was met.

There Are No Outliers in the Data Set. Outlier detection was completed in SPSS using *z*-scores and boxplots. A *z*-score of GT 3.0 or LT -3.0 was considered an outlier, and cases outside the stems of the boxplots were considered outliers. If a case was an outlier according to both methods, it was considered a "true outlier" and was culled from the data set. There were no outliers identified using this method.

Normal Distribution of Variables. Shapiro-Wilk tests were performed to test the assumption of normal distributions in the variables. The Shapiro Wilk demonstrated nonnormal distributions for both of the posttest scores (posttest HAMD $W(95) = .97$, $p < .05$; posttest SCL-61 $W(95) = .96$, $p < .006$). An examination of the histogram plots showed that the posttreatment scores skewed toward lower scores on both posttests as depression and somatic symptoms improved with treatment. Given the relatively large sample size and the robustness of the ANCOVA itself, the analysis plan was not changed.

Multivariate Homogeneity of Error Variance. This assumption was tested using Levene's test on each dependent variable. The assumption was not violated for the HAM-D pretreatment scores (F [5, 89] = 1.63, $p = .16$) or for the HAM-D posttreatment scores (F [5, 89] = 1.58, $p = .17$), nor was this assumption violated for the SCL-61 pretreatment scores (F [5, 89] = 1.37, $p = .24$) or the SCL-61 posttreatment scores, (F [5, 89] = 1.05, $p = .39$).

ANCOVA Results

Research Question 1. Research Question 1 was as follows: Does optimism/pessimism level affect depression level change after controlling for baseline

depression severity in a group of depressed adults treated with antidepressants or placebo?

A repeated measures ANCOVA was completed to address the first research question. Table 9 displays the results. The within-subjects variable was time, with two levels: pretreatment and posttreatment. There were two between-subjects factors: the treatment group and the Optimist/Pessimist group. Pretreatment MMPI Depression score and sex were used as covariates. Overall, the ANCOVA model was not significant for the within-subjects effect of time ($F[1,87] = 2.29$, $p = .134$). The categorization of Optimist or Pessimist was not a significant factor in the outcome of change in HAM-D score after pretreatment depression, and sex was entered as covariates, as the interaction between time and Optimist/Pessimist group was not significant ($F[1,87] = 1.81$, $p = .18$). The null hypothesis was retained (see Table 8). Overall, after considering the covariates, there was no significant change in HAM-D scores. As shown in Table 9, none of the between-subjects effects were significant.

Table 9

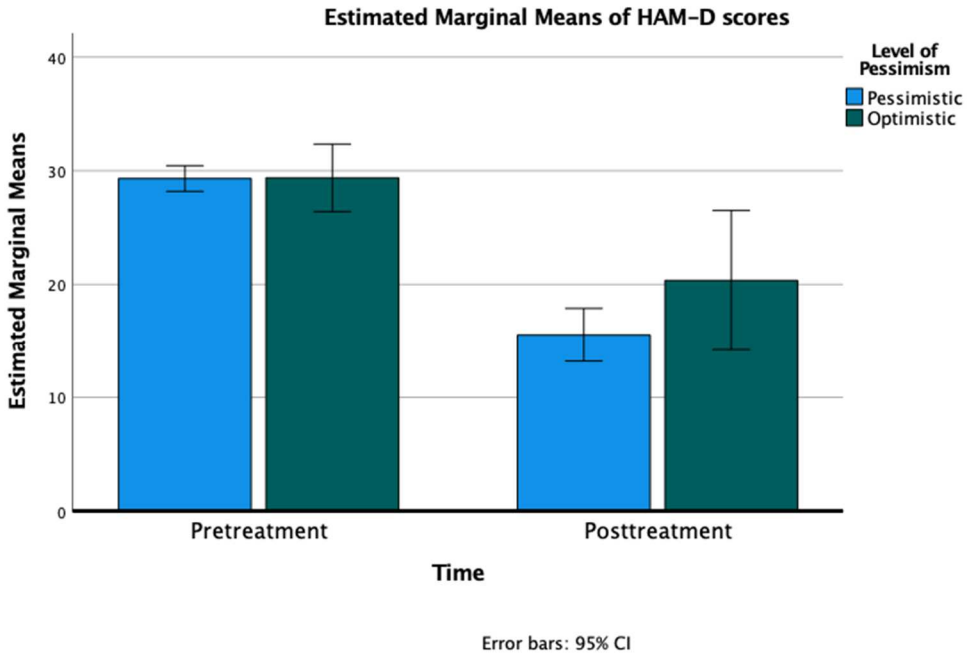
Results of the ANCOVA for the HAM-D Scores

