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The Impact of Depakote on Agitation and Short-Term Memory in Nursing Home Dementia Residents

Kristin E. Fazzolari-Pleace
Walden University

Walden University

College of Social and Behavioral Sciences

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Kristin Fazzolari-Pleace

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Review Committee
Dr. Magy Martin, Committee Chairperson, Psychology Faculty
Dr. Steven Little, Committee Member, Psychology Faculty
Dr. Tom Diebold, University Reviewer, Psychology Faculty

Chief Academic Officer
Eric Riedel, Ph.D.

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Abstract

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MS, Walden University, 2012

BS, Hilbert College, 1999

Dissertation Submitted in Partial Fulfillment

of the Requirements for the Degree of

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Abstract

Researchers have linked dementia to common psychiatric symptoms such as agitation and aggression, known as behavioral and psychological symptoms of dementia (BPSD). To treat residents manifesting BPSD, nursing homes (NHs) use psychoactive medications. However, research is limited and inconsistent regarding the impact of Depakote treatment on agitation and short-term memory (STM) in NH residents who have dementia. The purpose of this nonexperimental quantitative study was to evaluate for 1 year the impact of Depakote treatment on agitation and STM in NH residents as measured by each resident's Minimum Data Set (MDS). Moncrieff and Cohen's drug-centered theory served as the theoretical foundation for the study. Archival data from the consulting pharmacist and NH MDS included 16 NH dementia residents. Data were analyzed using a repeated-measures within-subject ANOVA. Results indicated no significant impact of Depakote treatment on agitation and STM scores over a 1-year period. Results may be used to assess the impact and efficacy of a common yet largely unexamined invasive treatment on an underserved, vulnerable population.

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Dedication

This dissertation is dedicated to my dad who passed 6 years ago and was not able to see me through this entire journey. I know he would be very proud of my accomplishments. Also, to my amazing and supportive husband, who has been so patient and supportive through this long journey. This project is also dedicated to my two amazing sons, Dominic and Anthony. They came to adjust to my educational requirements over the years, and I can only hope that I have set a positive example for them of how to achieve your goals even when life and health get in your way.

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I would like to thank Dr. Magy Martin, my committee chair, for her guidance and timely feedback and for stepping up from committee member to chair when it was needed. I would also like to thank Dr. Steven Little for coming on to my committee to guide me with the methodology aspects of my dissertation. Dr. Little went above and beyond his responsibilities to help me when I needed it. Lastly, I would like to acknowledge the support of Walden's Writing Center staff who helped me with insertions of documents and formatting issues when I needed it. Moreover, finally, classmates. Through classroom discussions, I received a great deal of support and helpful suggestions.

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Chapter 1: Introduction to the Study

Dementia, a progressive and irreversible loss of cognitive functioning caused by brain cell damage (Desai, Heaton, & Kelton, 2012), affects approximately 5 to 8 million Americans. More than half of those afflicted have common psychotic symptoms along with aggression, agitation, and cognitive decline (Mittal, Kurup, Williamson, Muralee, & Tampi, 2011). These symptoms collectively are termed behavioral and psychological symptoms of dementia (BPSD). BPSD is the most common reason for nursing home (NH) admission (Meinhold et al., 2005), and psychoactive medications (e.g., Depakote) are the most common treatment for BPSD in the NH setting (Richter, Mann, Meyer, Haastert, & Köpke, 2011). However, there is only limited research on the impact of this treatment. Studies (Desai et al., 2012; Mittal et al., 2011) showed inconsistent results among disparate populations. The current quantitative study addressed the impact of Depakote on agitation and short-term memory (STM) in NH residents with dementia over a 1-year period. Positive social change implications included (a) contribution to the limited extant research on this important subject and (b) assessment of the impact and efficacy of a common yet largely unexamined invasive treatment on an underserved, vulnerable population.

Chapter 1 provides a brief overview of research literature related to the impact of Depakote on agitation and STM in NH residents with dementia and the research gap on this topic. Also, the significance of the research problem to the field of psychology is discussed. The quantitative design and independent/dependent variables of the study are

described. Chapter 1 also includes the research questions and null and alternative hypotheses, including an explanation of how they related to the study approach and research questions. Next, the nature of the study (including design, variables, and methodology) is discussed. Definitions are provided for operative variables and terms. Finally, assumptions, scope, limitations, and significance of the study are discussed.

Background

Dementia is common in the older adult population and is characterized by agitation, aggression, disorientation, cognitive decline, verbal and physical outbursts, and decline in eating, sleeping, walking, and talking (Dharmarajan & Gunturu, 2009). These characteristics of dementia—collectively referred to as BPSD—affect not only the individual with dementia but also his or her loved ones and caregivers. The impact of dementia on the afflicted individual's support system often leads the family to turn to nursing homes (NHs). According to Dutcher et al. (2014), 30-40% of individuals with Alzheimer's Disease (AD) and other forms of dementia in the United States reside in NHs, where psychoactive medications (e.g., Depakote) are the most common treatment (Richter et al., 2011). Depakote is an anticonvulsant medication often used to manage agitation, which occurs in most NH residents with dementia (Meeks & Jeste, 2008). Although there is a vast amount of literature regarding Depakote used for agitation and its effects on cognition, the studies indicated inconsistent results. Dutcher et al. (2014) reported Depakote had no significant effect on slowing cognitive decline; in contrast, Meinhold et al. (2005) reported marginal improvement in STM. There remains a gap in

the literature regarding the impact of Depakote on agitation and STM in NH dementia residents. The current study addressed that gap and helped provide avenues for social change by providing new information on the most common treatment (i.e., Depakote) for a prevalent and exigent societal problem (i.e., BPSD).

Problem Statement

Although the most ostensible feature of dementia is a cognitive decline, behavioral disturbances, also referred to as neuropsychiatric symptoms or BPSD, are often present and cause difficulty in managing NH residents with dementia (Pinheiro, 2008). BPSD, which commonly manifests as agitation, has no FDA approved treatment (Desai et al., 2012). Although various treatment options exist for BPSD in NH residents with dementia, psychoactive medications continue to be the most widely used to manage BPSD despite reports of inconsistent benefits and adverse effects (Rayner, O'Brien, & Schoenbachler, 2006). Psychoactive medications, such as the anticonvulsant Depakote, have been proven to have adverse effects in NH residents with dementia (Dutcher et al., 2014). Dolder, Nealy, and McKinsey (2012) postulated that anticonvulsants such as Depakote are commonly used to manage agitation and aggression because of a lack of alternative treatment options that balance efficacy and safety. Lack of scientific consensus in the relevant literature indicated a need for additional studies. The results of the current study may be used to assess the impact and efficacy of a common yet largely unexamined invasive treatment on an underserved, vulnerable population.

Purpose of the Study

The purpose of this quantitative study, which included a nonexperimental design, was to examine the impact of Depakote on agitation and STM in NH residents with dementia within a 1-year period. The independent variable was Depakote treatment, and the dependent variables were agitation and STM. The focus of this study was to determine to what extent Depakote affects levels of agitation and STM. There was also a variable of interest, effect over time (trials). Boxplots were used to determine whether the length of time the resident was receiving Depakote had any significant effect on agitation and STM scores.

Research Questions and Hypotheses

Research Question 1 (RQ1): To what extent does Depakote treatment affect levels of agitation in NH dementia residents over a 1-year period?

$H_0$1: There is no statistically significant difference in levels of agitation with Depakote treatment over a 1-year period as evidenced by no change in agitation from preexisting data in Section E of the MDS.

H_a1: There is a statistically significant difference in levels of agitation with Depakote treatment over a 1-year period as evidenced by decreased agitation from preexisting data in Section E of the MDS.

Research Question 2 (RQ2): To what extent does Depakote treatment affect STM in NH dementia residents over a 1-year period?

$H_0$2: There is no statistically significant difference in STM with Depakote treatment over a 1-year period as evidenced by no change in STM functioning from preexisting data in Section C of the MDS.

H_a2: There is a statistically significant difference in STM with Depakote treatment over a 1-year period as evidenced by decreased STM functioning from preexisting data in Section C of the MDS.

Theoretical Framework for the Study

Moncrieff and Cohen's (2009) drug-centered theory was the theoretical framework for this study. According to drug-centered theory, psychoactive medications are extrinsic substances that alter how the body works, and the advantages and disadvantages of psychoactive medication use should be carefully evaluated and distinguished from the effects of treatment in general. Drug-centered theory can help identify (a) how psychoactive medications interact with/induce experiences of distress and (b) when the medicated individual should seek psychological help.

Previous theories of psychoactive medication prescription acted to counter neurochemical substrates of disorders or symptoms (Moncrieff, 2008; Moncrieff, Cohen, & Mason, 2009). Neglecting to consider potential psychoactive effects of the psychiatric medications have made it difficult for researchers to establish disease-specific actions and to distinguish whether outcomes occur because of the medication's actions on an underlying pathological process or as a consequence of being in an altered state (Moncrieff & Cohen, 2009). There are many disorders that can mimic psychoactive

effects, such as sedation, cognitive slowing, behaviors, altered sense of perception, sleep, and psychosis. Drug-centered theory indicates that although evidence of the superiority of psychoactive medications might imply disease-specific effects, superior effects can also be explained within a drug-centered framework, which suggests that the characteristic psychomotor and emotional restriction induced by psychoactive medications is more effective at suppressing psychotic agitation than other sedatives.

Moncrieff and Cohen's (2009) drug-centered theory directly related to this study by providing residents or staff members the ability to evaluate and report effects of Depakote treatment in their particular situations. The drug-centered theory has also prompted the psychiatric research community to produce relevant, unbiased information about the range of short- and long-term psychoactive medication effects on cognition, behaviors, and bodily systems. The drug-centered theory is discussed in more detail in the Chapter 2 literature review.

Nature of the Study

This quantitative study addressed to what extent Depakote treatment affects levels of agitation and STM in NH residents with dementia. Also, levels of agitation and STM for Depakote treatment were measured in four intervals over a 1-year period with and without random effects. Residents' levels of agitation and STM were measured using the NH's preexisting quarterly MDS assessments. I used a repeated measures ANOVA design with within-subject effects. A Depakote trial group was measured over four

intervals in a 1-year period, and effect over time (with and without random effects) provided adequate statistical power.

The dependent variables for this study were agitation and STM, and the independent variable was Depakote treatment measured by MDS results and scores. Many NH residents with dementia receive psychoactive medications with questionable benefits. Looking at these variables provided an understanding of how Depakote treatment in dementia patients impacted agitation and STM within the sample.

Variables were analyzed using a repeated-measures within-subject (with and without random effects) ANOVA. The repeated-measures ANOVA was the appropriate design for this study because it allowed for testing one group while subjecting them to repeated measures. This study had one independent variable (Depakote treatment group), two dependent variables (agitation and STM), and significant trials effect (trials/multiple measures over time), followed up with boxplots to determine whether the length of time the resident was receiving Depakote played any significant role in agitation scores and STM scores. A repeated measures design was used because all preexisting data were measured on each variable.

I used archival data from an existing dataset. I did not have direct contact with residents, which obviated potential ethical issues from dealing with a vulnerable, cognitively impaired population. Data were gathered from a consulting pharmacy's database and NH MDS to measure the impact of Depakote on agitation and STM in NH dementia residents. This system of data gathering ensured that data reflected residents

who (a) had been diagnosed with dementia, (b) had exhibited BPSD, and (c) had been receiving Depakote over a 1-year period. MDS is a tool mandated for use in NHs for clinical assessments of all residents covered under Medicare and Medicaid. The MDS provides a comprehensive assessment of each resident's functional capabilities, health-related issues, behaviors, psychological symptoms, and cognitive functioning. The MDS is described in more detail in Chapter 3.

Definitions of Key Terms

Agitation: An increased verbal and/or motor activity as well as restlessness, anxiety, tension, and fear with or without provocation (Zagaria, 2006).

Alzheimer's disease (AD): The most common type of dementia, characterized by cognitive impairment, difficulty communicating, poor judgment, disorientation, confusion, behavior changes, and difficulty speaking, swallowing and walking (Alzheimer's Association, 2017).

Behavioral and psychological symptoms of dementia (BPSD): A heterogeneous range of psychological reactions, psychiatric symptoms, and behaviors, which may be disruptive and unsafe and may impair patient care (Mittal et al., 2011).

Dementia: A general term for memory loss and other mental abilities caused by physical changes in the brain that interfere with daily life (Alzheimer's Association, 2017).

Depakote: An anticonvulsant medication originally developed to treat epilepsy and bipolar disorder and to prevent migraine headaches; it can be given intravenously or orally and has long- and short-acting forms (FDA, 2011).

Psychoactive medications: Drugs or chemical substances that act primarily on the central nervous system and alter brain function, resulting in temporary changes in behavior, mood, perception, and consciousness (Brandt & Pyhtila, 2013).

Short-term memory (STM): The type of memory used to retain information for a short time; it has a working memory component that is used to manipulate information in consciousness (Engle, Laughlin, Tuholski, & Conway, 1999).

Assumptions

I assumed that all residents were NH residents of one of the two local NHs from which data were obtained. This assumption was accounted for by obtaining preexisting data directly from the two NHs. I also assumed that all residents had a diagnosis of dementia. The preexisting data from the pharmacy consultant and the two NHs verified residents' diagnosis of dementia. Next, I assumed that the residents were receiving Depakote for BPSD, which was also accounted for by viewing the preexisting data from the pharmacy consultant. This dataset included the reason why the residents were receiving Depakote. I assumed that assessment tools used in this study were appropriate for the identified sample of NH residents with dementia on Depakote treatment for agitation. I assumed all data gathered were preexisting data from NH MDS and pharmacy records and were as accurate as possible, entailing a further assumption that NH staff

members completing the MDS had the clinical knowledge/competency to do so. I assumed that anonymity and confidentiality were preserved by using preexisting data and that use of these data obviated potential ethical concerns.

Scope and Delimitations

I examined the impact of Depakote on agitation and STM in NH dementia residents over a 1-year period. This area was chosen for this study because prior research indicated adverse effects of psychoactive medications but revealed a gap on the impact of Depakote on agitation and STM in NH dementia residents over a 1-year period. The sample was chosen to represent NH residents receiving Depakote for BPSD. This study did not include unmeasured variables, such as gender, race, and age, because of the smaller sample size. Residents receiving Depakote for seizure disorder were excluded. Prior research indicated adverse effects of Depakote, but this study looked specifically at the impact of Depakote on agitation and STM in NH dementia residents within a 1-year period. The primary issues of validity were anticipating that NH social workers who completed preexisting MDSs provided correct information regarding each resident's behavior and cognition.

Limitations

Limitations of this study pertained to potential problems with the study design. For example, the NHs where the data were gathered represented only the United States and may not represent the entire NH population. Data were drawn from a specific population, and the sample size was limited due to the study requirements of (a) residents

had been on Depakote treatment for one year and (b) Depakote having been prescribed specifically for BPSD. This study did not include every race, age bracket, geographic background, or socioeconomic status represented in the NHs. Threats to external validity related to repeated measures because it was difficult to control for the effects of prior outcomes using the same subject.

Another factor that contributed to limitations in this study was the lack of primary data (i.e., depending exclusively on accurate MDS assessments). Data in MDS and pharmacy records may not have been meticulously and accurately collected. However, because of the vulnerable nature of the target population, it was best to use preexisting data to avoid ethical complications when gathering the data. Clear directions were given to the NH and pharmacy contacts regarding the specific sections needed from MDS records and pharmacy records (i.e., only residents who had been on Depakote for BPSD for at least one year, with seizures excluded). Nevertheless, there were factors that could have jeopardized internal validity (e.g., if the scorer of the MDS changed, there may have been outcome changes). Precautions were taken to maintain the confidentiality of the data provided, and NH administrators and pharmacy representatives were informed that the study would be confidential.

Significance of the Study

Over the last century, there has been a dramatic increase in psychoactive medication use due to the increase in the geriatric population, and patient pharmacokinetic and pharmacodynamic responses to these medications differ

considerably (Hilmer, McLachlan, & Le Couteur, 2007). There is limited evidence regarding the complexities of geriatric pharmacology, and it is underappreciated in clinical trials.

According to Spira and Edelstein (2007), agitation is exhibited by 55% to 90% of individuals with dementia with various approaches and treatments attempted. Psychoactive medications are prescribed to up to a third of older adults and are the most widely used medications in NHs to treat agitation, Depakote being one of them (Huybrechts et al., 2012). Understanding potential connections between agitation and STM with Depakote treatment may contribute to positive social change by raising awareness regarding concerns of the NH dementia population that may have been overlooked. It is important to understand the theoretical concepts behind psychoactive medication use, agitation, and memory in those with dementia. It is essential to treat these symptoms in this population, but research indicated that there had not been any therapeutic solutions and Depakote continues to be used for these purposes. This study addressed that gap by examining the impact of Depakote on agitation and STM in NH dementia residents over a 1-year period. Findings from this study may provide insight on the impact of Depakote used to treat agitation and its adverse effects about agitation and STM. Findings may provide updated information related to NH use of Depakote for managing BPSD in an underserved, vulnerable population.

Summary

Chapter 1 provided a brief overview of existing literature on the study topic, including a gap in the literature regarding the impact of Depakote treatment on agitation and STM. I gave a brief description of drug-centered theory as the theoretical framework for this study. The NH is a unique environment that requires workers to be trained to deal with dementia and BPSD. BPSD is difficult to manage in NH dementia residents, and therefore psychoactive medications are often used (Meeks & Jeste, 2008). Studies showed Depakote treatment as inconsistent, with positive and negative effects. Despite conflicting findings and safety warnings concerning the use of psychoactive medications in NH residents, Depakote treatment has remained common because of longer resident lifespans and an increased number of people with dementia (Huybrechts et al., 2011). This study contributed to the body of literature by addressing the impact of Depakote treatment on agitation and STM in NH dementia residents over a 1-year period. Implications for social change include assessing the impact and efficacy of a common yet largely unexamined invasive treatment on an underserved, vulnerable population. Chapter 2 provides a more comprehensive overview of drug-centered theory, which provided the theoretical framework for studying the impact of Depakote on agitation and STM in NH dementia residents within a 1-year period. Chapter 2 also addresses inconsistencies in the extant literature concerning the impact of Depakote on agitation and STM.

Chapter 2: Literature Review

A review of the literature produced a limited number of studies on the impact of Depakote on agitation and STM in NH dementia residents. The purpose of this nonexperimental quantitative study was to go beyond the boundaries of the current literature to examine the impact of Depakote treatment on agitation and STM in NH dementia residents over a 1-year period. The focus was on the extent to which Depakote affects levels of agitation and STM. The Depakote treatment group was the independent variable; agitation and STM were the dependent variables. I employed a within-subject design (trials/effects over time), including repeated measures for all variables. Additionally, I employed boxplots to determine whether the length of time resident was receiving Depakote had any significant effect on agitation scores and STM scores.

Depakote is an anticonvulsant medication used to manage agitation in approximately 80% of NH residents with dementia (Meeks & Jeste, 2008). Medicare reports show dementia affects approximately 5 to 8 million Americans, of which more than half have BPSD (Mittal et al., 2011). According to Dutcher et al. (2014), 30-40% of individuals with AD and other forms of dementia in the United States reside in NHs. BPSD is a major reason for increased agitation and cognitive decline, for which there is no FDA approved treatment (Desai et al., 2012).

The following literature review includes the most current research relating to NH residents with a diagnosis of dementia who are prescribed Depakote for BPSD. The collective body of literature informed this study of the impact of Depakote on NH

dementia resident behaviors, psychological symptoms, and cognition. This chapter begins with a description of the literature search strategy. The next section contains a review of literature on Moncrieff and Cohen's (2009) drug-centered theory and is followed by a review of studies on psychoactive medication use in NH dementia residents, Depakote use in NH dementia residents, adverse effects of Depakote use in NH dementia residents, benefits of Depakote use in NH dementia residents, BPSD and Depakote use in NH dementia residents, Depakote and agitation in NH dementia residents, Depakote and cognitive functioning in NH dementia residents, and Depakote and STM in NH dementia residents. Next is a review of studies that addressed Depakote use to manage agitation in NH dementia residents, a review that exposes inconsistencies in findings and effects of Depakote use to manage agitation in NH dementia residents. Some of the studies cited in this literature review involved the use of MDS to obtain measures of behavior and/or cognitive functioning (i.e., the same database that was used in the current study). Finally, the study variables are discussed (i.e., Depakote use, agitation, and STM), and an explanation for the impact of Depakote on agitation and STM in NH dementia residents is provided. The chapter concludes with a rationale for the choice of research method, a summary of the literature review, and suggestions for additional research.

Literature Search Strategy

The databases used to discover the literature were accessed through the Walden University Library; the databases included PsychINFO, SAGE Premier, Academic Search Complete, PsychARTICLES, ProQuest Central, MEDLINE, and BIOMED

Central. I also used the Google Scholar search engine. The following keywords, terms, and phrases were used: *dementia*, *AD*, *agitation*, *BPSD*, *STM*, *cognition*, *Depakote*, *NH residents*, *psychoactive medications*, *Depakote and agitation and dementia*, *Depakote and BPSD*, *Depakote and agitation and NH residents*, *Depakote and NH*, *effects and Depakote and agitation*, *Depakote and cognition*, *Depakote and STM and dementia*, *Depakote and AD*, and *psychoactive medications and NH*. Most of the literature was found using SAGE Premier and Google Scholar, and the search using the phrase *Depakote and BPSD* yielded the most literature. Initial searches were performed in October 2015, and additional searches were conducted for new references in May 2016, July 2016, and January 2017.

Theoretical Foundation: Drug-Centered Theory

The theoretical framework for this study was Moncrieff and Cohen's (2009) drug-centered theory, which states that because psychoactive medications are extrinsic substances altering how the body works, advantages and disadvantages of psychoactive medication use should be weighed and distinguished from the effects of treatment in general. Drug-centered theory can inform perspectives about how psychological alterations produced by psychoactive medications interact with experiences of distress and the need to seek psychological help. Many disorders can mimic psychoactive effects, such as sedation, cognitive slowing, behaviors, altered sense of perception, sleep, and psychosis.

Moncrieff and Cohen (2009) theorized that although evidence of the superiority of psychoactive medications might imply disease-specific effects, superior effects can also be explained within a drug-centered framework (e.g., the psychomotor and emotional restriction characteristic of psychoactive medications suppresses psychotic agitation more effectively than other sedatives). The drug-centered theory provides a useful lens through which NH resident clinicians or staff members can evaluate and report the effects of Depakote treatment in NH dementia residents. The drug-centered framework further prompts members of the psychiatric research community to produce relevant, unbiased information about the short- and long-term effects of psychoactive medications on cognition, behaviors, and bodily systems.

Literature Review Related to Key Variables

Overview of Psychoactive Medication Administration in Treating BPSD in NH Dementia Residents

Throughout much of the literature reviewed, dementia-associated psychotic symptoms have been labeled BPSD, which represents the most difficult dementia sequelae for NHs to manage. BPSD is often discussed in the literature as a heterogeneous range of psychological reactions, psychiatric symptoms, and behaviors, which can be disruptive and unsafe and can impair the care of the resident in a given environment; moreover, the likelihood and intensity of BPSD increase as dementia progresses (Mittal et al., 2011).

Meeks and Jeste (2008) conducted a meta-analysis of 15 randomized controlled trials involving psychoactive medications including risperidone, olanzapine, aripiprazole, and quetiapine used to treat agitation or psychosis in NH dementia residents. Most residents were female NH residents with AD. Meeks and Jeste found that psychosis improved with risperidone, and neuropsychiatric disturbances improved with risperidone and aripiprazole. Meeks and Jeste also noted that effects were more noticeable in residents without psychosis, NH residents, and residents with severe cognitive impairment. In contrast, subsequent placebo-controlled trials of risperidone, quetiapine, and aripiprazole in AD residents revealed that atypical and typical antipsychotics are effective in reducing aggression and psychosis (Meeks & Jeste, 2008). Although these findings suggested that psychoactive medications are effective in treating BPSD, the results were questioned by the National Institute of Mental Health (NIMH) Clinical Antipsychotic Trial of Intervention Effectiveness Study for Alzheimer's Disease (Lieberman, 2006). This NIH-sponsored study indicated that risperidone and olanzapine but not quetiapine were effective in that fewer residents taking them versus placebo dropped out due to lack of efficacy. The year-long study indicated that antipsychotics were not effective overall because of the primary outcome, all-cause discontinuation rate, was similar for all three drugs and placebo. The result suggested a muddled, inconclusive picture regarding the efficacy of psychoactive medication administration for treating BPSD in NH residents with dementia; this inconclusiveness created the need for further

examination of a prevalent treatment program for a pressing societal problem, a need addressed by this study.

Risks of Psychoactive Medication Use in NH Dementia Residents

There are no drugs that have been FDA approved to treat BPSD in dementia residents, but psychoactive medications are often used to alleviate these symptoms (Meeks & Jeste, 2008). One of the most concerning issues regarding NH administration of psychoactive medications is they present a safety hazard and are linked to a variety of negative outcomes (Molinari et al., 2013). In the NH setting, negative outcomes include cerebrovascular effects, heart failure, and sudden death (Schneider, Dagerman, & Insel, 2006). According to Meeks and Jeste (2008), the likelihood that NH residents are on a variety of medications for various health issues further complicates the administration of psychoactive medications, thereby increasing the chances for adverse drug reactions and potentially increasing psychosis and agitation.

BPSD Versus Other MH Symptoms

There are many side effects from psychoactive medication use in NH dementia residents. For example, NH residents with dementia face greater risk of hospitalization, falls, cognitive impairment, and mortality (Ballard et al., 2009, 2011; Belleville, 2010; Cooper, Freeman, Cook, & Burfield, 2007; Dragonich, Zancy, Klafta, & Karrison, 2001; Frey, Ortega, Wiseman, Farley, & Wright, 2011). BPSD represents just one of the prevailing mental health (MH) concerns among the NH population. According to Molinari et al. (2013), NH dementia residents with behavioral issues are often

categorized as having BPSD rather than being carefully evaluated for other MH issues. Using the benchmarks of decreased numbers of falls and hospitalizations, Molinari et al. addressed the question of whether providing an MH assessment to all NH residents upon admission would decrease the use of psychoactive medications. The study was conducted at four for-profit NHs and included 23 residents who were cognitively able to provide valid responses. These results were compared to a group of 23 NH residents who did not receive MH assessments. Using a chi-square analysis, Molinari et al. found at a 1-month follow-up comparing the assessment and nonassessment groups on various measures of psychopharmacological and nonpsychopharmacological interventions that the residents who received brief MH assessments were less likely to start on psychoactive medications despite the fact that a high number of residents were admitted to the facility with orders for psychoactive medications. Molinari et al. also revealed that despite favorable comparisons with the nonassessment comparison group, psychoactive medication use was still high in the assessment group, and the intervention was not helpful in getting residents off psychoactive medications.

In other words, Molinari et al. (2013) found that consideration of alternative ways of addressing residents' MH needs decreased the likelihood of starting residents on psychoactive medications. Also, Molinari et al. found that residents who received the MH assessment were also more likely to receive subsequent MH consultation. This finding is significant in that it shows a positive correlation between consideration of alternative MH treatments and the need for a psychoactive medication regimen. This approach may be

used to minimize previously demonstrated negative impacts of psychoactive medication administration on NH residents with dementia. However, the study would have benefitted from larger sample size and more diverse demographics, which would have increased study power and validity. The sample was disproportionately non-Hispanic White and comprised short-stay residents.

Psychoactive Medications on Mental, Behavioral, and Physical Health

Richter et al. (2011) found that psychoactive medications induce a distinctive alteration in mental and physical states when used to treat BPSD in NH residents with dementia. Moncrieff, Cohen, and Porter (2013) addressed how the effects of psychoactive medications might modify various psychiatric symptoms. For example, psychoactive effects can directly modify mental and behavioral symptoms and affect the results of placebo-controlled trials. Although the term *psychoactive* is related to mental alterations, mental conditions are connected to physical conditions, causing a global effect. Moncrieff et al. concluded that despite much of the research in neuropharmacology, more extensive research is needed to clarify the long-term mental, behavioral, and physical effects of psychoactive medications. Such research would assist in diagnosis and treatment and enable further discussion of the purpose and ethics of the frequent use of psychoactive medications to manage behaviors in older adults.

Prevalence of Psychoactive Medication Use in NH Dementia Residents

Much of the literature addressed the challenges for NHs in managing BPSD as well as the distress that BPSD causes residents, leading to excessive use of psychoactive

medications. Previous research indicated psychoactive medications are the most widely used medications among NH residents and that 50% to 80% of residents are on at least one psychoactive medication (Meinhold et al., 2005; Richter et al., 2011). Richter et al. (2011) looked at the comparison of four different variables (i.e., psychoactive medication, different classes of psychoactive medication, psychoactive medication administered for bedtime only, and associations between prescription of psychoactive medications and institutional and resident characteristics). Richter et al. conducted a cross-sectional comparison of data from three large studies of 5,336 NH residents from 136 long-term care facilities. Richter et al. found that in all three comparison studies, 74.6%, 51.8%, and 52.4% of all residents were on at least one psychoactive medication. Another comparison indicated that 66% and 47% of residents were prescribed for bedtime use only. None of the three studies indicated a statistically significant association between psychoactive medication prescription and NH characteristics; however, consistent positive associations were found for a higher level of care dependency and permanent restlessness, and consistent negative associations were found for older age and male gender. In other words, residents requiring more care and presence of permanent restlessness had a higher rate of psychoactive medications prescribed. These findings provide evidence that psychoactive medications are highly prevalent in managing BPSD in NH residents. The researchers were not able, however, to show a correlation between NH characteristics and psychoactive medication prescription rates. In contrast, despite the important role of nursing staff, there was no impact on psychoactive medication

prescriptions on a nurse staffing level. Specifically, it was found that psychoactive medications are the first course of treatment for BPSD and that there is a need for effective programs to reduce prescription of psychoactive medications in NH residents.

Impact of Depakote on Agitation in NH Dementia Residents

Agitation affects up to 70% of older adults with dementia, and Depakote derivatives have been used for more than ten years to control agitation in dementia residents in NHs (Narayana, Clifton, Luxenberg, & Curran, 2014). The Narayana et al. 2014 review examined whether the evidence supports the use of Depakote in the treatment of agitation in dementia residents. The review concluded that Depakote does not improve agitation in dementia but increases the frequency of side effects. Within the body of literature that has focused on using Depakote for agitation in NH dementia residents, there is limited research discussing the impact of Depakote on agitation and STM in NH dementia residents.