Source	Type III sum of squares	*df*	Mean square	*F*	*p*
Intercept	410.71	1	410.71	6.63	.012
Treatment group	143.05	2	71.53	1.15	.320
Optimist/pessimist group	71.53	1	71.52	1.15	.286
Interaction between treatment group and optimist/pessimist group	.38	2	.19	.003	.997
Error	5393.80	87	62.00		

Although the analyses did not demonstrate a significant effect for time, mean HAM-D scores decreased from pretreatment to posttreatment. Membership in the Optimist or Pessimist group was not related to that decrease. As shown in Figure 2, there was no apparent difference between the Optimist/Pessimist groups in the degree of HAM-D change after treatment.

Figure 2

HAM-D Scores by Optimist/Pessimist Group Membership



Research Question 2. Research Question 2 was: Does optimism-pessimism level affect somatic symptom change after controlling for baseline somatic symptom severity in a group of depressed adults treated with antidepressants or placebo?

A repeated measures ANCOVA was completed to address the second research question. Table 10 displays the results. The within-subjects variable was time, with two levels:

pretreatment and posttreatment. There were two between-subjects factors: the treatment group and the Optimist/Pessimist group. Pretreatment MMPI Hypochondriasis score and sex were used as covariates. Overall, the ANCOVA model was not significant for the effect of time (F[1,87] = 1.28, p = .26). The categorization of Optimist or Pessimist was not a significant factor in the outcome of change in SCL-61 somatic symptom score after pretreatment somatic scores and sex were entered as covariates, as the interaction between time and Optimist/Pessimist was not significant (F[2,87] = 2.35, p = .13). The null hypothesis was retained (see Table 10). Overall, after considering the covariates, there was no significant change in SCL-61 somatic symptom scores. The main effect of the treatment group was significant (see Table 10).

Table 10

Results of the ANCOVA for the SCL-61 Somatic Symptom Scores

Source	Type III sum of squares	*df*	Mean square	*F*	*p*
Intercept	53916.73	1	53916.73	62.38	.001
Treatment group	9827.34	2	4913.67	5.69	.005
Optimist/pessimist group	12.49	1	12.49	.01	.905
Interaction between treatment group and optimist/pessimist group	3083.92	2	1541.96	1.78	.174
Error	75202.026	87	864.39		

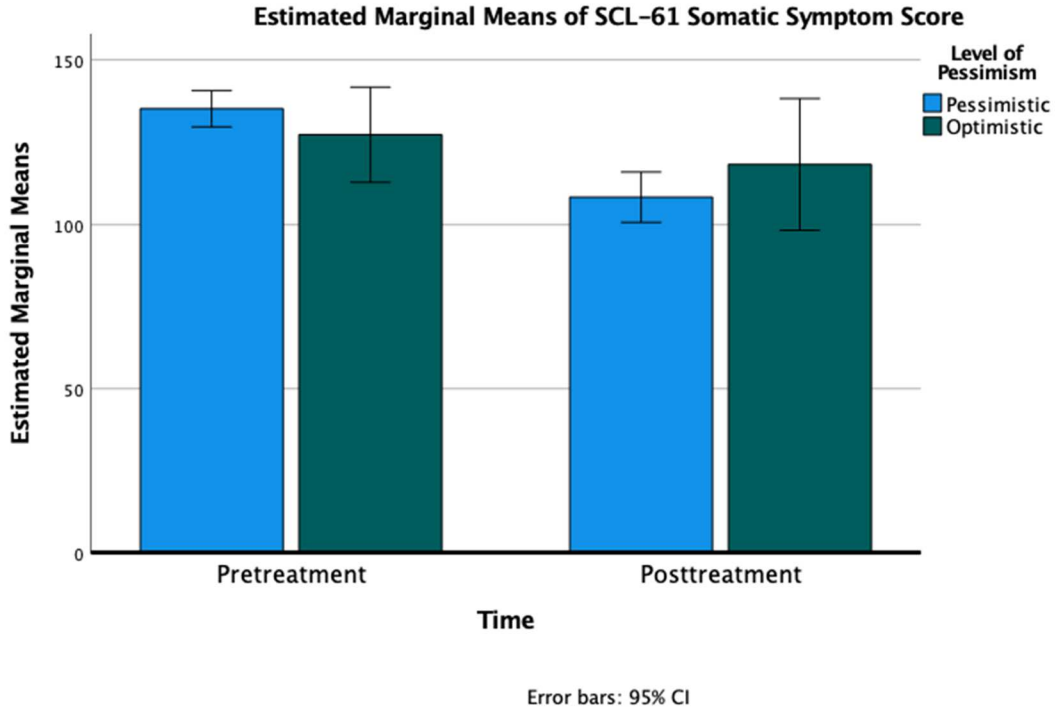
Pairwise comparisons with Bonferroni correction were calculated to determine the treatment group differences. Pairwise comparisons revealed significant differences between the Alprazolam and Placebo groups (p < .004) and the Imipramine and Placebo