Advantages of Depakote Use in NH Dementia Residents

BPSD is known to occur in up to 90% of individuals with dementia at some point in their disease progression (Meinhold et al., 2005). There are many different manifestations of dementia, including cognitive decline, alterations in mood/thought/behavior, and inability to conduct activities of daily living (ADLs). As in the Richter et al. study, Meinhold et al. (a) described BPSD as depression, hallucinations, delusions, agitation, aggression, combativeness, disinhibition, and hyperactivity and (b) found that pharmacological measures are often used to control agitation and aggression in

patients with dementia. The impact or benefits of psychoactive medications are not exclusive to one specific medication; thus, the anticonvulsant agent Depakote may have some advantages when used for agitation and aggression in dementia residents due to its lower rates of drug interactions and adverse effects in the dementia population. The authors addressed the impact of Depakote on behavioral, mood, and cognitive measures in NH dementia residents who had a history of behavior problems associated with dementia. The researchers utilized pharmacy databases and MDS assessments to obtain data for residents that were receiving Depakote for behavior problems related to dementia. Some exclusions were applied, such as residents that were receiving Depakote for seizures (i.e., the indication for which Depakote is FDA-approved). A total of 450 residents were identified with behavior problems related to dementia and receiving Depakote, and MDS assessments indicated that Depakote reduced the frequency of negative behaviors; expressions of verbal distress; indicators of sad, apathetic and anxious appearance; and sleep cycle problems (Meinhold et al., 2005). In sum, the authors found the use of Depakote for agitation and aggression in dementia residents was effective, safe, and tolerable. They concluded Depakote might have multiple beneficial effects in NH dementia residents with a history of dementia with behavioral problems. Results further suggested that if Depakote were used as a secondary agent and started earlier in treatment, it may have more beneficial effects.

Disadvantages of Depakote Use in NH Dementia Residents

The aforementioned results are inconsistent with the results from Narayana et al. (2014), who concluded that Depakote is not beneficial for BPSD management in that Depakote does not improve BPSD symptoms and increases the frequency of side effects. Also, Tariot et al. (2005) compared Depakote and placebo treatments in residents with AD. The authors performed three randomized, double-blind, placebo-controlled clinical trials in 153 NH residents with AD complicated by agitation, and out of those 153 residents, 78 were assigned placebo and 75 Depakote. The clinical trial was six weeks in length, and Depakote dosages were increased every 3 days until the target dose was reached. The authors reported that Depakote did not show benefit over placebo in alleviating AD-associated agitation in NH residents. Primary and secondary measures of behavior, using the Last Observation Carried Forward (LOCF) and Completer approaches, showed consistent results. The authors further reported none of the earlier placebo-controlled studies had proved Depakote was effective for agitation in NH dementia residents. The Tariot et al. (2005) study was the largest to prospectively address agitation as the primary outcome. Previous and subsequent literature regarding the use of Depakote for managing agitation in NH dementia residents have reported similar findings. Herrman et al. (2007) reached findings along the same lines, reporting Depakote is ineffective for managing agitation in NH residents with AD and may be poorly tolerated in this population. The authors of the 2007 study discussed similar previous randomized controlled trials that also showed no statistically significant benefits of

Depakote on primary outcome measures, though it showed efficacy for some secondary measures. The major limitations of this study were the small sample size and crossover design, but despite these limitations, significant worsening of symptoms during Depakote treatment was found.

Impact of Depakote Doses and Levels in NH Dementia Residents

Herrman et al. (2007) found that lower Depakote doses and slower titration schedules could improve tolerability (i.e., longer Depakote treatment phases could prove beneficial in managing agitation in NH residents with AD). Similarly, Tariot et al. (2005) used Depakote doses and levels similar to those in previous trials (doses are typically lower in AD residents versus in those with seizure disorders or mania in younger populations). The authors found it highly unlikely that higher doses would have been tolerated well enough to justify their use, and they could not conclude that a longer treatment period would have been more effective.

Tolerability and Effectiveness of Depakote in NH Dementia Residents

Mizukami et al. (2010) addressed the negative impacts of BPSD and how they are often managed using psychoactive medications, despite the mortality rate. The authors emphasized the urgent need for safer BPSD treatment in dementia patients. The aim of their study, similar to Tariot et al. (2005) and Herrman et al. (2007), who reported that Depakote was ineffective for managing agitation in NH dementia residents, was to examine the efficacy and tolerability of Depakote in patients with BPSD. The study consisted of 110 dementia patients treated with Depakote for behavioral disturbances

(excluding those with reversible causes of dementia or with a diagnosis other than dementia [e.g., delusional disorder]). The authors reported that unsteady gait, sleepiness, nausea, dizziness, and headache were experienced by 13 of the 110 residents, though serum tests showed no abnormal findings. They also found that out of the 110 patients, ten were very much improved, 42 much improved, 34 minimally improved, 21 had no change, and three worsened (i.e., the very much or much improvement was observed in 52 out of the 110 patients). They observed some adverse effects such as hallucinations/delusions (6/54), irritability/excitement (70/85), aggression/agitation (29/43), insomnia/delirium (35/58), inappropriate/purposeless behaviors (11/40), and other symptoms not specified (25/46). In contrast to Tariot et al. and Herrman et al., this study revealed Depakote was effective in 47.3% of the patients with BPSD without experiencing serious adverse effects. The researchers also found Depakote was effective in managing irritability, aggression, and agitation while there was no effect on hallucinations/delusions and inappropriate behaviors. The authors cited results from previous studies that reported the effectiveness of Depakote against agitation and aggression in dementia patients. In contrast, results from randomized controlled trials have been inconsistent, indicating a need for further research into the impact of Depakote on agitation and aggression. Porsteinsson (2006) also addressed the use of Depakote as an intervention for BPSD, reporting results from four placebo-controlled trials and concluding that none of the studies was sufficient to define clinical practice due to conflicting, inconclusive results. The 2006 study reported that three of the four studies

had suggested possible short-term efficacy, tolerability, and safety of Depakote for agitation and some other neuropsychiatric symptoms associated with dementia patients; in the fourth study, there were no demonstrated benefits of Depakote over placebo. The author concluded further research is needed to determine optimal use of Depakote for the treatment of BPSD and to see if there are any long-term benefits in using Depakote to manage BPSD.

Dolder et al. (2012) reported that the use of anticonvulsants (of which Depakote is the most commonly prescribed) had yielded inconsistent results in residents with dementia. The authors reported that Depakote had been believed to produce symptomatic improvements in dementia due to its actions on GABA and N-methyl-$_{D}$-aspartate (NMDA) receptors. They also reported that in vitro and in vivo studies have demonstrated Depakote may have neuroprotective effects on AD due to numerous potential mechanisms such as prevention of beta-amyloid aggregation, decreased beta-amyloid and neuritic plaque production, and induction of neurogenesis. That is, Depakote can have positive effects on certain brain functions and chemical reactions in AD residents. In contrast, the researchers mentioned that Depakote is not usually recommended in evidenced-based treatment guidelines and position statements for dementia residents. The authors further discussed conflicting findings over the years regarding the use of Depakote in dementia residents for agitation, reporting that older reviews were based primarily on open-label and retrospective studies and usually ended with positive statements regarding the use of Depakote in dementia residents. In contrast,

they reported more recent reviews that indicated controlled trials did not primarily end with positive conclusions. As in the Tariot et al. study, residents were institutionalized and had a diagnosis of dementia, and all residents are receiving Depakote for a seizure disorder, other medical related conditions, and MH diagnoses, were excluded from the study. Also, residents that were admitted to NHs with other medical causes (e.g., delirium, medication withdrawal, intoxication) for their behavior disturbances were excluded. The authors gathered the resident's demographic, diagnostic, medication, and drug-level data from resident charts. The objective of their study was to describe the dosages of Depakote among residents admitted to a geriatric ward for dementia-related behaviors. The researchers took several subjects into consideration, such as Depakote dosages before admission (if applicable) and at discharge, Depakote serum level about discharge dose, the weight of the resident, and if the resident was on any antipsychotics or benzodiazepines. Twenty studies were included in the Dolder et al. review: 18 of the studies examined the effects of Depakote on residents with psychosis or behaviors in dementia; another study examined the effects of Depakote to prevent behaviors, and another trial investigated the tolerability of Depakote. The average age of residents was 80, with a diagnosis of AD, and agitation and aggression were the most common indications for Depakote use. The authors concluded Depakote might be beneficial in some residents with dementia-associated agitation based primarily on lower doses of Depakote being associated with symptomatic improvement. In contrast, they reported the same range of Depakote serum levels had not shown significant behavioral improvement,

leaving important questions unanswered. In sum, the researchers reported Depakote does not seem to be beneficial in preventing behavioral symptoms and, also, can produce problematic side effects in some residents with dementia.

Gareri et al. (2009) examined Depakote-induced delirium in an AD patient with moderate cognitive impairment associated with behavioral disorders, including aggression, agitation, and severe insomnia. They reported that, after an initial benefit, the patient suddenly developed hyperactive delirium, including worsening of insomnia and agitation, severe confusion, delusions, and visual hallucinations alternated to sedation. The researchers reported that Depakote was immediately stopped and symptoms continued for approximately a week, while another medication was introduced and was successful after three more days. This 2009 case study addressed (a) the possible negative effects of prescribing Depakote to AD patients to manage agitation and aggression and (b) the importance of minimizing the use of and titrating psychoactive medications in dementia patients.

Depakote on Delaying or Preventing the Start of Symptoms and Slowing Cognitive Decline

Tariot et al. (2011) attempted to determine whether Depakote treatment could delay or prevent the start of symptoms of agitation or psychosis. The study consisted of 313 individuals with moderate AD who had not yet experienced agitation or psychosis. The researchers utilized a multicenter, randomized, double-blind, placebo-controlled trial with flexible doses of Depakote. The study was conducted over 24 months, followed by a

2-month period of single-blind placebo treatment. The authors reported a total of 122 residents (59 receiving Depakote and 63 receiving placebo) completed 24 months of treatment; 42 (27 receiving Depakote and 15 receiving placebo) reached 24 months having discontinued the medication; 150 reached month 26. They found that there was no difference between groups in the manifestation of agitation or psychosis and that there was no difference in groups in change on any secondary outcome. Results revealed the Depakote group had higher rates of somnolence, gait disturbance, tremor, diarrhea, and weakness; and 88% of the residents that underwent magnetic resonance imaging (MRI) showed greater loss in hippocampal and whole-brain volume, accompanied by greater ventricular expansion (Tariot et al., 2011). In conclusion, the authors found Depakote that treatment did not delay the start of agitation or psychosis or slow cognitive or functional decline in patients with moderate AD, and the medication was associated with significant toxic effects.

Impact of Depakote on STM in NH Dementia Residents

When searching the databases listed at the beginning of this chapter, there was little to be found on the impact of Depakote on STM, identifying a significant gap in the literature. STM is described as the system that temporarily stores and manages information that is necessary to complete complex cognitive tasks (Ai-Guo et al., 2015). Richter et al., (2015) discussed the effects of person-centered care on psychoactive drug use in NHs. The authors discussed the high rates of psychoactive medication use within NHs: they reported recent data showing that 30% of German NH residents were on

psychoactive medications, many for inappropriate reasons. One of the adverse effects they mentioned is diminished cognitive function. Though they mentioned worsening of cognitive functioning, their research did not specifically touch on the impact of Depakote on STM.

Impact of Psychoactive Medication on Cognitive Function in NH AD Residents

Dharmarajan and Gunturu (2009) discussed the high rates of AD in NH residents and the possible causes. They described AD as a common, acquired disorder that is manifested as slow, progressive memory loss with at least one cognitive dysfunction (aphasia, apraxia, agnosia, or executive dysfunction), resulting in impaired occupational and social performance. The researchers mentioned the deterioration in cognition from earlier levels must occur in the absence of delirium or other causes of dementia (e.g., Parkinson's disease or vascular dementia). The researchers discussed (a) the use of psychoactive medications, including Depakote, to manage agitation and difficult behaviors in NH dementia residents and (b) the impact of the medication on cognitive function. Similar to the Richter study, the authors of the 2009 study touched lightly on adverse effects and cognitive functioning but not specifically on STM.

Case Study on the Negative Impact of Depakote

Manckoundia et al. (2008) did a case study of a 68-year-old woman who developed dementia symptoms after starting Depakote for seizures. Before starting Depakote, the woman did not have any neurological complications, but after several months of Depakote use, she presented with a decline in cognitive function and

withdrawal from social activities. The authors reported that her psychological assessment revealed memory difficulties, predominantly STM with impairment of recall memory, disorientation to time, and difficulty finding words and writing. Her mini-mental (MMSE) score was 15, and discrete lesions were evident on an MRI. The researchers believed Depakote was the possible cause of the woman's psychomotor slowing. After Depakote was discontinued, the woman experienced rapid clinical and objective improvement, and three weeks after stopping Depakote, her psychomotor slowing had resolved; her speech was normal; her MMSE score was 25, and she started engaging in social activities again. This case study indicated that Depakote had a negative impact on short-term memory and when discontinued, STM returned to baseline.

Impact of Mood Stabilizers on Cognitive Functioning

Dutcher et al. (2014) researched exposure to medications (such as Depakote) commonly used in NH dementia residents. The authors reported that slower cognitive decline was associated with antidepressants and antipsychotics, but poorer outcomes were observed with mood stabilizers, such as Depakote. The study was a 2-year longitudinal investigation of 18,950 NH residents who were newly diagnosed with dementia and resided in the NH for at least part of the two-year study period. The authors obtained their data from the Chronic Conditions Data Warehouse (CCW) and nursing home MDS records. Their study included residents' exposure to four classes of medications: antidementia medications (ADMs), antipsychotics, antidepressants, and mood stabilizers. Outcomes included the performance of ADLs and cognition using

Cognitive Performance Scale (CPS). Study results indicated ADM was not associated with a change in ADLs over time but was associated with a slower CPS decline; antidepressant use was associated with slower declines in ADLs and CPS. They reported that sex modified the effect of both antipsychotic and mood stabilizer use on ADLs; female users declined more quickly than males. They also reported that antipsychotic use was associated with slower CPS decline, whereas mood stabilizer use had no effect (i.e., Depakote did not have any effect on slower CPS decline). The authors reported that despite the statistically significantly slower declines in cognition with ADMs, antidepressants, and antipsychotics and the slower ADL decline found with antidepressants, it is unlikely that these benefits are of clinical significance.