groups ($p < .02$). Overall, the Placebo group appeared to fare better with a greater difference in SCL-61 scores at posttreatment compared to the other two groups.

Despite the finding that the pretreatment SCL-61 somatic symptom scores differed between the Optimist and Pessimist groups at pretreatment, there was no difference in posttreatment between the Optimist/Pessimist groups (see Figure 3).

Figure 3

SCL-61 Somatic Symptom Scores by Optimist/Pessimist Group Membership



Summary

The data analysis was described in this chapter. The groups were demographically comparable, with no significant differences by age. However, men were more likely to be categorized as Optimists, and women were more likely to be categorized as Pessimists. There were more Pessimists than Optimists. There were more women than men overall,

and more women were assigned to the Alprazolam and Placebo groups. There was no age difference between the treatment and optimist/pessimist groups.

The results of the first ANCOVA model indicated no significant differences in the treatment group (Alprazolam, Imipramine, and Placebo) and Optimist/Pessimist group regarding the change in depression score. The second ANCOVA model confirmed significant differences in the treatment group with a change in somatic symptom severity between the Alprazolam and Placebo groups and the Imipramine and the Placebo groups. The Placebo group had a greater difference in SCL-61 scores posttreatment compared to the other two groups. However, there was no difference posttreatment between the Optimist/Pessimist groups.

Overall, the ANCOVA model was not significant for the within subjects' effect of time. The categorization of Optimist or Pessimist was not a significant factor in the outcome of change in HAM-D score after pretreatment depression, and sex was entered as covariates, as the interaction between time and Optimist/Pessimist group was not significant. Although the analyses did not demonstrate a significant effect for time, mean HAM-D scores decreased from pretreatment to posttreatment. Membership in the Optimist or Pessimist group was not related to that decrease, and there was no apparent difference between the groups in HAM-D change. Despite the finding that the pretreatment SCL-61 somatic symptom scores differed between the Optimist and Pessimist groups at pretreatment, the Placebo group had a greater difference in SCL-61 scores posttreatment was no difference at posttreatment between the Optimist/Pessimist groups.

Chapter 5: Discussion, Conclusions, and Recommendations

Introduction

Through this study, I aimed to investigate whether a relationship exists between optimism–pessimism and treatment outcomes in individuals clinically diagnosed with depression and treated with either placebo or medication. The study was based on explanatory style theory (Seligman, 1989), which helped create the scales for measuring optimism–pessimism. The study also investigated whether optimism/pessimism changed the reporting of somatic symptoms in depressed participants treated with either a placebo or medication.

The study design was developed to determine whether there were relationships between the change in depression and somatic symptom scores and treatment outcomes of depression and somatic complaints while controlling for pretreatment depression levels and somatic symptom severity. The pessimist or optimist category was determined using PSM scores, while the depression scores were obtained using HAM-D and the somatic symptom scores measured using SCL-61. All the data were secondary and collected via archived data from a double-blind, placebo-controlled experiment. The goal was to determine whether optimism/pessimism impacted treatment outcomes. The significance of this research is that it provides evidence regarding the value of MMPI PSM scales regarding their predictive ability in the outcome of medication- and placebo-based treatment of depression and somatic symptoms.