Possible Benefits in Cognitive Functioning with Depakote Use

A study by Ai-Guo et al. (2015) using mice looked at how astrocytes and microglia activated by amyloid-β (Aβ) contribute to the inflammatory process that develops around an injury in the brain. The authors reported that Depakote had been shown to have an anti-inflammatory function, and their study looked to explore the therapeutic effect of Depakote on the neuropathology and memory deficits in transgenic mice. They reported mice treated with Depakote showed markedly improved memory deficits and decreased Aβ deposition compared with the non-Depakote treated mice. Also, the extensive astrogliosis and microgliosis, as well as the increased expression in interleukin-1β (IL-1β) and tumor necrosis factor-α (TNF-α) in the hippocampus and cortex of the transgenic mice, were significantly reduced after Depakote, which lessened

neuronal degeneration. Simultaneously, Depakote alleviated the levels of p65 NF-$_k$B phosphorylation and enhanced the levels of acetyl-H3, Bc1-2, and phosphoglycogen synthase kinase (GSK)-3β that occurred in the hippocampus of the transgenic mice. These results indicate that Depakote could significantly improve spatial memory impairment and Aβ deposition, at least about inflammation. The researchers described spatial memory as being the part of the memory responsible for recording information regarding the environment and spatial orientation, and about STM, the spatial memory includes tasks such as learning, reasoning, and comprehension. Similarly, Yao et al. (2014) described AD as a very common progressive neurodegenerative disorder that affects learning and memory abilities in the brain. The researchers discussed key findings from recent studies of epigenetic mechanisms of memory that suggests chromatin remodeling disorders via histone hypoacetylation of the lysine residue contribute to the cognitive impairment in AD. These findings indicate that the inhibition of histone acetylation induced by histone deacetylases (HDACs) inhibitors contributes to the recovery of learning memory. This, in turn, indicates it is possible for STM to ameliorate during Depakote use. As in the Ai-Guo et al. study, Yao et al. looked at various mechanisms that could significantly change brain functioning in transgenic mice. They discussed how Depakote enhanced long-term recognition memory and spatial learning and memory in AD transgenic mice. Their research showed that Depakote could significantly elevate histone acetylation through HDACs activity inhibition and increase plasticity-associated gene expression within the hippocampi of mice. In sum, their study

suggested Depakote, when serving as an HDAC inhibitor, can be considered as a potential psychoactive medication for the improvement of cognitive function in AD. Furthermore, this research points up the gap in and need for research on the impact of Depakote on STM in NH dementia residents, as existing studies continue to yield inconsistent results.

Summary and Conclusions

This chapter provided a detailed account of current professional literature about the impact of Depakote on agitation and STM in NH dementia residents. A considerable number of studies have focused on various aspects of Depakote, agitation, and STM, including risks versus benefits, the effectiveness of psychoactive medications, and how the use of psychoactive medications might modify already present psychiatric symptoms. Depakote uses to manage BPSD in NH dementia residents was consistently reported not to improve agitation in dementia and to increase the frequency of side effects (Narayana et al., 2014). Although Depakote treatment options exist to treat agitation in NH dementia residents, inconsistent findings from existing research studies indicate the need for further research on the use of Depakote to manage agitation and aggression in BPSD in NH residents. Additionally, research studies considered the potential for a decline in cognitive function among NH residents with AD while on Depakote but did not touch specifically on STM in NH residents with AD. Several studies in the literature review conducted research addressing exposure to psychoactive medications and cognitive function (Dutcher et al., 2014; Ai-Guo et al., 2015; Yao et al., 2014).

Again, due to inconsistencies in findings from the literature reviewed, there is a continued call for further research into the impact of Depakote on agitation and STM in NH dementia residents. The impact of Depakote on agitation and STM in NH residents with dementia remains unclear. The goal of this study was not to solve the identified problem but to add to the existing literature, thereby increasing knowledge on the impact of Depakote treatment on agitation and STM in NH dementia residents over a 1-year period. Chapter 3 lays the groundwork for the research design, methodology, and data collection procedures used to complete this study.

Chapter 3: Research Method

The purpose of this quantitative study, which included a nonexperimental design, was to examine the impact of Depakote on agitation and STM in NH residents with dementia over a 1-year period. The independent variable was Depakote treatment, and the dependent variables were agitation and STM. There was also a variable of interest, effect over time (trials). The study included a confounding variable, which was the residents' original Depakote start date to test for within-subject effects.

This chapter contains the research methods employed in this study. A brief review of the design and rationale of the study, including setting and sampling procedure, procedures for recruitment, participation and data collection, and instrumentation, is presented. Next, the data analysis plan is discussed, including research questions, hypotheses, and statistical tests. A review of the threats to external, internal, and construct validity, including reliability of the instrument, data assumptions, sample size, and the measures taken to protect residents' rights, concludes this chapter.

The independent variable for this study was Depakote treatment, and the dependent variables were agitation and STM. According to the Diagnostic and Statistical Manual of Mental Disorders (DSM-5, 2013), agitation and memory loss are two symptoms of dementia. Resident behaviors, psychological symptoms, and cognition were examined to determine whether Depakote use impacted agitation and STM. The residents in this study were required to have a diagnosis of dementia and be prescribed Depakote for BPSD from their medical doctor or psychiatrist. Any residents receiving Depakote to

treat seizure disorder were excluded from this study (FDA, 2010). All data for this study were archived data from an existing dataset, and there was no direct communication between me and the residents.

This chapter presents a detailed description of the methodology used in this study to facilitate replication by other researchers. The major sections include the sampling and sampling procedures along with all procedures of recruitment, participation, and data collection. Next, I describe the instrument and operationalization constructs including the developers, appropriateness to this study, and reliability and validity. Additionally, threats to validity such as external, internal, and construct validity are presented. Finally, ethical procedures and concerns related to this study are described.

Research Design and Rationale

This quantitative study included a nonexperimental design. Data for sample selection were collected data using consulting pharmacy records. Data were then measured by MDS assessment results to determine whether Depakote use affected levels of agitation and STM in NH dementia residents over a 1-year period.

I used a quantitative approach including a repeated-measures within-subjects ANOVA to analyze data from two archived databases containing NH residents receiving Depakote. Because the purpose of this study was to determine whether Depakote impacted levels of agitation and STM over a 1-year period, a repeated-measures within-subject (effect over time) design was appropriate for this study. The repeated-measures design facilitated testing the null hypothesis and the alternative hypothesis, and all

archived data were measured on each variable. The null hypothesis stated there are no statistically significant differences in levels of agitation or STM in the Depakote treatment group over a 1-year period. The alternative hypothesis stated there are statistically significant differences in levels of agitation and STM in the Depakote treatment group over a 1-year period.

Because I used archival data from two existing data sets, I did not have direct contact with the residents, which mitigated ethical issues related to vulnerable populations and cognitively impaired residents. Data were gathered from the consulting pharmacies' database and two NHs MDS records to measure the impact of Depakote use on agitation and STM in NH dementia residents. This system of data gathering ensured the diagnosis of dementia, prior behaviors, current behaviors, and resident receipt of Depakote over the 1-year period. MDS is a tool mandated for use in NHs for clinical assessments of all residents on Medicare and Medicaid. The MDS provides a comprehensive assessment of each resident's functional capabilities, health-related issues, behaviors, psychological symptoms, and cognitive function.

Methodology

Population

The target population for this study was dementia residents from two local NHs. The residents for this study consisted of NH dementia residents receiving Depakote treatment for BPSD from January 1, 2016, through December 31, 2016, because this was the most recent data representing a full calendar year.

Sampling and Sampling Procedures

Purposive sampling was used for this study because the NH sample was being sampled with a specific purpose (specific to NH residents on Depakote and its impact on agitation and STM; see Frankfort-Nachmias, Nachmias, & DeWaard, 2015). Purposive sampling is a nonprobability technique that was used based on characteristics of the NH dementia residents (population) and Depakote treatment (objective). This sampling strategy was suitable for this study's research questions and variables addressing the extent to which Depakote impacts agitation and STM in NH dementia residents over a 1-year period.

The sample for this study was obtained from archival data collected from January 1, 2016, through December 31, 2016, by the consulting pharmacy and the NHs MDSs. The procedure for drawing the sample involved various steps. First, all residents were from the two local NHs and were receiving Depakote for BPSD. Second, the consulting pharmacy records were provided with deidentified data including psychoactive medications, the reason for Depakote prescription, Depakote dosage, and the start date of Depakote administration. Residents receiving Depakote for any reason other than agitation/behaviors related to dementia were excluded. Also, residents were required to be on Depakote for the entire 1-year period. Third, MDS records were obtained through the NHs. MDS records included deidentified data including date of birth, gender, and Sections C (Cognitive) and E (Behavioral) of the MDS. The consulting pharmacy and NHs are the owners of the data, so permission was not needed from the residents. Data

agreements between the data providers and me ensured protection and confidentiality of resident information.

The study's sampling frame was the group of residents from two local NHs that met eligibility criteria for selection. More specifically, the sampling frame was the consulting pharmacy list of NH residents receiving Depakote, comprising the sample selection of MDSs from the two NHs (permission granted through data agreements between the data providers and me). Only residents on Depakote for behaviors/agitation from that list met study selection criteria. Any NH resident on Depakote for any other reason other than BPSD was excluded from the study because the study was specific to dementia residents. Moreover, only selected data collected from those residents who had been receiving Depakote for the entire 1-year period were used. Gender, race, and age were not examined in this study because of the smaller sample size.

The sample size of a study is determined before research begins as an effort to ensure a sufficient number of responses (Creswell, 2014). Having a sufficient sample size ensures enough data (Creswell, 2014). I used G*Power 3.1 (see Faul, Erdfelder, Buchner, & Lang, 2009) to calculate sample size for a one-way repeated-measures ANOVA. When a population is of limited size, Cohen (1982) stated it is appropriate to set the alpha at .10 or higher, and Stevens (2002) stated a priori power should be at least .70. With four repeated measures correlated at .50, a medium-size population effect size of Cohen's f = .25 would be detectable with a sample size of 15, and an even smaller effect size of Cohen's f = .20 would be statistically significant within the sample.

Procedures for Recruitment, Participation, and Data Collection

Resident data were data archived from January 1, 2016, to December 31, 2016, from two local NHs. At the beginning of this research process, I contacted both NH administrators face-to-face to introduce the study, explain the purpose of the study, and request use of residents' MDS archival data. Immediate approval was given by both NH administrators at that time to obtain residents' MDS data for this study. Next, I contacted the pharmacy consultant via e-mail to introduce the study. I explained the purpose of the study and requested archival data (i.e., residents on Depakote, Depakote administration start and end dates, the reason for Depakote use, gender, and date of birth). The pharmacy consultant agreed to provide the requested data for this study.

The data collection for the main dataset required the consulting pharmacy to provide data from residents of two local NHs who are on Depakote treatment for BPSD. The consulting pharmacy has proprietary rights to all the de-identified information that was provided. The procedure for gaining access to the data set involved a data use agreement between the data provider (consulting pharmacy) and the data recipient (me), which permitted limited use of the de-identified data set for research purposes only. The data use agreement (Appendix A) with the consulting pharmacy specified access to a limited deidentified data set (i.e., date of birth, gender, psychoactive medications, the reason for Depakote prescription, the dosage of Depakote, and the start date of Depakote administration).

Collection of secondary data involved administrators and MDS coordinators from both participating NHs. NH administrators authorized NH MDS coordinators to give access to MDS records of study residents from the main data set. NH MDS coordinators provided copies of study residents' Sections C (Cognitive) and E (Behavioral) of the MDS (deidentified dataset). NHs have the sole rights to all MDS records needed for this study; therefore, resident permission was not needed. The procedure for gaining access to the data set involved a data use agreement similar to the agreement with the consulting pharmacy. The data use agreement was between the data provider (NH) and data recipient (me), which permitted limited use of the de-identified data set for research purposes only. The data use agreement with the NHs specified a limited data set (i.e., date of birth, gender, and Sections C [Cognitive] and E [Behavioral] of the MDS).

Use of the data sets was granted from the consulting pharmacy and the two local NHs (i.e., the owners of the confidential data being provided to me). All residents had a diagnosis of dementia and were not able to provide consent; therefore, the NHs had the right to determine the utility of sharing the data for research purposes. Residents were identified by ID 1-16, and NH names remained confidential to protect all information shared for this study. These data were inputted into R Statistical Software for statistical analysis.

Instrumentation and Operationalization of Constructs

Minimum Data Set (MDS)

The MDS is a standardized tool for assessment and facilitation of care management in NHs developed by U.S. Centers for Medicare & Medicaid Services (CMS; CMS.gov, 2015). The MDS is a care management tool used in NHs and is a set of screening and assessment tools that are part of a Resident Assessment Instrument (Cirillo, 2017). The MDS provides an assessment of NH residents' functional capabilities and helps identify problem areas. The MDS is performed on every resident in Medicare and/or Medicaid-certified long-term care facilities.

The MDS is designed to be reliable and accurate and to include the resident (if cognitively able) in the assessment process. CMS (2015) reported that enhanced accuracy of the MDS supports the intent that MDS be a tool to improve clinical assessment and support the credibility of programs that rely on MDS. The MDS includes frequency reports designed to summarize each NH resident's information, creating an MDS assessment record. The MDS assessment information for each NH resident is consolidated to create a profile of the most current standard information for the resident. A detailed copy of Sections C (Cognitive) and E (Behavioral) of the MDS is located in Appendix D.

The MDS is structured by sections A-Q, V, X, and Z, including a title and intent for each section. Each section provides instructions on how to complete the section. The MDS consists of the following areas: functional, physical, psychological, and health.

Section titles are as follows: Identification Information (A), Hearing, Speech, and Vision (B), Cognitive Patterns (C), Mood, Behavior (D), Preferences for Customary Routine and Activities (F), Functional Status (G), Bladder and Bowel (H), Active Disease Diagnosis (I), Health Conditions (J), Swallowing/Nutritional Status (K), Oral/Dental Status (L), Skin Conditions (M), Medications (N), Special Treatments and Procedures (O), Restraints (P), Participation in Assessment and Goal Setting (Q), Care Area Assessment (CAA) Summary (V), Correction Request (X), Assessment Administration (Z).

An example is from Section C, Cognitive Patterns. This area begins with C0100: Should Brief Interview for Mental Status (C0200-C0500) be conducted? This interview should be attempted with all residents. This area is coded as follows: (0) No (resident is rarely/never understood)–Skip to and complete C0700-C1000, Staff Assessment for Mental Status, (1) Yes–Continue to C0200, Repetition of Three Words. For example, if the response were coded 1, the next section would be Brief Interview for Mental Status (BIMS) (C0200) Repetition of Three Words. The resident is asked to repeat three words, for example, sock, blue, and bed. This area is coded by the number of words repeated after the first attempt: (0) None, (1) One, (2) Two, (3) Three. After the resident's first attempt, the examiner repeats the words using cues such as sock, something you wear; blue, a color; bed, a piece of furniture, and these words may be repeated up to two more times. Followed by Temporal Orientation (C0300) (orientation to year, month, and day). (A) Able to report correct year is coded (0) Missed by > 5 years or no answer, (1) Missed by 2-5 years, (2) Missed by 1 year, (3) Correct; (B) Able to report correct month is coded

(0) Missed by >1 month or no answer, (1) Missed by 6 days to a month, (2) Accurate within 5 days; (C) Able to report correct day of the week is coded (0) Incorrect or no answer, (1) Correct (CMS.gov, 2015). Followed by Recall (C0400) (Ask resident to repeat the three words asked earlier). (A) Able to recall "sock", (B) Able to recall "blue," (C) Able to recall "bed," all coded using (0) No, could not recall, (1) Yes, after cueing, (2) Yes, no cue required (CMS.gov, 2015).

At this point in the assessment, scores are added up from sections C0200-C0400, ranging from 00-15 (15 being the most cognitively intact). The assessor would enter 99 if the resident was unable to complete the interview.

If the resident was unable to complete the interview, a staff assessment is completed. This area begins with C0600, Should Staff Assessment for Mental Status be Conducted? This area is coded (0) No (resident was able to complete interview) Skip to C1300, Signs, and Symptoms of Delirium, (1) Yes (resident was unable to complete interview) Continue to C0700, Short-term Memory OK. Staff Assessment for Mental Status (C0700) Short-term Memory OK (seems or appears to recall after 5 minutes) is coded (0) Memory OK, (1) Memory problem, (C0800) Long-term Memory OK (Seems or appears to recall long past) is coded (0) Memory OK, (1) Memory problem, (C0900) Memory/Recall Ability (check all that resident was normally able to recall) is coded (A) Current season, (B) Location of own room, (C) Staff names and faces, (D) That he or she is in a nursing home, (Z) None of the above were recalled. Followed by Cognitive Skills for Daily Decision Making (C1000) (Made decisions regarding tasks of daily life) is

coded (0) Independent—decisions consistent/reasonable, (1) Modified independence—some difficulty in new situations only, (2) Moderately impaired—decisions poor; cues/supervision required, (3) Severely impaired—never/rarely made decisions (CMS.gov, 2015).

The next area is Delirium (C1300) Signs and Symptoms of Delirium. (A) Inattention, (B) Disorganized thinking, (C) Altered level of consciousness, (D) Psychomotor retardation, all of which are coded using (0) Behavior not present, (1) Behavior continuously present, does not fluctuate, (2) Behavior present, fluctuates (comes and goes, changes in severity). Lastly in the cognitive section, is Acute Onset Mental Status Change (C1600), Is there evidence of an acute change in mental status from the resident's baseline?. This area is coded (0) No, (1) Yes (CMS.gov, 2015).

Another example is from Section E, Behavior, (E0100) Psychosis (Check all that apply) (A) Hallucinations (perceptual experiences in the absence of real external sensory stimuli), (B) Delusions (misconceptions or beliefs that are firmly held, contrary to reality), (Z) None of the above. Followed by Behavioral Symptom—Presence and Frequency (E0200), (A) Physical, behavioral symptoms directed toward others (hitting, kicking, pushing, scratching, grabbing, abusing others sexually), (B) Verbal behavioral symptoms directed toward others (threatening others, screaming at others, cursing at others), (C) Other behavioral symptoms not directed toward others (physical symptoms such as hitting or scratching self, pacing, rummaging, public sexual acts, disrobing in public, throwing or smearing food or bodily wastes, or verbal/vocal symptoms like

screaming, disruptive sounds). This area is coded using the following: (0) Behavior not exhibited, (1) Behavior of this type occurred 1 to 3 days, (2) Behavior of this type occurred 4 to 6 days, but less than daily, (3) Behavior of this type occurred daily. Overall Presence of Behavioral Symptoms (E0300), Were any behavioral symptoms in questions E0200 coded 1, 2, or 3? (0) No—skip to E0800, Rejection of care, (1) Yes—Considering all of E0200, Behavioral Symptoms, answer E0500 and E0600. Impact on Resident (E0500), Did any of the identifying symptom(s): (A) Put the resident at significant risk for physical illness or injury, (B) Significantly interfere with the resident's care, (C) Significantly interfere with the resident's participation in activities or social interactions, all coded with (0) No or (1) Yes. Impact on others, (E0600), Did any of the identifying symptom(s): (A) Put others at significant risk for injury, (B) Significantly intrude on the privacy or activities of others, (C) Significantly disrupt care or living environment, all coded with (0) No or (1) Yes (CMS.gov, 2015).

Rejection of Care—Presence and Frequency (E0800), Did the resident reject evaluation or care (bloodwork, taking medications, ADL assistance) that is necessary to achieve the resident's goals for health and well-being (excluding already addressed behaviors): (0) Behavior not exhibited, (1) Behavior of this type occurred 1 to 3 days, (2) Behavior of this type occurred 4 to 6 days, but less than daily, (3) Behavior of this type occurred daily (CMS.gov, 2015).

Wandering—Presence and Frequency (E0900), Has the resident wandered: (0) Behavior not exhibited, (1) Behavior of this type occurred 1 to 3 days, (2) Behavior of

this type occurred 4 to 6 days, but not daily, (3) Behavior of this type occurred daily. Wandering—Impact (E1000): (A) Does the wandering place the resident at significant of getting to a potentially dangerous place (stairs, outside of the facility), (B) Does the wandering significantly intrude on the privacy or activities of others, all coded (0) No or (1) Yes. Finally, to sum up the behavior section E, Changes in Behavior or Other Symptoms (E1100), How does resident's current behavior status, care rejection, or wandering compare to prior assessment, coded as (0) Same, (1) Improved, (2) Worse, (3) N/A because no prior MDS assessment (CMS.gov, 2015).

The MDS includes reports for statistical and comparison purposes, such as Facility Characteristics Report, Facility Level Quality Measure Report, Resident Level Quality Measure Report, Monthly Comparison Report, and Quality Measures Reports versus Nursing Home Compare. These reports provide NHs with the statistical data gathered from their facilities as well as facilities in their same state and other states for comparison purposes. For example, when an NH is rated by a number of stars, the MDS reports will provide the NH with knowledge of the areas that their facility lacks in compared to other facilities. This will help improve quality of care. Also, the MDS reports specify areas for improvement. Tucker (2013) reported that research over the past four years, since the MDS was restructured, has shown an increase in reliability and validity, specifically inaccuracy of the mood and cognitive status sections. One of the main components of this improvement is involving the NH residents (if cognitively able)

and/or direct care staff in the assessments, which provides more accurate and valid responses.

Data Analysis Plan

This study was conducted using R statistical software version 3.3.1 to examine to what extent Depakote affects levels of agitation and STM in NH dementia residents over a 1-year period. These variables were measured using the consulting pharmacy's records and two NH MDS records in four intervals over a 1-year period and imputed into R statistical software for statistical analysis. A repeated measures ANOVA was used to analyze data in R statistical software. Additionally, boxplots were done to examine if the length of time the resident was on Depakote had any significance on resident's agitation and STM scores.

Restatement of the Research Questions and Hypotheses

RQ1: To what extent does Depakote treatment affect levels of agitation in NH dementia residents over a 1-year period?

$H_0$1: There is not a statistically significant difference in levels of agitation with Depakote treatment over a 1-year period as evidenced by no change in agitation from preexisting data in Section E of the MDS.

H_a1: There is a statistically significant difference in levels of agitation with Depakote treatment over a 1-year period as evidenced by decreased agitation from preexisting data in Section E of MDS.

RQ2: To what extent does Depakote treatment affect STM in NH dementia residents over a 1-year period?

$H_0$2: There is not a statistically significant difference in STM with Depakote treatment over a 1-year period as evidenced by no change in STM functioning from preexisting data in Section C of the MDS.

H_a2: There is a statistically significant difference in STM with Depakote treatment over a 1-year period as evidenced by decreased STM functioning from preexisting data in Section C of MDS.

Statistical Testing

This study used a repeated measures ANOVA statistical test that screens for multiple measures from a group of people (Frankfort-Nachmias, Nachmias, DeWaard, 2015). A repeated measures ANOVA is used when there are multiple dependent variables, and the researcher is looking for differences amongst treatment groups. Therefore, since this study consisted of two dependent variables (agitation and STM), one independent variable (Depakote treatment group), and repeated measures within factor (effect over time) using four intervals over a 1-year period, the data were screened using a repeated measures ANOVA to test the null and alternative hypotheses. Hence, the ANOVA statistical analysis was appropriate to examine to what extent Depakote affected levels of agitation and STM in the Depakote treatment group. Additionally, ANOVA was used to consider a within factor effect over time, which was pertinent to testing this study's hypothesis and provided adequate statistical power. The study included a

confounding variable, which was the residents' original Depakote start date, to test for within-subject random effects. Hence, box plots were employed for comparison of a number of months the resident was on Depakote before the study. Further, the results of this study were interpreted using certain key parameter estimates.

Threats to Statistical Conclusion and Validity

External Validity

External validity refers to a generalization of the treatment outcomes. For example, the external validity in this study related to repeated measures as it is difficult to control for the effects of prior outcomes using the same subject (Frankfort-Nachmias, Nachmias, & DeWaard, 2015). A repeated measures ANOVA was used for this study because it allows both dependent variables to be measured on the same independent variable. Also, this study had a within-subject variable of interest (effect over time) to examine if the residents changed from the first trial to final trial, which minimized threats to external validity. No pretest was conducted which could have potentially influenced the residents' responsiveness or sensitivity to the experimental variable (Creswell, 2014), thereby resulting in no threats to a reactive or interaction effect of testing for this study. Also, there were no threats of multiple treatment interferences (no multiple treatments will be given to the same subject). Reactive effects of experimental arrangements for effects are nonexperimental and can be easily generalized.

Internal Validity

The internal validity of a study refers to whether results of the study can be used to determine (a) if treatment makes a difference or not and (b) if there is sufficient evidence to support the claim (Creswell, 2014; Frankfort-Nachmias, Nachmias, & DeWaard, 2015). In this study, a repeated measures ANOVA was used to determine the impact of Depakote on agitation and STM. There was a possibility this design could increase the chances of internal validity by testing both dependent variables (representing different measurements) against the same independent variable. However, there are always factors that can jeopardize internal validity. For example, if the scorer of the MDS changes, there is a possibility of outcome changes. Furthermore, about the threats above to internal validity, the instrument (MDS) for this study was designed to be a reliable tool for clinical assessments (CMS.gov, 2015).

Construct Validity

Construct validity refers to how well a test or tool measures the theorized psychological construct it was designed to measure (Cronbach & Meehl, 1955). The tool used in this study, the MDS, was designed to measure agitation/behaviors and cognition. This study was not exposed to construct validity, such as hypothesis guessing by residents, bias experimental design, and researcher expectations (secondary data was used; Cronbach & Meehl, 1955). However, this study may have been exposed to a threat to the construct validity in that it was difficult (a) to establish disease-specific actions and (b) to distinguish whether outcomes occurred because of the medication's actions on an

underlying pathological process or as a consequence of being in an altered state (Moncrieff & Cohen, 2009). Those above was identified in the limitation section. This potential limitation is an area for further research.

Ethical Procedures

Protection of Residents' Rights

Ethical behaviors and protection of this studies residents are a serious matter for psychological studies. Every action in this study was taken with careful consideration for the residents. Agreement to gain access to the consulting pharmacists' data was received by way of a formal data use agreement (see Appendix A). The agreement was signed by both the data provider and data recipient to permit the usage of the dataset from the consulting pharmacist. The agreement was limited to the resident's gender, date of birth, psychoactive medications, the reason for Depakote prescription, Depakote dosage level, and the start date of Depakote. The contract excluded all medications other than psychoactive medications. A detailed copy of the data use agreement contract is located in Appendix A.

Access to the NHs MDS records was gained by way of formal data use agreements (see Appendices B and C). The agreements were signed by both NHs (data providers) and the data recipient to permit usage of the MDS dataset. The agreements were limited to the resident's date of birth, gender, and Sections C (Cognitive) and E (Behavioral) of the MDS. The contracts excluded all identifying information. Detailed copies of the data use agreement contracts are located in Appendices A-C.

This study did not involve any interactions with or observations of human subjects. Permission was gained from the Institutional Review Board (IRB) by completing an application to ensure that the ethical principles of beneficence, justice, and respect for persons were upheld in this study. In this study, secondary data were used, thereby limiting ethical concerns related to recruitment materials and processes as well as data collection. In the collection of the original data, the collectors of the data, the consulting pharmacist and two local NHs, ensured that residents were treated fairly. Confidentiality and limits to confidentiality were discussed and guaranteed in the original data collection process. Furthermore, families of the residents were involved in making a choice regarding psychoactive medications, including risks and benefits. There were no reports of families declining the administration of psychoactive medications or any adverse events that occurred during the original data collection process. There was also no report of any resident's Depakote being discontinued during the 1-year study period.

Data are confidential as stated in the resident's initial paperwork upon admission to the NHs. Before starting psychoactive medications, the NHs notify the resident and/or families of their consent. There have been no breaches of confidentiality or concerns about this data set.

The pharmacy consultant and the NHs abide by strict measures to preserve the confidentiality of the data. The procedure involved no access to data from the pharmacy consultant other than for the NHs purpose. The pharmacy consultant stored resident data securely on his computer. Additionally, the NHs abide by strict measures to preserve the

confidentiality of the data. MDSs are stored on computers of clinical staff that complete them, in the MDS 3.0 software program. Both data providers back data up on discs for safety purposes in the event that computers crash.

The data dissemination of the pharmacy consultant was limited to himself, both NHs, and the pharmacy that dispenses the medications. The data dissemination of the NH MDS's was limited to the NHs, insurance companies (for payment purposes), and the New York State Department of Health (NYSDOH) to ensure proper completion and adherence. The residents and/or families were not recipients of the reports or data. In compliance with NYSDOH guidelines, an NH resident's data are held for seven years following the resident's death.

Hard copies of data received from the pharmacy consultant and the NHs are secure in a locked fireproof box, and the researcher is the only person with access to that locked box. The raw data were coded into R statistical software for statistical analysis, using my personally secured (administrator password-protected) computer equipped with Webroot antivirus software and anti-spyware protection. Following analysis, statistical data were then securely stored on a separate hard drive with restricted access by administrator password protection. Also, write permission was disabled to prevent altering of the data so that they remained safe. Antivirus and anti-spyware were run on a daily basis and updates were applied to maintain the security of the data set. Lastly, the data will be kept for five years as required by Walden University, and copies will be stored in two different locations (Walden University, 2014). After the five-year period,

the data will be securely shredded and disposed of, and electronic files will be erased from computers.

There are no other ethical issues related to this study. Using secondary data eliminated the ethical risk of conducting the study at my internship site, eliminating conflicts of interest in this study. Also, use of secondary data protects both the vulnerable population (geriatric NH residents) and me from ethical risks.

Summary

This chapter included justification for the research design and rationale for my use of a quantitative approach with repeated measures within-subject design to analyze archival data. The rationale for the use of a repeated measures ANOVA was discussed. It was agreed that a repeated measures design for this study facilitated testing the hypotheses of whether or not there is a statistically significant difference in levels of agitation and STM with Depakote treatment over a 1-year period. Moreover, effect over time was a variable of interest that measured if the residents changed from the first trial to the final trial. The methodology, including the target population (local NH dementia residents) sampling strategy (purposive sampling), procedures, sampling frame, and power analysis were used to determine sample size and discussed in depth to ensure that this study was replicable. Next, the data collection procedures of the archival data along with the published instrument (MDS) were presented. Further, the threats to the study's external, internal, and construct validity were examined, as well as how these threats were addressed and presented. Finally, ethical procedures including data agreements (i.e.,

what data will be included in the data set provided), treatment of human residents (with beneficence, justice, and respect), and treatment of data (with confidentiality maintained and protected) were examined and addressed. The subsequent chapter will explore the analysis of the data set, the study's findings, and a summary of the answers to the research questions.

Chapter 4: Results

The purpose of this nonexperimental quantitative study was to examine the impact of Depakote on agitation and STM in NH dementia residents over a 1-year period. This study was conducted to answer two research questions:

1. To what extent does Depakote treatment affect levels of agitation in NH dementia residents over a 1-year period?
2. To what extent does Depakote treatment affect STM in NH dementia residents over a 1-year period?

The first null hypothesis stated no statistically significant difference in levels of agitation with Depakote treatment over a 1-year period as evidenced by no change in agitation from preexisting data in Section E of the MDS. The second hypothesis stated no statistically significant difference in STM with Depakote treatment over a 1-year period as evidenced by no change in STM functioning from preexisting data in Section C of the MDS. The hypotheses were tested using repeated-measures ANOVA. This chapter presents the method for collecting data and the results of the data analysis to address the research questions and hypotheses. First, I provide the details of the data collection, including descriptive statistics of the sample. Then I present the results of the data analysis. This chapter concludes with a summary of findings.