Interpretation of the Findings

Overall, the study results indicate that levels of optimism or pessimism measured by the PSM scales were not significantly related to treatment outcomes in patients suffering from depression. Neither of the ANCOVA models was significant for the within-subjects effect of time, demonstrating that when the covariates were taken into account, there was no significant change in either the HAM-D or SCL-61 scores between the pretreatment and posttreatment assessments. There was a trend for interaction between time and treatment in the change of SCL-61 scores over time; however, the small sample size may have influenced the power of the analysis to identify significant change. The interaction between the optimist/pessimist group and time was insignificant for the HAM-D comparison, taking sex and pretreatment depression into account as covariates.

Sex needed to be taken into account due to a difference in optimism/pessimism between men and women, with men more likely to be categorized as optimists and women more likely to be categorized as pessimists. This may have resulted from a disparity in the sample itself, with the majority of the sample (71%) being women. Overall, there is no sex difference in optimist/pessimist categorization in the general population (Hinz et al., 2017). However, some researchers have indicated that because women tend to process their emotions more than men, they may be more prone to developing a pessimistic outlook than men (Heinonen et al., 2005).

As discussed in Chapter 2, MMPI PSM scale scores have been related to depression severity (e.g., Burkhart et al., 1980; Gross et al., 2000; Suzuki et al., 2014).

However, this study did not show any association or predictive quality between optimism/pessimism and depression. The PSM has shown predictive ability in depression symptoms in previous research (i.e., Peterson, 2001; Seligman, 2000; Suzuki et al., 2014), but my findings did not replicate the work of previous researchers in that regard. A visual inspection of the HAM-D scores before and after treatment appeared to show an overall decrease in depression after treatment and also appeared to show a greater decrease in depression in the pessimist group compared to the optimist group; however, these differences did not reach the level of statistical significance.

The results also indicate that after accounting for sex and pretreatment somatic symptoms as covariates, optimist/pessimist group assignment was not a significant predictor of SCL-61 somatic symptom score change with treatment. However, somatic symptom score changes significantly differed by treatment group. The post hoc analysis showed that imipramine and placebo groups and alprazolam and placebo differed significantly. The placebo group demonstrated larger decreases in SCL-61 scores than the active treatment groups. This was an interesting and unexpected finding. Researchers have not identified personal characteristics that predict placebo effectiveness in altering perceptions of somatic symptoms (Walters et al., 2019); however, my analysis would indicate that pessimism/optimism did not influence the placebo response in my sample.

Mendels and Schless (1986), who collected the data used in this study, reported that the alprazolam group demonstrated the most improvement in depression scores compared to the other two groups. Although I did not find a significant effect of treatment in my data analysis, I took covariates into account that the study's original

authors did not. I found that imipramine was associated with improved depression slightly more than alprazolam but not significantly so, while placebo was associated with more somatic symptom improvement than either of the active treatment groups. This may have been simply a Type II error, or there may have been some other unaccounted-for characteristics that varied between groups that explain this difference in the outcome.

There were differences between optimist and pessimist groups regarding depression change; however, that difference did not reach statistical significance. The changes in depression score with treatment appeared to be greater in the pessimist group than in the optimist group. Again, it is possible that research with larger sample size, perhaps with data collected prospectively, might identify group differences that I was unable to find in my study.

Kurt et al. (2010), Murberg (2012), and Singh et al. (2016) all found that MMPI PSM scores significantly predicted somatic complaints; however, I only found this association during pretreatment and not posttreatment. In the pretreatment SCL-61, somatic symptoms scores differed between the optimist/pessimist groups, with higher scores in the pessimist group than the optimist group; however, there was no difference between these groups at posttreatment, and the optimist/pessimist group was not a significant predictor of treatment outcome regarding somatic symptoms. The small sample size may have also influenced the power of the ANCOVA to identify interactions between the optimist/pessimist group and time, as SCL-61 scores appeared to decrease in the pessimist group after treatment, although not significantly so.