Data Collection and Characteristics of the Sample

Data Collection

NH residents with dementia and agitation who were receiving Depakote over the 1-year period were the sample for this study. A total of 16 residents from the two NHs were used for the sample. Two NHs were necessary to obtain the required sample size based on selection criteria that residents had been on Depakote for the 1-year period, had been diagnosed with dementia, had been prescribed Depakote to treat agitation, had not been prescribed Depakote for other diagnoses. A sample from one facility was too small for the study, and a second facility was needed. There were no variations between the two NH MDS data sets because both NH MDS coordinators were expected to follow the same MDS completion requirements. Demographics were not included in this study because the sample was too small for demographics to be significant (see Cohen, 1982). Data from the consulting pharmacy providing residents who met the study criteria were sent via secured e-mail to the participating NH data providers following IRB approval. I obtained study data within three days of the NH receiving the data from the consulting pharmacy. I physically acquired all residents' MDSs from the data providers, which included four separate MDSs per resident for repeated measures during the study period. This data collection process went as proposed and there were no discrepancies.

Characteristics of Sample

All residents were residents at one of the two participating NHs, had a diagnosis of dementia, and were receiving Depakote over the 1-year period. Their Depakote start

dates varied from 2013 through 2016, but they were all receiving Depakote from January 2016 through December 2016, which was the period evaluated. Each resident had four measures of agitation and STM scores over the 1-year period. Possible behavior/agitation scores ranged from 0 to 3, 0 indicating no behavior exhibited, 1 indicating behavior occurred 1 to 3 days, 2 indicating behavior occurred 4 to 6 days (but less than daily), and 3 indicating behavior occurred daily. From the 16 residents (labeled as ID), 11 scored 0 on all four behavior measures; three scored three 0's and one 2 on the four measures; one scored two 0's and two 2's on the four measures; one scored three 0's and one 3 on the four measures. Agitation scores are shown in Table 1.

Regarding STM, possible scores are 00-15 (15 indicating the most cognitively intact). STM was measured four times over the 1-year period. Some residents' STM varied slightly and others' more significantly over the 1-year period. STM scores are shown in Table 1.

Table 1

Characteristics of the Study Sample

ID	Time	Depakote start date	Agitation	STM score
1	1	1/31/13	0	07
1	2		0	02
1	3		0	04
1	4		2	00
2	1	5/321/15	0	08
2	2		0	00
2	3		0	00
2	4		0	00
3	1	1/30/16	0	09
3	2		0	14
3	3		0	14
3	4		0	15
4	1	10/25/14	0	00
4	2		2	00
4	3		0	00
4	4		0	00
5	1	12/27/15	0	14
5	2		0	12
5	3		0	10
5	4		0	10
6	1	5/31/15	2	13
6	2		0	15
6	3		0	14
6	4		0	14
7	1	2/22/14	0	06
7	2		0	07
7	3		0	07
7	4		0	07
8	1	7/26/13	0	00
8	2		0	00
8	3		0	00
8	4		0	00
9	1	9/28/13	0	15

(table continues)

ID	Time	Depakote Start Date	Agitation	STM Score
9	2		0	15
9	3		0	15
9	4		0	12
10	1	5/31/15	0	00
10	2		0	00
10	3		0	01
10	4		0	00
11	1	1/23/16	0	00
11	2		2	13
11	3		2	13
11	4		0	15
12	1	7/29/13	0	05
12	2		0	06
12	3		0	12
12	4		0	04
13	1	10/24/15	0	14
13	2		0	14
13	3		0	12
13	4		0	13
14	1	10/25/14	0	15
14	2		0	15
14	3		0	15
14	4		3	15
15	1	6/28/15	0	11
15	2		0	11
15	3		0	06
15	4		0	11
16	1	7/19/13	0	00
16	2		0	00
16	3		0	00
16	4		0	00

Data Screening

Before data analysis, data were screened to ensure residents met selection criteria. The consulting pharmacy provided the two NHs with data from the residents who met study criteria. Study criteria were as follows: residents had to be on Depakote for the 1-year period, had to have a diagnosis of dementia, and had to be prescribed Depakote to treat agitation, Residents on Depakote for other diagnoses were excluded. This allowed the NHs to provide me with a final deidentified data set that included 16 residents with a diagnosis of dementia who were receiving Depakote for agitation for the 1-year period addressed in this study. The MDS was the tool used for assessment measures, and four quarterly MDSs per resident were provided for repeated-measures ANOVA with within-subject effects.

Overview of Design and Procedures

Agitation and STM were assessed for each resident through archival data from the pharmacy consultant and the two NHs. The MDS was the instrument used by the NHs for initial assessments, and MDS data were provided to me for analysis. The MDS was used to determine the resident's agitation and cognitive scores over a 1-year period. The sections of the MDS that were used to measure the resident's agitation and cognition scores were Section C (Cognition) and Section E (Behavior). The agitation scores ranged from 0 to 3. Out of the 64 repeated measures, there were five scores of 2, one score of 3, and 58 scores of 0. The STM scores ranged from 00 to 15 and varied from resident to resident. The mean and *SD* of agitation were 0.20 and 0.65. The mean and *SD* for STM

were 7.5 and 6.10. The mean scores and standard deviations for the dependent variables are shown in Table 2.

Table 2

Descriptive Statistics for Agitation and STM

Variable	Mean	*SD*
Agitation	0.20	0.65
STM	7.5	6.10

Data Analysis Results and Major Findings

To test the hypotheses and examine the impact of Depakote on agitation and STM in NH dementia residents over a 1-year period, I conducted a repeated-measures within-subjects ANOVA; however; because of possible correlation within the subjects, I employed boxplots to determine whether the length of time residents were on Depakote had any significance. Data were analyzed using R Statistical Software Version 3.3.1 for Windows or Mac. Preliminary tests were done on the data before running the repeated-measures ANOVA. Simple plots and boxplots were employed to get an overall view of the data before proceeding to more complex analyses using repeated-measures ANOVA. The simple plots in Figure 1 and Figure 2 showed no clear increasing or decreasing pattern for agitation and STM scores over time.

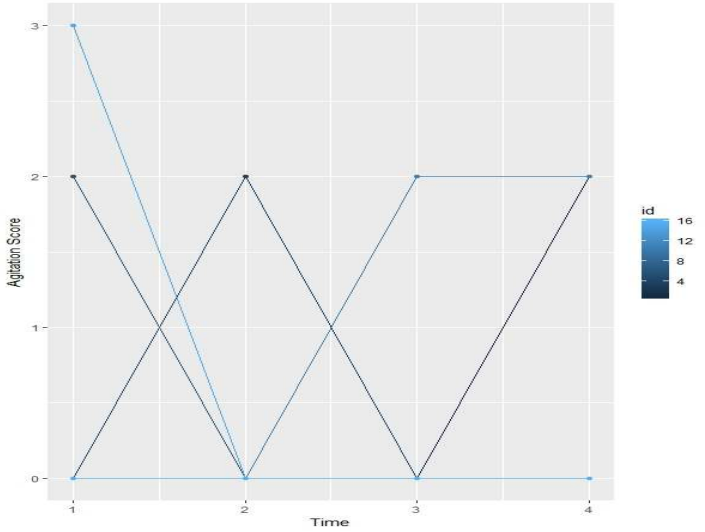

Figure 1. Simple plots for agitation scores over a 1-year period.

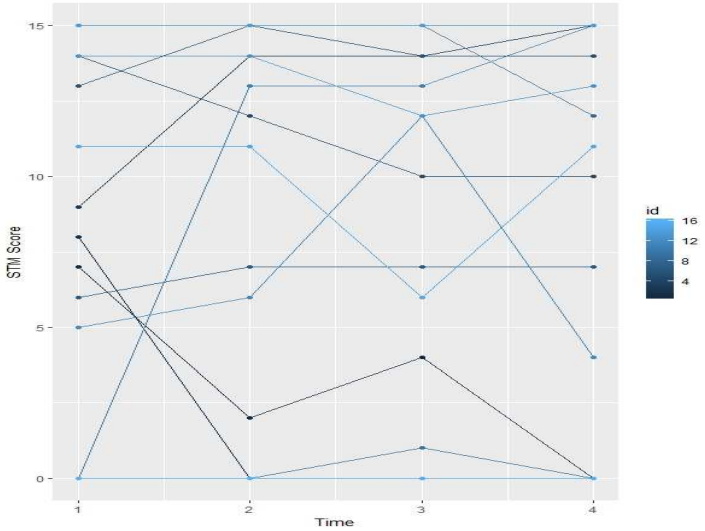

Figure 2. Simple plots for STM scores over a 1-year period.

To simplify the data, I used boxplots with differences between time intervals. The boxplots in Figure 3 and Figure 4 indicated no difference between the scores after another time interval by having medians close to 0 for all cases.

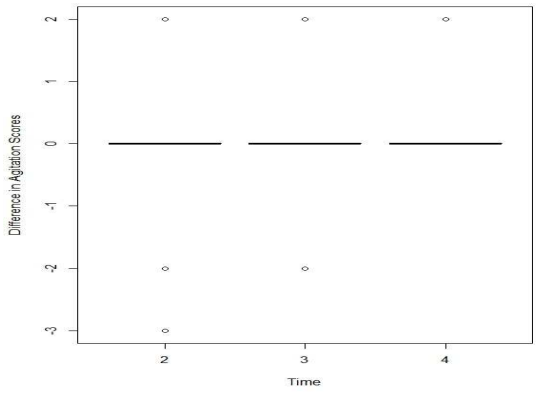

Figure 3. Boxplots for differences in agitation over each quarter.

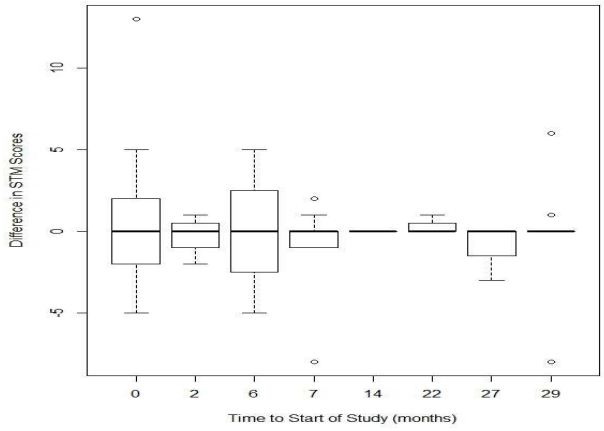

Figure 4. Boxplots for differences in STM over each quarter.

To rule out the effect of varying Depakote start dates; the differences in scores over time were compared to the number of months from the Depakote start date to the start of the study. The boxplots in Figure 5 and Figure 6 show there was no clear correlation between Depakote start dates to the start of the study and agitation and STM scores.

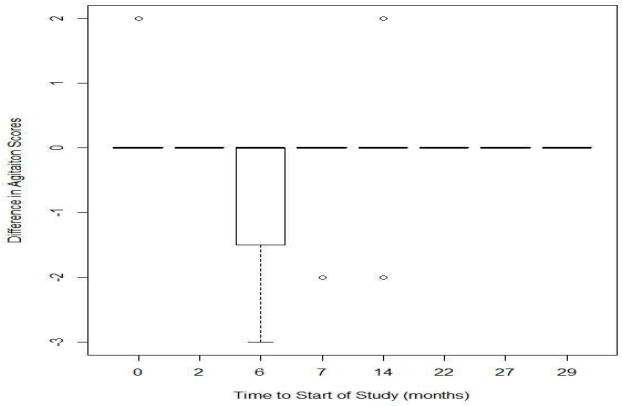

Figure 5. Boxplots for differences in agitation scores compared to Depakote start date.

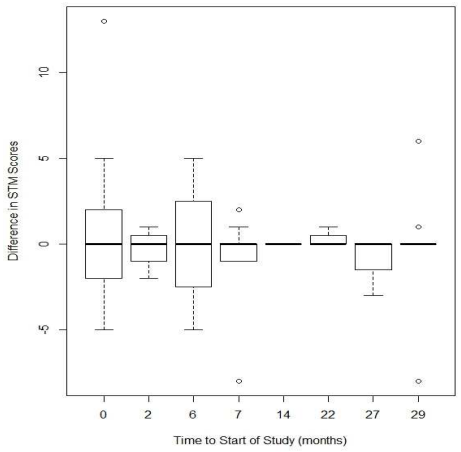

Figure 6. Boxplots for differences in STM scores compared to Depakote start date.

Repeated measures ANOVA was used to test the difference in agitation over the 1-year period of Depakote treatment. A typical rule-of-thumb for ANOVA is to reject the null when the p-value is below 0.05. Under this cut-off, there was no significant evidence to reject the null hypothesis $H_0 1$ for agitation (F-value 0.07, p-value = 0.7988) and STM scores (F-value 0.0023, p-value = 0.9617).

A further ANOVA test was done to examine if repeated measures ANOVA was required with this data set. Accounting for the random effects of ID is the main difference between an ANOVA and a repeated measures ANOVA. In this test, the ANOVA was used to check if there was a significant difference model with or without the random effects of ID. There was no significant difference between with and without random effects models for agitation (df=3, p-value = 0.2266), however, there was a significant improvement in the model when accounting for the random effects of ID for STM scores

(df =3, p-value < 0.0001). A repeated measures ANOVA was required for STM scores; the repeated measures ANOVA still did not show a significant difference in STM scores over time.

Research Question 1

To what extent does Depakote treatment affect levels of agitation in NH dementia residents over a 1-year period?

$H_0$1: There is not a statistically significant difference in levels of agitation with Depakote treatment over a 1-year period as evidenced by no change in agitation from preexisting data in Section E of the MDS.

H_a1: There is a statistically significant difference in levels of agitation with Depakote treatment over a 1-year period as evidenced by decreased agitation from preexisting data in Section E of MDS.

The repeated measures ANOVA found no significant difference (F = 4.96, df = (3), p = 0.2266) between agitation and Depakote treatment over the 1-year period; therefore, there was no significant evidence to reject the null hypothesis.

Research Question 2

To what extent does Depakote treatment affect STM in NH dementia residents over a 1-year period?

$H_0$2: There is not a statistically significant difference in STM with Depakote treatment over a 1-year period as evidenced by no change in STM functioning from preexisting data in Section C of the MDS.

*H*a2: There is a statistically significant difference in STM with Depakote treatment over a 1-year period as evidenced by decreased STM functioning from preexisting data in Section C of MDS.

The repeated measures ANOVA found no significant difference ($F = 26.95$, $df = (3)$, $p = <.0001$) between STM and Depakote treatment over the 1-year period; therefore, there was no significant evidence to reject the null hypothesis.

Exploratory Analysis of Length of Time on Depakote and Agitation and STM

Although the length of time on Depakote was not used in any of the primary analyses, it is possible that the effects of length of time on Depakote can impact agitation and STM scores. Specifically, it might be that a resident who has been on Depakote treatment for a longer period may have lower agitation scores and higher STM scores. However, boxplots did not show any clear correlation between length of time on Depakote and agitation scores. Though findings did show significant differences in the exploratory analysis of STM scores and length of time on Depakote, results were still not significant over the 1-year study period.

Summary

Based on the findings of these analyses, the null hypothesis could not be rejected for the two research questions explored, which examined the impact of Depakote on agitation and STM in NH dementia residents over a 1-year period. Reported findings showed that Depakote had no effect on agitation scores over the 1-year period.

Additionally, with random effects, there was still no significant difference in agitation scores.

Findings did not support the hypotheses that there were significant differences in STM scores in NH dementia residents on Depakote over a 1-year period. Although findings did show significant differences in the exploratory analysis of STM and length of time on Depakote, results were still not significant over the 1-year study period. Chapter 5 will include a summary of this study and an explanation of why and how the study was conducted. Conclusions based on the results and impacts of these conclusions will be presented. Implications of this study will be discussed, along with recommendations for future research.

Chapter 5: Discussion, Conclusion, and Recommendations

Study Overview

The purpose of this nonexperimental study was to examine the impact of Depakote on agitation and STM in NH dementia residents over a 1-year period. Despite the vast amount of literature on Depakote used for agitation and its effects on cognition, the existing studies indicated inconsistent results. Results of the current study indicated no significant impact of Depakote on agitation and STM scores. Although the length of time on Depakote was not addressed in primary analyses, testing of IDs with and without random effects for agitation indicated no significant differences. The current study findings revealed significant improvement in the model when accounting for random effects of ID for STM scores, but no significant difference in the repeated-measures ANOVA in STM scores was found over the 1-year period.

The importance of this study was justified by the dearth of empirical data regarding Depakote's impact on agitation and STM in NH dementia residents. The use of Depakote and its impact on NH dementia residents was well documented in the literature. However, because of the inconsistent findings in the literature on Depakote's impact on agitation and STM in NH dementia residents, this study was needed to clarify the impact and efficacy of a common yet largely unexamined invasive treatment on an underserved, vulnerable population. This chapter includes the purpose of the study, the research questions, and an interpretation of the findings in the context of related literature and the

theoretical framework. I also describe the limitations of the study, recommendations for further research, and implications for social change.

Interpretation of Findings

The data analysis was conducted to answer two research questions addressing the extent to which Depakote treatment affected agitation and STM in NH dementia residents over a 1-year period. Agitation and STM were measured using the MDS assessment scores. The dataset was obtained from the two participating NHs following the consulting pharmacies' dataset of residents who met the study criteria.

The initial data analysis supported the null hypothesis for Research Question 1. The alternative hypothesis was not accepted because results indicated that Depakote did not significantly affect agitation scores over the 1-year period. Even when random effects were accounted for, there was no significant impact.

The second analysis also supported the null hypothesis for Research Question 2. The alternative hypothesis was not accepted because results indicated that Depakote did not affect STM scores over the 1-year period. However, when accounting for random effects, results showed significant improvement in STM scores. Although random effects analyses showed significant improvement in STM scores, results did not indicate a significant difference in STM scores over the 1-year period. Therefore, the null hypothesis was not rejected.

Literature Review and Research Findings

This study addressed the inconsistencies in previous research findings based on Moncrieff and Cohen's (2009) drug-centered theory, which purported that because psychoactive medications are extrinsic substances altering how the body works, there are advantages and disadvantages to their use that should be weighed and distinguished from the effects of general treatment. Depakote is an anticonvulsant medication used to manage agitation in approximately 80% of NH residents with dementia (Meeks & Jeste, 2008). Medicare reports showed dementia affects approximately 5 to 8 million Americans, of which more than half have BPSD (Mittal et al., 2011).

There are many different manifestations of dementia, including cognitive decline, alterations in mood/thought/behavior, and inability to conduct activities of daily living. As in the Richter et al. (2015) study, Meinhold et al. (2005) described BPSD as depression, hallucinations, delusions, agitation, aggression, combativeness, disinhibition, and hyperactivity, and found that pharmacological measures are often used to control BPSD. Researchers have noted that Depakote may have some advantages when used for agitation and aggression in dementia residents because of its lower rates of drug interactions and adverse effects in the dementia population. Meinhold et al. used pharmacy databases and MDS assessments to obtain data for residents who were receiving Depakote for behavior problems related to dementia. Similar to the current study, some exclusions were applied, such as residents who were receiving Depakote for seizures. Meinhold et al. found the use of Depakote for agitation and aggression in

dementia residents was effective, safe, and tolerable. This current study confirmed that Depakote use did not increase agitation scores, suggesting Depakote is effective for agitation.

Herrman et al. (2007) reached findings along the same lines, reporting Depakote is ineffective for managing agitation in NH residents with AD and may be poorly tolerated in this population. Herrman et al. discussed similar previous randomized controlled trials that also showed no statistically significant benefits of Depakote on primary outcome measures, though it showed efficacy for some secondary measures. The major limitations of that study were the small sample size and crossover design, but despite these limitations, significant worsening of symptoms during Depakote treatment was found. Similar to the current study, no significant differences were found with Depakote treatment, though results showed some efficacy for secondary measures, but not enough to be significant. A difference between Herrman et al.'s (2007) study and the current study is that Herrman et al. found a significant worsening of symptoms during Depakote treatment, and the current study did not indicate that. The current study is also similar to Herrman et al.'s in that the small sample size could have impacted the results.

STM is the system that temporarily stores and manages information that is necessary to complete complex cognitive tasks (Ai-Guo et al., 2015). Richter et al. (2015) showed that one of the adverse effects of Depakote treatment is diminished cognitive function. Results of the current study indicated that, when accounting for random effects of the ID for STM scores, there was a significant improvement; however, the repeated-

measures ANOVA did not show a significant difference in STM scores over the 1-year period.

Dharmarajan and Gunturu (2009) discussed the high rates of AD in NH residents and the possible causes. Dharmarajan and Gunturu described AD as a common, acquired disorder that manifests as slow, progressive memory loss with at least one cognitive dysfunction (aphasia, apraxia, agnosia, or executive dysfunction), resulting in impaired occupational and social performance. Dharmarajan and Gunturu discussed (a) the use of psychoactive medications, including Depakote, to manage agitation and difficult behaviors in NH dementia residents and (b) the impact of the medication on cognitive function. Similar to the Richter et al. (2015) study, Dharmarajan and Gunturu addressed adverse effects and cognitive functioning but not specifically on STM. The current study did not address other adverse effects, only agitation, and STM scores because there was little previous research on STM and Depakote. The current study did not indicate any significant effect of Depakote increasing memory loss, but possibly slowing cognitive decline. This is because there was no significant difference in STM over the 1-year period, indicating IDs maintained cognitive functioning over the 1-year period.

Ai-Guo et al. (2015) used mice to examine how astrocytes and microglia activated by amyloid-β (Aβ) contributed to the inflammatory process that develops around brain injury. Ai-Guo et al. reported that Depakote had been shown to have an anti-inflammatory function, and their study addressed the therapeutic effect of Depakote on the neuropathology and memory deficits in transgenic mice. Ai-Guo et al. reported that

mice treated with Depakote showed markedly improved memory deficits and decreased Aβ deposition compared with the non-Depakote treated mice. Similar to these findings, results from the current study indicated no differences in STM over the 1-year period; therefore, there is a possibility that Depakote may contribute to the slower cognitive decline. In contrast, Manckoundia et al. (2008) found different results of Depakote's influence on cognitive functioning. The individual in this case study showed a significant decline in cognitive functioning once she began the Depakote. The Depakote was then discontinued due to these adverse effects, and she regained her cognitive function.

When reviewing the literature, I observed considerable inconsistencies in study findings. Some researchers reported that Depakote was effective for agitation, while other researchers reported that Depakote increased agitation and other BPSD symptoms. Some researchers reported that Depakote slowed cognitive decline, while other researchers indicated that Depakote caused a decline in cognitive functioning.

Theoretical Framework and Research Findings

The theoretical framework for this study was Moncrieff and Cohen's (2009) drug-centered theory, which suggests that because psychoactive medications are extrinsic substances altering how the body works, advantages and disadvantages of psychoactive medication use should be weighed and distinguished from the effects of general treatment. Drug-centered theory can inform perspectives about how psychological alterations produced by psychoactive medications interact with experiences of distress and the need to seek psychological help. Many disorders can mimic psychoactive effects,

such as sedation, cognitive slowing, behaviors, altered sense of perception, sleep, and psychosis.

Moncrieff and Cohen (2009) theorized that although evidence of the superiority of psychoactive medications might imply disease-specific effects, superior effects can also be explained within a drug-centered framework (e.g., the psychomotor and emotional restriction characteristic of psychoactive medications suppresses psychotic agitation more effectively than other sedatives). The drug-centered theory provides a useful lens through which NH resident clinicians or staff members can evaluate and report the effects of Depakote treatment in NH dementia residents. The drug-centered framework further prompts members of the psychiatric research community to produce relevant, unbiased information about the short- and long-term effects that psychoactive medications exert on cognition, behaviors, and bodily systems.

Limitations of the Study

One of the limitations of this study was that the sample was drawn from only two NHs in the United States and did not adequately represent all aspects of the NH population. Residents were obtained for this study through the two local NHs with preexisting data. Data collection served as a limitation because there were several selection criteria: needing to be on Depakote for the 1-year period, having a diagnosis of dementia, and receiving Depakote treatment for agitation/behaviors. Residents who were taking Depakote for other reasons were excluded.

Another limitation was that I assumed that the NH MDS coordinators were answering the MDS questions accurately and completing the resident/staff interviews on time. If the resident/staff interviews were not completed each quarter, scores could be inaccurate. The credibility of findings depended on the honesty and diligence of the data providers.

Implications for Social Change

Implications for social change include assessing the impact and efficacy of a common yet largely unexamined invasive treatment on an underserved, vulnerable population. Much of the existing research on Depakote and STM has been solely focused on the adverse effects of various psychotropic medications and cognitive functioning; however, this study specifically employed Depakote because it tends to be the most widely used medication to treat agitation in NH dementia residents (Richter, Mann, Meyer, Haastert, & Köpke, 2011). Also, this study specifically looked at how Depakote effected STM in NH dementia residents. There has been noted research on Depakote and its impact on cognitive functioning; however, research is lacking in STM. Research on this subject is fairly complex because members of the target population are often unable to express how they feel. Therefore, cognitive testing is important in this population in that it allows for the determination of memory loss. The most common form of memory loss in dementia residents starts with STM, and this does not seem to be a focus of much of the existing literature. Therefore, this study focused on how Depakote impacts STM in NH dementia residents.

This research adds to the literature of research conducted solely on the impact of Depakote on agitation and STM in NH dementia residents. This research can provide avenues for social change by providing new information to appropriate professionals, on the most common treatment (i.e., Depakote) for a prevalent and exigent societal problem (i.e., BPSD).

Recommendations for Future Research

The current study has increased understanding of the impact of Depakote on agitation and STM in NH dementia residents over a 1-year period. It is recommended that additional research is done on this topic. The current study used quantitative measures; however, conducting this study using qualitative measures would possibly produce more comprehensive analysis and statistical significance as it would gather specific themes that could be explored to address the impact of Depakote on agitation and STM. Results indicated there was no significant difference in agitation and STM scores among the 16 residents in this study. Future research should also include residents chosen with specific demographics and characteristics to broaden the sample criteria.

As research has previously noted, the impact or benefits of psychoactive medications are not exclusive to one specific medication; thus, the anticonvulsant agent Depakote may have some advantages when used for agitation and aggression in dementia residents due to its lower rates of drug interactions and adverse effects in the dementia population (Meinhold et al., 2005). Previous research has found the use of Depakote for agitation and aggression in dementia residents was effective, safe, and tolerable. Also, it

has been found that Depakote may have multiple beneficial effects in NH dementia residents with a history of dementia with behavioral problems. Additional research is needed in the areas of (a) using Depakote treatment as a secondary agent and (b) starting the treatment earlier in the dementia process to explore if it may have more beneficial effects.

Recommendations for Action

This study provides an understanding of the impact of Depakote on agitation and STM in NH dementia residents over a 1-year period. There were no significant relationships or differences between Depakote and agitation scores. A total of 16 residents scores were measured and compared to the other with random effects over a 1-year period. Agitation scores varied but were close enough that statistical analysis showed no significance. With this information, avenues for additional exploration on Depakote and agitation in NH dementia residents can be explored by utilizing a larger sample and additional demographics to highlight the effects of Depakote in a larger population with demographics for a substance to the research.

Although this study did not indicate any statistical significance among Depakote and STM scores, there is now research on STM and how it is impacted by Depakote treatment. Research has now highlighted that STM scores improved from Depakote start date, though not significantly during a 1-year period. This information can potentially help professionals when completing cognitive testing in NH dementia residents on Depakote treatment. Notably, this research may provide some awareness into the fact that

Depakote treatment can play a role in STM; however, appropriate measures must be taken to maintain confidentiality and safety among members of this vulnerable population.

Conclusion

This current study focused on a sample of 16 NH residents with a diagnosis of dementia receiving Depakote treatment for agitation. The research was designed to utilize preexisting data from the consulting pharmacy and two local NHs. The results of the repeated measures ANOVA with within-subjects did not reveal a significant relationship between Depakote and levels of agitation or STM. Results of repeated measures ANOVA with within subjects and random effects did not reveal a significant relationship between Depakote and agitation scores but revealed an improvement in STM scores. Although this study revealed an improvement in STM scores, it did not reveal any significant difference in STM scores over the 1-year study period. Adding a confounding variable, length of time on Depakote did not show any significance in agitation or STM scores. The findings from this study suggest that there may be other factors moderating agitation and STM scores, and further exploration is warranted on the impact of Depakote on agitation and STM in NH dementia residents over a 1-year period.

Results from this study also suggest that there is much more to be explored among the NH dementia population. Because dementia residents are unable to provide information themselves, the researcher must rely on the NH staff for information. It is important for future research to be sure that the information received from preexisting

data is accurate and honest. Without accuracy, results can be affected. Results of this study also reveal that the sample may be too small and that a larger sample would provide more substance to the research.

Understanding of these results may lead to earlier administration of Depakote to NH dementia residents. This research can provide insight to mental health professionals, medical staff, NH staff, and psychologists regarding the treatment needs of NH dementia residents experiencing BPSD. I hope that these findings will bring much-needed awareness to this underserved population so that appropriate care and treatment are enforced, policies for treatment interventions developed and implemented, education made available, and future research made a priority. Research can give a voice to this important and growing population.

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Appendix A: Approved Data Use Agreement

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Data Use Agreement

This Data Use Agreement ("Agreement"), effective as of 04/09/17 ("Effective Date"), is entered into by and between Kristin Fazzolari-Pleace, Ph.D. Candidate ("Data Recipient") and James Czajkowski, RPh, Pharmacy Consultant ("Data Provider"). The purpose of this Agreement is to provide Data Recipient with access to a Limited Data Set ("LDS") for use in research in accord with laws and regulations of the governing bodies associated with the Data Provider, Data Recipient, and Data Recipient's educational program. In the case of a discrepancy among laws, the agreement shall follow whichever law is more strict.

1. Definitions. Due to the study's affiliation with Laureate, a USA-based company, unless otherwise specified in this Agreement, all capitalized terms used in this Agreement not otherwise defined have the meaning established for purposes of the USA "HIPPA Regulations" and/or "FERPA Regulations" codified in the United States Code of Federal Regulations, as amended from time to time.

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    - Medication start date, reason for medication, DOB, and gender.
    - All residents included will be receiving Depakote for behavioral issues, residents receiving Depakote for seizure disorder or other diagnoses will be excluded.