Seligman's (1989) explanatory style theory, upon which this study was based, indicates that a pessimist holds negative experiences as internal and consistent and positive experiences as external and fleeting (Seligman, 1989). In contrast, an optimist attributes positive experiences as internal and consistent and negative experiences as external and fleeting (Seligman, 1989). Given this difference, it may be hypothesized that the optimist's depression and somatic symptom levels would improve or remain the same with time or treatment and that the pessimist would worsen or remain the same. The findings were not consistent with Seligman's theory. The depression score change in the current research, while not significant, was greater for the pessimist group. This could be consistent with Seligman's definition of pessimism, which indicates that positive experiences are external and fleeting. Perhaps the experience of going through this study was positive, external, and fleeting, causing depression scores to increase, even if only minimal and temporary. Another explanation for this change might be regression to the mean for the pessimist group or simply that the ebb and flow of symptoms were such that depression was worse at pretreatment and progressed to mean or average levels over time, regardless of treatment.

Similar findings were inconsistent with Seligman's (1989) explanatory theory regarding the somatic symptoms scores. During pretreatment, the pessimist group had higher scores, and posttreatment scores appeared to decrease, but not significantly. Again, this could have resulted from a positive external, fleeting experience and perhaps a regression to the mean that the optimist group did not experience similarly. It is possible that a longitudinal study could help examine changes in symptoms over time by

optimist/pessimist group and explore this phenomenon more closely. Seligman's explanatory style theory provides a basis for optimism to be learned. It is also possible that an intervention based on the theory would reveal a more direct association between treatment and optimism/pessimism.

The placebo effect has been a topic of ongoing controversy. Kirsch and Sapirstein (1999) challenged the effectiveness of antidepressant medication when compared to a placebo because, at the time, the research that had been conducted did not demonstrate that antidepressants were significantly more effective than placebo, or rather the authors did not think that the advantage that antidepressants had over placebo were worth the cost of side effects. They opined that until the benefits outweighed the risks, antidepressants should not be used (Kirsch & Sapirstein, 1999). Later, Kirsch et al. (2008) reported that FDA data showed that the effect size of antidepressants was about 0.32 and not the expected 0.50 and used this finding to support not prescribing antidepressants as they were as effective as placebos. Jarrett (2019) noted that in a later analysis of the same data, baseline levels of depression were accounted for, and it was found that antidepressants were much more effective than placebos. Thus, placebo research with the same data has contradicted itself, and findings can depend on the analysis used.

My study appears to provide some limited support for the placebo effect. Although the treatment group did not affect depression score change, the placebo group demonstrated a more significant change in somatic symptom score than the alprazolam and imipramine groups. As my original hypothesis predicted, this change was not

accounted for by optimism or pessimism. There is no clear explanation for this finding; it is inconsistent with the theory used as a framework for the study.

Limitations of the Study

One of the study limitations is that I relied on archival data from a previous study (Mendels & Schless, 1986). I did not collect the data prospectively. Because the current study derives its validity from the previous results, any errors in the previous study, either through data collection or treatment implementation, have been carried forward in the data I used to test my hypotheses. Additionally, my research results may have differed from the original study because I had to eliminate four participants due to missing data not relevant to the Mendels and Schless (1986) research.

The study participants included men and women from Oregon and Washington state. The sample size of 96 was slightly less than the power analysis estimate of 98. This may limit this study's generalizability outside of that general geographic area and other countries. In addition, the sample size, while considerable compared to many psychology studies, was still relatively small.

The HAM-D (Hamilton, 1980) was used to measure depression as an outcome variable, and the SCL-61 (Derogatis et al., 1974) measured somatic symptoms as an outcome variable. While these two scales are established and reliable (Derogatis et al., 1974; Hamilton, 1980), other scales could have been used to measure depression and somatic symptoms. The scales used are still in use today; hence, my study findings are fit for generalization. Because I used archived data from previous research that occurred in the 1980s, the medications used in Mendels and Schless (1986) are not now commonly

used to treat depression. However, this is merely a historical limitation inevitable with technological advances and intellectual progress. Overall, as ascertained from an interview with one of the researchers of the study in question, the data are reliable.