4. Responsibilities of Data Recipient. Data recipient agrees to:

a. Use or disclose the LDS only as permitted by this agreement or required by law;

b. Use appropriate safeguards to prevent use or disclosure of the LDS other than as permitted by this agreement or required by law;

c. Report Data Provider any use or disclosure of LDS of which it becomes aware that is not permitted by this Agreement or required by law;

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e. Not use the information in the LDS to identify or contact the individuals who are the data subjects.

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6. Term and Termination

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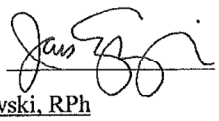
IN WITNESS WHEREOF, each of the undersigned has caused this Agreement to be duly executed in its name and on its behalf.

DATA PROVIDER

Date: 04/13/17

Electronically Signed: 

Name: James Czajkowski, RPh

Title: Pharmacy Consultant
Registered Pharmacist

DATA RECIPIENT

Date: 04/13/17

Electronically Signed: 

Name: Kristin Fazzolari-Pleace, Ph.D. Candidate

Title: Walden Clinical Psychology Student

Appendix B: Approved Data Use Agreement

Appendix B: Approved Data Use Agreement

Data Use Agreement

This Data Use Agreement ("Agreement"), effective as of 04/18/17 ("Effective Date"), is entered into by and between Kristin Fazzolari-Pleace, Ph.D. Candidate ("Data Recipient") and Phyllis Leffler, Administrator, Emerald North Nursing Facility ("Data Provider"). The purpose of this Agreement is to provide Data Recipient with access to a Limited Data Set ("LDS") for use in research in accord with laws and regulations of the governing bodies associated with the Data Provider, Data Recipient, and Data Recipient's educational program. In the case of a discrepancy among laws, the agreement shall follow whichever law is more strict.

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    - First name and last initial, Behavior and cognitive sections of MDS, DOB, gender.
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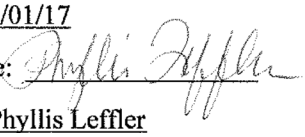
Date: 05/01/17

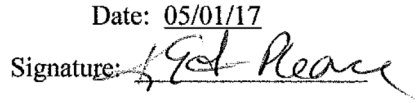
Signature: 

Name: Phyllis Leffler

Title: Administrator

DATA RECIPIENT

Date: 05/01/17

Signature: 

Name: Kristin Fazzolari-Pleace, Ph.D. Candidate

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Appendix C: Approved Data Use Agreement

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Data Use Agreement

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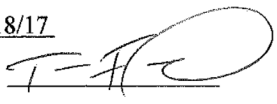
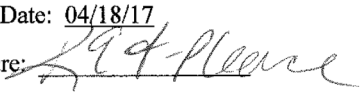
Signature: 

Name: Thomas E. Farrell

Title: Assistant Administrator/ Acting Administrator, Emerald South Nursing Facility

DATA RECIPIENT

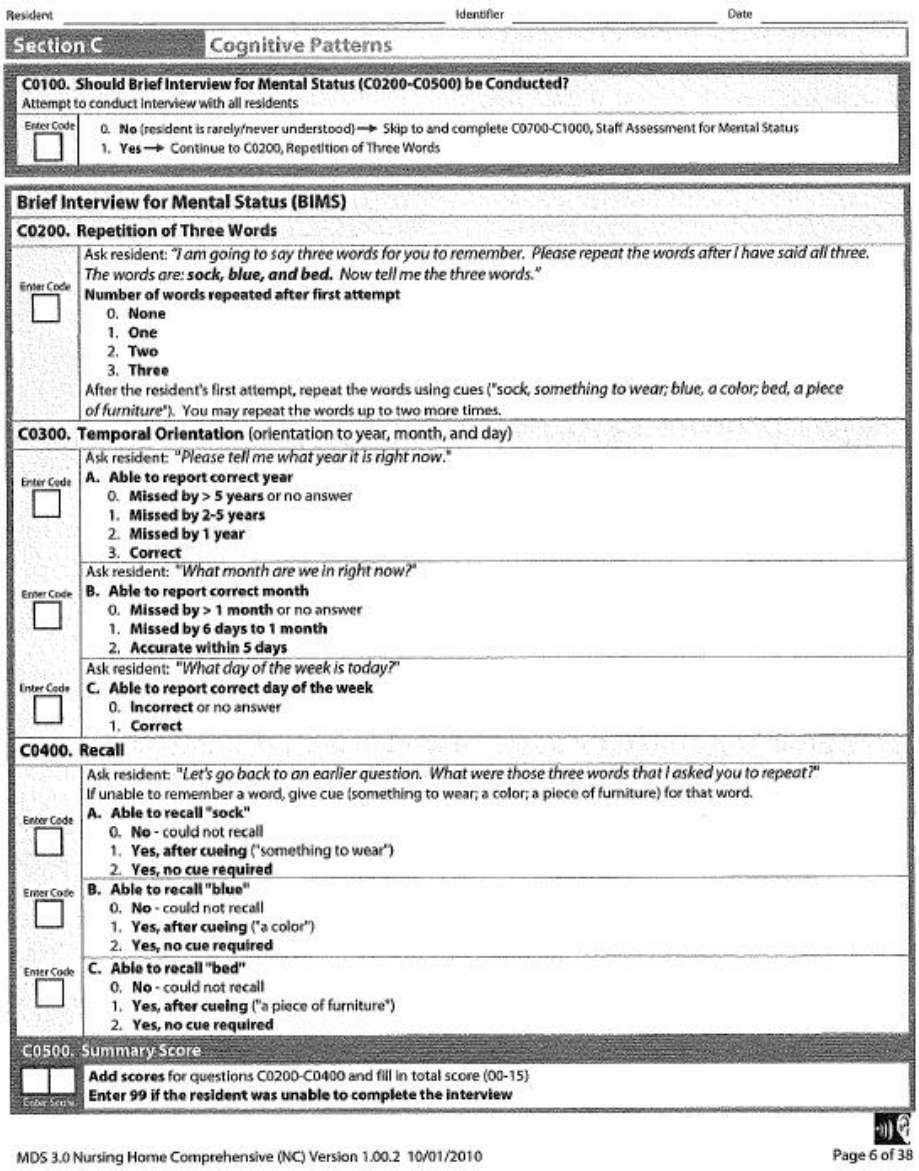
Date: 04/18/17

Signature: 

Name: Kristin Fazzolari-Pleace, Ph.D. Candidate

Title: Walden Clinical Psychology Student

Appendix D: Example MDS Section C (Cognitive) and Section E (Behavioral)



Resident _____ Identifier _____ Date _____

Section C Cognitive Patterns

C0100. Should Brief Interview for Mental Status (C0200-C0500) be Conducted?
Attempt to conduct interview with all residents

Enter Code ☐
0. **No** (resident is rarely/never understood) → Skip to and complete C0700-C1000, Staff Assessment for Mental Status
1. **Yes** → Continue to C0200, Repetition of Three Words

Brief Interview for Mental Status (BIMS)

C0200. Repetition of Three Words

Enter Code ☐
Ask resident: *"I am going to say three words for you to remember. Please repeat the words after I have said all three. The words are:* ***sock, blue, and bed.*** *Now tell me the three words."*
Number of words repeated after first attempt
0. **None**
1. **One**
2. **Two**
3. **Three**
After the resident's first attempt, repeat the words using cues ("*sock, something to wear; blue, a color; bed, a piece of furniture*"). You may repeat the words up to two more times.

C0300. Temporal Orientation (orientation to year, month, and day)

Enter Code ☐
Ask resident: "*Please tell me what year it is right now.*"
A. Able to report correct year
0. **Missed by > 5 years** or no answer
1. **Missed by 2-5 years**
2. **Missed by 1 year**
3. **Correct**

Enter Code ☐
Ask resident: "*What month are we in right now?*"
B. Able to report correct month
0. **Missed by > 1 month** or no answer
1. **Missed by 6 days to 1 month**
2. **Accurate within 5 days**

Enter Code ☐
Ask resident: "*What day of the week is today?*"
C. Able to report correct day of the week
0. **Incorrect** or no answer
1. **Correct**

C0400. Recall

Enter Code ☐
Ask resident: "*Let's go back to an earlier question. What were those three words that I asked you to repeat?*"
If unable to remember a word, give cue (something to wear; a color; a piece of furniture) for that word.
A. Able to recall "sock"
0. **No** - could not recall
1. **Yes, after cueing** ("something to wear")
2. **Yes, no cue required**

Enter Code ☐
B. Able to recall "blue"
0. **No** - could not recall
1. **Yes, after cueing** ("a color")
2. **Yes, no cue required**

Enter Code ☐
C. Able to recall "bed"
0. **No** - could not recall
1. **Yes, after cueing** ("a piece of furniture")
2. **Yes, no cue required**

C0500. Summary Score

Enter Score ☐☐
Add scores for questions C0200-C0400 and fill in total score (00-15)
Enter 99 if the resident was unable to complete the interview

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Resident _____ Identifier _____ Date _____

Section C Cognitive Patterns

C0600. Should the Staff Assessment for Mental Status (C0700 - C1000) be Conducted?

Enter Code ☐

0. **No** (resident was able to complete interview) → Skip to C1300, Signs and Symptoms of Delirium
1. **Yes** (resident was unable to complete interview) → Continue to C0700, Short-term Memory OK

Staff Assessment for Mental Status

Do not conduct if Brief Interview for Mental Status (C0200-C0500) was completed

C0700. Short-term Memory OK

Enter Code ☐

Seems or appears to recall after 5 minutes
0. **Memory OK**
1. **Memory problem**

C0800. Long-term Memory OK

Enter Code ☐

Seems or appears to recall long past
0. **Memory OK**
1. **Memory problem**

C0900. Memory/Recall Ability

↓ **Check all that the resident was normally able to recall**

- ☐ **A. Current season**
- ☐ **B. Location of own room**
- ☐ **C. Staff names and faces**
- ☐ **D. That he or she is in a nursing home**
- ☐ **Z. None of the above** were recalled

C1000. Cognitive Skills for Daily Decision Making

Enter Code ☐

Made decisions regarding tasks of daily life
0. **Independent** - decisions consistent/reasonable
1. **Modified independence** - some difficulty in new situations only
2. **Moderately impaired** - decisions poor; cues/supervision required
3. **Severely impaired** - never/rarely made decisions

Delirium

C1300. Signs and Symptoms of Delirium (from CAM©)

Code **after completing** Brief Interview for Mental Status or Staff Assessment, and reviewing medical record

Coding:
0. **Behavior not present**
1. **Behavior continuously present, does not fluctuate**
2. **Behavior present, fluctuates** (comes and goes, changes in severity)

↓ **Enter Codes in Boxes**

- ☐ **A. Inattention** - Did the resident have difficulty focusing attention (easily distracted, out of touch or difficulty following what was said)?
- ☐ **B. Disorganized thinking** - Was the resident's thinking disorganized or incoherent (rambling or irrelevant conversation, unclear or illogical flow of ideas, or unpredictable switching from subject to subject)?
- ☐ **C. Altered level of consciousness** - Did the resident have altered level of consciousness (e.g., **vigilant** - startled easily to any sound or touch; **lethargic** - repeatedly dozed off when being asked questions, but responded to voice or touch; **stuporous** - very difficult to arouse and keep aroused for the interview; **comatose** - could not be aroused)?
- ☐ **D. Psychomotor retardation** - Did the resident have an unusually decreased level of activity such as sluggishness, staring into space, staying in one position, moving very slowly?

C1600. Acute Onset Mental Status Change

Enter Code ☐

Is there evidence of an acute change in mental status from the resident's baseline?
0. **No**
1. **Yes**

Resident _____ Identifier _____ Date _____

Section E Behavior

E0100. Psychosis

↓ Check all that apply

- ☐ A. **Hallucinations** (perceptual experiences in the absence of real external sensory stimuli)
- ☐ B. **Delusions** (misconceptions or beliefs that are firmly held, contrary to reality)
- ☐ Z. **None of the above**

Behavioral Symptoms

E0200. Behavioral Symptom - Presence & Frequency

Note presence of symptoms and their frequency

Coding:
0. **Behavior not exhibited**
1. **Behavior of this type occurred 1 to 3 days**
2. **Behavior of this type occurred 4 to 6 days,** but less than daily
3. **Behavior of this type occurred daily**

↓ Enter Codes in Boxes

- ☐ A. **Physical behavioral symptoms directed toward others** (e.g., hitting, kicking, pushing, scratching, grabbing, abusing others sexually)
- ☐ B. **Verbal behavioral symptoms directed toward others** (e.g., threatening others, screaming at others, cursing at others)
- ☐ C. **Other behavioral symptoms not directed toward others** (e.g., physical symptoms such as hitting or scratching self, pacing, rummaging, public sexual acts, disrobing in public, throwing or smearing food or bodily wastes, or verbal/vocal symptoms like screaming, disruptive sounds)

E0300. Overall Presence of Behavioral Symptoms

Enter Code ☐

Were any behavioral symptoms in questions E0200 coded 1, 2, or 3?
0. **No** → Skip to E0800, Rejection of Care
1. **Yes** → Considering all of E0200, Behavioral Symptoms, answer E0500 and E0600 below

E0500. Impact on Resident

Did any of the identified symptom(s):

Enter Code ☐ **A. Put the resident at significant risk for physical illness or injury?**
0. **No**
1. **Yes**

Enter Code ☐ **B. Significantly interfere with the resident's care?**
0. **No**
1. **Yes**

Enter Code ☐ **C. Significantly interfere with the resident's participation in activities or social interactions?**
0. **No**
1. **Yes**

E0600. Impact on Others

Did any of the identified symptom(s):

Enter Code ☐ **A. Put others at significant risk for physical injury?**
0. **No**
1. **Yes**

Enter Code ☐ **B. Significantly intrude on the privacy or activity of others?**
0. **No**
1. **Yes**

Enter Code ☐ **C. Significantly disrupt care or living environment?**
0. **No**
1. **Yes**

E0800. Rejection of Care - Presence & Frequency

Enter Code ☐

Did the resident reject evaluation or care (e.g., bloodwork, taking medications, ADL assistance) **that is necessary to achieve the resident's goals for health and well-being?** Do not include behaviors that have already been addressed (e.g., by discussion or care planning with the resident or family), and/or determined to be consistent with resident values, preferences, or goals.
0. **Behavior not exhibited**
1. **Behavior of this type occurred 1 to 3 days**
2. **Behavior of this type occurred 4 to 6 days,** but less than daily
3. **Behavior of this type occurred daily**

Resident _____ Identifier _____ Date _____

Section E Behavior

E0900. Wandering - Presence & Frequency

Enter Code ☐

Has the resident wandered?
0. **Behavior not exhibited** → Skip to E1100, Change in Behavioral or Other Symptoms
1. **Behavior of this type occurred 1 to 3 days**
2. **Behavior of this type occurred 4 to 6 days**, but less than daily
3. **Behavior of this type occurred daily**

E1000. Wandering - Impact

Enter Code ☐

A. Does the wandering place the resident at significant risk of getting to a potentially dangerous place (e.g., stairs, outside of the facility)?
0. **No**
1. **Yes**

Enter Code ☐

B. Does the wandering significantly intrude on the privacy or activities of others?
0. **No**
1. **Yes**

E1100. Change in Behavior or Other Symptoms

Consider all of the symptoms assessed in Items E0100 through E1000

Enter Code ☐

How does resident's current behavior status, care rejection, or wandering **compare to prior assessment (OBRA or PPS)?**
0. **Same**
1. **Improved**
2. **Worse**
3. **N/A** because no prior MDS assessment