Recommendations

The overall focus of my research was to determine how optimism/pessimism influences treatment outcomes regarding depression and somatic symptoms, and there are a variety of treatment approaches that might be used in addition to antidepressant medications. Further research is needed to investigate the possible association between optimism–pessimism and treatment outcomes for depression and somatic symptoms, as a greater understanding of these associations, could significantly improve treatment programs. Studies with larger sample sizes would provide more generalizable results that would likely have broader impacts on the treatment of depression and somatic symptoms and might clarify some of the findings of my study. Future research might also involve using more current antidepressant medications, such as SSRIs, to explore whether optimism/pessimism is related to treatment outcomes in those medications. Another interesting variable would be psychotherapy techniques in addition to or instead of antidepressant medications to treat depression and somatic symptoms. Optimism/pessimism may make a difference in psychotherapy treatment engagement and, therefore, treatment outcome. Measuring optimism/pessimism before and after treatment may also help determine whether outlook changes with treatment, is associated with treatment outcome, or makes any difference when taken into account with other indicators of treatment success.

Previous research by DeRubeis and Crits-Cristoph (1998) and Seligman (1990) indicated that cognitive therapy could target negative thinking, thereby decreasing pessimism and increasing optimism in clients treated for mental illnesses including major depressive episodes and somatic symptom disorder. Cognitive therapy is based on the concept that how individuals interpret events and situations impacts emotional responses, impacting how they think about and address any situation (Pretzer & Walsh, 2022). Cognitive therapy may increase patients' optimism levels by teaching skills to help individuals perceive and interpret events in a more open-minded, open-ended, and optimistic manner. The depressed mood has been correlated with a pessimistic outlook, although I did not find this to be the case in my research. Cognitive therapy has been proposed to increase optimism toward improving treatment outcomes (Malouff & Schutte, 2016).

In a cohort of criminal justice clients participating in substance abuse treatment, optimism/pessimism levels were significant predictors for subsequent substance use and recidivism (Brown et al., 2004). Parents' education levels correlate with children's optimism (Daraei & Ghaderi, 2012). Optimism correlates with depressive symptoms, indicating a protective measure against depression. In a group of patients suffering from posttraumatic stress, cognitive behavioral therapy increased optimism and openness to experiences, both indicators of positive treatment outcomes (Knaevelsrud et al., 2010). However, there is a paucity of research on how optimism-pessimism impacts psychotherapy outcomes in depressed patients or if successful treatment may be indicated with a change from pessimism to optimism in patients.

Seligman's (1990) research indicates that optimism can be taught. A future study might include evaluating depression and optimism-pessimism levels and then determining whether teaching an optimistic mindset impacts treatment outcomes. A treatment study incorporating optimism training as an independent variable would be able to directly assess the manipulation of optimism-pessimism and its impact on treatment outcomes.

Implications

The findings indicated that optimism-pessimism did not significantly impact the treatment outcomes of depression or somatic symptoms. These findings may be used to create social change because individuals do not need to present specific personality characteristics to find antidepressants beneficial. Mental health practitioners might be able to better treat clients with depression or somatic symptoms using other evidence-based methods, as it appears that medications work regardless of optimism-pessimism. This, in turn, would benefit society, as predicting effective treatment would not require additional testing.

Evidence-based research can help determine what combinations of drugs and cognitive-behavioral therapy or psychotherapy are effective in treating depression. Knowing patients' optimism/pessimism scores may help clinicians better understand their psychological terrain, so to speak, to be better prepared to assist clients in addressing their mental health issues. Whether or not optimism-pessimism itself can be targeted as a way to improve outcomes of the treatment of depression and somatic symptoms is another matter, but one worthy of additional future research. The findings of this study

would suggest not, or at least one may infer, that the degree to which clients are optimistic or pessimistic does not significantly impact medication treatment outcomes. This research indicates that the information resulting from the MMPI PSM scores does not indicate whether one person would benefit more than another from medication treatment. This has social change implications in that the findings indicate that medication alone may be effective in treating depression in pessimistic individuals; one may not need to change one's outlook for treatment to work effectively. Therefore, implementing any prequalification procedures prior to treatment would not be necessary.

Conclusion

This study investigated the influence of optimism–pessimism on the depression and somatic symptom severity of patients being treated for MDD using medication and placebo while taking pretreatment levels of symptoms and sex into account. The study explored the relationship between the variables of archived data from a past study using repeated-measures ANCOVA, which helped remove the covariates' effect and reveal the independent variable's unbiased effect. The results show that optimism–pessimism did not significantly influence the treatment outcome in depression and somatic symptom severity. The results do not provide evidence that could impact the treatment of depression and somatic symptoms in patients with MDD by considering the mediating effect of optimism–pessimism on treatment outcomes.

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